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BSPP News

The Newsletter of the British Society for Plant Pathology

25th Anniversary edition

Contents

BSPP BOARD MEMBERS 2006

And a date for your 2007 diary:

The **2007 BSPP Presidential Meeting** will be held at the **University of Bath, Sept 12th-14th.** The theme will be **'Attack and Defence in Plant Pathogen Interactions.'** We look forward to seeing you there.

Happy Christmas and New Year

Newsletter edited by Dr Matt Dickinson, University of Nottingham

… and getting his just desserts.

On a more trivial note, another paper dealt with mycotoxin contamination in grain-based pet foods. Being a devoted cat owner I paid great attention to this presentation and was alarmed to discover that kitty is particularly sensitive to the fusarium mycotoxin, zearalenone. In the absence of a simple zearalenone assay that I can use to test kitty's dinner, I will have to rely on continued efforts to improve resistance to fusarium in both wheat and maize, which are common components of dried cat food. The presentation that followed this alarming insight into what might be in kitty's supper dealt with the more serious issue of lymphocytic choriomeningitis virus. This can cause meningitis, encephalitis or meningoencephalitis in humans and there are concerns that it may represent a hidden danger within grain store premises. The reason for this is that its primary host is the house mouse, which is an almost ubiquitous resident of grain stores (except in the UK, of course). The take home message from this for me was that kitty should be sent out to catch a murine supper in the grain store. She may be doing herself and her owner a favour.

Roger Williams, HGCA

25th Anniversary Celebratory Meeting
Imperial College, London, 19th December 2006

10.15 – 10.45	**Registration and Tea / Coffee**
10.45 – 11.00	**Welcome – Professor Peter Mills, BSPP President**
11.00 – 11.30	**Peter Scott, Immediate Past President of ISPP** "The global impact of plant diseases"
11.30 – 12.00	**Chris Gilligan, University of Cambridge** "Epidemiology: from individuals to populations"
12.00 – 12.30	**Ian Smith, Formerly Director-General EPPO, Paris** "Past, current and future disease threats to plants in the United Kingdom"
12.30 – 13.00	**James Brown, John Innes Centre, Norwich** "From genetics to plant breeding: what do we know and what do we need to know?"
13.00 – 13.40	**LUNCH**
13.40 – 14.10	**Naomi Pain, Syngenta, Jealotts Hill Research Centre** "Agrochemical Control: Screening, costs, efficacy and environment"
14.10 – 14.40	**John Whipps, Warwick-HRI** "Alternative control methods"
14.40 – 15.10	**John Mansfield, Imperial College at Wye** "The pathogen; mechanisms of attack and novel targets"
15.10 – 15.30	**TEA / COFFEE**
15.30 – 16.00	**Kim Hammond-Kosack, Rothamsted Research** "The host: Resistance gene isolation and realising the potential"
16.00 – 16.30	**Tony Gilland, Institute of Ideas, London** "Public understanding of plant pathology; what do the public know and what shapes what they know?"
16.30 – 17.00	**Jeff Waage, Imperial College at Wye** "Foresight: plant pathology in a global disease context"
17.00	**BSPP Annual General Meeting**

MEETING ABSTRACTS

The global impact of plant diseases

Peter Scott

BSPP was born 25 years ago because Britain had no professional society devoted to plant diseases and their impact. In fact BSPP *had* to be born, because the societies closest to plant pathology couldn't agree on who should manage the journal *Plant Pathology*, with its practical emphasis on mechanism, impact and management of plant disease. The impact of plant disease is everywhere, and is inadequately appreciated by policy makers - something which immediately suggests a role for BSPP. Considering food crops alone, the 14 staples are subject to more than 100 principal diseases, and to a total of thousands. At least 10% of global food production is thought to be lost to plant disease. Though this estimate lacks a strong base, such losses must make a huge global impact when, according to the World Bank, more than 800 million people do not have adequate food, and more than 1 billion live on less than $1 a day.

The most serious impacts in human terms occur in developing countries, which provide innumerable examples of damaging pathogens of crops and forests, with serious impacts on food security. Impacts in industrial countries may be less obvious, because of better management and because there may be food surpluses, but substantial costs are still incurred. Plant diseases have shaped history: the impact of potato blight in Ireland in the 1840s was starvation for around 1 million people, while more than 1 million attempted to emigrate. In the US, the southern corn leaf blight epidemic of 1970-1 did not cause starvation but threatened the whole corn industry, through its dependence on a narrow genetic base. Equally serious is the poorly quantified ongoing impact of the thousands of species of fungi, bacteria, viruses and oomycetes on crops, forests and wild plants that we plant pathologists live by. It would be a mistake for the discipline of plant pathology to thrive primarily as a field of study for molecular biology, powerful though this is in understanding plant disease. The BSPP recognizes also that plant disease has economic and environmental impact that truly matter in social and economic terms. The International Society for Plant Pathology (ISPP) has a Task Force on Global Food Security with a small programme aimed at influencing public opinion and policy. It is exploring the development of a new journal, which may be called the *Journal of Food Security*.

Plant diseases are here to stay. We can dream of a world without them, but natural selection will ensure that it remains a dream. BSPP, from its position of authority as a science-based organization, will have much to attend to during its next 25 years. Raising awareness of the impact of plant disease is a critical - and challenging - place to start.

At present there are no simple, reliable methods available to do this. However, one paper described a technique based on near-infrared reflectance spectroscopy and high speed sorting that shows some promise with respect to removing maize kernels contaminated with fumonisin and aflatoxin.

Initial studies suggest that it may be possible to develop this approach, with the aid of neural networks, to identify individual maize kernels infected with a range of fungi even where more than one is present in the same kernel. This could be of considerable benefit to plant breeders as it may offer a rapid method for resistance screening.

A mycotoxin issue of particular concern in Brazil at present is aflatoxin contamination of Brazil nuts. The nuts are collected by hand from the forest floor and transported by river for up to 60 days before being dried on intake at the processing plant. During this time, conditions are often conducive to proliferation of *Aspergillus flavus* and contamination with associated aflatoxins. The EU has particularly stringent limits for aflatoxin content in Brazil nuts, which has had a significant impact on the importation to the EU of in-shell nuts. Unfortunately, it appears that there is relatively little that can be done to reduce the risk of aflatoxin contamination and the critical control point is careful sorting, on the basis of visual symptoms of infection, at intake at the processing plant. Whilst Brazil nut production is a relatively minor component of Brazilian agriculture, it is an important industry for some of the 27m people living in the Amazon region where employment opportunities are relatively limited.

Roger Williams, HGCA, singing for his supper at IWCSPP …

9th International Working Conference on Stored Product Protection (IWCSPP), Sao Paulo, Brazil

One of the most stimulating aspects of attending the 9th IWCSPP in Brazil was being reminded that applied science, including disciplines such as plant pathology, can make an enormous contribution to the prosperity and well-being of individuals and nations.

The conference, which was attended by 650 delegates from 46 countries, was opened by the Minister of Agriculture, Livestock and Food Supply. Much of his address focussed on the benefits that research and knowledge transfer have brought to Brazilian agriculture and the role that these will continue to play in the extraordinary increase in farm productivity occurring in Brazil. For example, grain production has more than doubled over the last 15 years yet the area planted has only increased by 20%. He attributed this to the effective application of research into practice. When was the last time that an important UK political figure gave UK science a pat on the back and said 'thanks guys'?

There was a slight risk that this upbeat view of research and advisory activities would be lost when the Minister decided to furnish delegates, at considerable length, with his views on the iniquity of agricultural subsidies around the world. Every silver lining has a cloud. To put his comments in context though, it is important to note that agribusiness accounts for 28% of Brazil's GDP, directly employs 17.7m people, and is therefore a very significant political issue.

By a strange coincidence the first day of the conference, 16 October, had been designated by the FAO as 'World Food Day'. This was intended to be an opportunity to reflect on the basic human right to safe food. It was therefore particularly sobering to hear that it is estimated that within Brazil alone, losses during grain storage would feed an estimated 20m people for a year.

Although pests account for considerable post-harvest losses worldwide, fungi are also extremely important, especially with respect to impacts on quality and safety of stored commodities.

Fusarium and associated mycotoxins received considerable attention at the conference, with papers on various aspects of their occurrence and control in both maize and wheat appearing throughout the programme. It was recognised that in most situations the critical control point for fusarium mycotoxins is in the field, which begs the question: what was the relevance of this topic to a conference on stored product protection? The relevance lies partly in the fact that intake at the grain store is, theoretically, one of the first opportunities to screen out mycotoxin contaminated grain from the food and feed chains.

Epidemiology: from individuals to populations

Christopher A. Gilligan, *University of Cambridge*

J.E. Van der Plank wrote in 1963 that "Chemical industry and plant breeders forge fine tactical weapons but only epidemiology sets the strategy". This is still true, more than forty years later. It underlines the continued quest for sustainable disease control, which, itself, rests on a paradox. Since most plants are self-evidently resistant to most pathogens, it seems perfectly reasonable to assume that advancing knowledge of the genetical, molecular, and cellular bases of host-pathogen interaction will identify the means not only to engineer or to select durable resistance but also to produce effective and environmentally neutral forms of chemical control. Yet failures still occur and the problems are exacerbated by escalating costs for release of new varieties and for the development and registration of new chemicals. These problems partly reflect differences in scales between screening and deployment underlining the need to integrate epidemiology – 'the science of disease in populations' – with molecular biology, and host-pathogen genetics and physiology. Most novel forms of disease control are screened for effectiveness at the small scale. Often this is done at scales as small as the single plant for initial screening, though more usually it involves multiple field plots and ultimately fields. Yet successful deployment – and the risk of failure – occurs at scales much larger than this, at the regional, national or even international scales. The epidemiological challenges are not all technologically driven: agriculture and natural vegetation continue to be confronted by new and recurrent epidemics. The problems in minimising the risks of failures of control and in managing emerging epidemics demand a common epidemiological approach that considers invasion, persistence, scaling and chance.

Having reviewed briefly the progress in epidemiology during the past 25 years, I shall illustrate likely future developments in constructing an epidemiological framework to model invasion, persistence and variability of epidemics that encompasses a wide range of scales and topologies through which disease spreads. By considering how to map control methods onto epidemiological parameters and variables, some new approaches towards optimising the efficiency of control at the landscape scale will be described. Some epidemiological strategies to minimise the risks of failure of chemical and genetical control will be presented and, if time permits, some consequences of heterogeneous selection pressures in time and space on the persistence and evolutionary changes of pathogen populations discussed. Finally, brief mention will be made of how we might embed epidemiological models in an economically-plausible framework for the deployment of control.

Past, current and future disease threats to plants in the United Kingdom

Ian M. Smith, *formerly Director-General, EPPO*

As an island, the United Kingdom has been particularly concerned to protect itself from the threat presented by the introduction of non-native pests (including plant pathogens). It has taken particular care to develop effective plant quarantine. Twenty five years ago, the British phytosanitary system was still more or less that inherited from the period before EU membership, with an approach akin to that of many island countries around the world, with prohibitions or severe restrictions of plant imports from most other countries. EU membership changed the system, so that by 1993 imports from other EU countries were able to enter relatively freely under the plant passport system, and imports from the rest of the world were regulated by the EU system, which applies prohibitions and restrictions in reaction to specifically identified risks. Through the 1990s, this more specific approach was reinforced by the development of the Sanitary and Phytosanitary (SPS) Agreement of the World Trade Organization, which requires members to provide, if challenged, technical justification of phytosanitary measures. Plant quarantine thus entered the world of "risk analysis", which primarily allows new pest risks to be evaluated and countered, but secondarily (and more politically) seeks to avoid trade disputes. The major problems which have arisen with the risks from animal diseases (BSE) and LMOs have also had their effects on plant quarantine, which has become more laboriously administrative and "risk-averse".

Another major development of the 1990s has been the improvement of diagnostics. Plant pathogens can be identified more readily, which is a direct advantage, but disputes about diagnosis, and the distinction between specified risky species and others, become more probable. New diagnostic techniques also make it easier to produce disease-free planting material, which in principle could move with little restriction in international trade. However, international standards are needed to back this development.

The Convention on Biological Diversity (CBD) has recently developed new agreements on the movement of alien species, which overlap to a certain extent with plant quarantine. This has led to a greater focus on risks to plants in native ecosystems, rather than in agriculture or forestry. With a greater political emphasis of the protection of the consumer and the environment, and a lesser emphasis on support for agriculture, these aspects are likely to take on more importance (though in practice risks from invasive alien plants and animals have received most attention).

These various developments over the last 25 years are illustrated by reference to individual cases of plant diseases of phytosanitary importance.

though the bacteria were present at densities usually associated quorum sensing dependent expression of lytic exo-enzymes. Perhaps this isolate of *Pbc* could serve as a candidate for the study of the biotrophic phase of the pathogen? Secondly, a presentation by Won-Sik Kim (Agriculture and Agri-Food Canada) described a method for detection of plum-pox virus (la Sharka) via Direct RT-PCR, which does not require RNA extraction. His method has proven to be more sensitive and more reliable that the standard ELIZA tests, and can be applied as a rapid, early disease warning system. The key component to his new method is the Direct Pathogen Extraction Buffer, which he has offered to provide to interested labs until he can finalize the intellectual property rights.

Unfortunately, I must report that I was disappointed with the Art in Phytopathology event. Previous to my arrival, I had been in contact with the event's organizers in order to ensure that some equipment would be available for me to present a multimedia composition of visual and musical artwork based on the *Pectobacterium* genome. However, the event was poorly organized and none of the equipment that I required was provided by the organizers. In fact there was only one exhibit in the display – a collage of micrographs of various water moulds, which had been assembled to create a picture of Heinrich Anton de Bary.

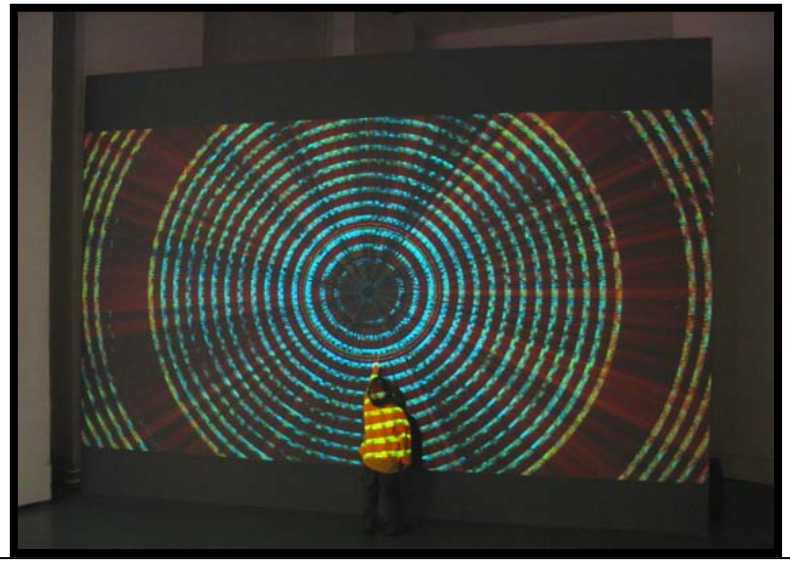
Overall, the APS/CPS/MSA 2006 Joint Meeting was an excellent experience for me. Primarily this was an opportunity for me to promote my contributions to the research being conducted at SCRI, on an international level. However, in combination with my attendance at a couple of UK-based international conferences previous to the APS/CPS/MSA 2006 Joint Meeting, I was able get a much better understanding of the relative quality of the research being conducted in the UK. Finally, I was able to make some personal contacts in other labs, which may prove useful for the development of future collaborations. I would like to thank the BSPP for its financial support which helped to make this valuable experience possible.

Michael Ravensdale, SCRI

moment. Specifically, *Xanthomonas campestris vesicatoria* has two T3SS effectors XopN and XopD which may functions via molecular mimicry. XopN contains HEAT repeats, a motif that is common in eukaryotes but rare in prokaryotes. It is hypothesized that these HEAT repeats may interact with plant LRR-RLKs, causing a kinase cascade which ultimately serves to disrupt plant defenses.

Likewise, XopD is a mimic of a eukaryotic protease, and it has been shown to cause growth defects in yeast and plants. Recent research into the *Pseudomonas* T3 effector AvrPto has revealed that this protein mimics plant ubiquitin ligases, the actions of which function to suppress host HR and HR based defenses. My work also fit into this contemporary theme, and my presentation: "Phytotoxins and suppression of resistance: New mechanisms of disease induction in the plant pathogen *Pectobacterium atrosepticum*" described the role and regulation of the plant hormone mimic, coronafacic acid, in *Pba* pathogenesis.

While I attended many talks of merit, there were two that I felt were the most intriguing. Firstly, a report from Gina Rodrigez (Simon Fraser University, Canada) described the occurrence of *Pectobacterium carotovorum* in wasabi. Unusually, the bacteria appeared to be isolated to the xylem where they caused what may be an HR induced necrosis. However, they did not cause host tissue maceration even



The Erwinia genome-based artwork that failed to see the light of day in Quebec City.

From genetics to plant breeding: what do we know and what do we need to know?

James Brown, *John Innes Centre, Norwich*

Plant breeding is one of the most revolutionary, life-changing technologies developed in the 20th century. Yet despite the proven success of this technology, the last 25 years have been marked by a severe decline in the teaching of plant breeding in universities and in research on genetics to underpin breeding. Meanwhile, expenditure has flourished on genetic manipulation, a technology which has promised far more than it has delivered. The situation in academia contrasts sharply with the continuing success of the largely private plant breeding industry. A chasm has therefore opened up between research on plant genetics and commercial plant improvement. I will argue that likely changes in the severity of different plant diseases over the next 25 years means that it is now more important than ever to use successful, trustworthy technology to combat them. I will discuss (1) reasons for the decline of plant breeding as an academic subject, (2) what we do (and don't) need to know about plant genetics, pathogen evolution and pathogenesis to support breeding for disease resistance, (3) how technological developments over the last 25 years have (and haven't) improved resistance breeding and (4) how the genuine benefits of GM can be integrated with the proven technology of breeding to control disease. Examples will be taken from fungal diseases of cereals.

Agrochemical control: Screening, costs, efficacy and environment

Naomi Pain, *Syngenta, Jealotts Hill Research Centre*

The last 25 years have seen significant changes in the structure of the agrochemical industry, and the pressures it faces. In agriculture, the need for novel fungicides has persisted. More sophisticated, potent chemistries with novel modes of action and effects have been introduced to the market place. Different properties have required growers to understand the products to use them to best effect, and agribusinesses, advisors and grower groups have provided education and recommendations for improved disease control. However, increasing regulatory requirements and hence development costs of novel products have necessitated an increase in efficiency and cost-effectiveness within the industry.

One result of this has been extensive consolidation. Within research and development, further changes have been made to improve cost savings. Novel technologies (miniaturisation, automated liquid handling, combinatorial chemistry, data analysis systems etc) have enabled the implementation of *in vivo* (living target based) and *in vitro* (biochemical target-based) high throughput assays. Success of these approaches is closely scrutinised, and the failure for the industry to deliver on expectations has seen a re-focussing of priorities. We are now seeing the focus shifting towards balance between through put and data quality to meet the business needs of delivery of "blockbuster" products.

Looking forward, in a relatively static agricultural market, the fungicide sector is showing growth. This can be attributed to a number of factors: the emergence of new diseases, increased significance of others, occurrence of resistance to existing products, increasing consumer and processor demand for high quality, consistent food. These areas will be explored further, and a view of the future of crop protection alongside other disease control approaches (biotech, native traits etc.) will be presented.

Joint Meeting of The American Phytopathological Society, The Canadian Phytopathological Society, and the Mycological Society of America, Québec City, Canada, July 29 - August 2 2006

The 2006 Joint Meeting of the APS, CPS and MSA was held at the Québec Convention Center in the city center of Québec City, just outside the walls of the historic Old City (Vieux-Québec). For the majority of my trip, the weather in Quebec City was very hot and humid. These conditions were ideal however for the enjoyment of the local blanche beer – Blanche de Chambly. Also of note was the quality of the local cuisine, with dinner at Restaurant Aux Anciens Canadiens (serving traditional Quebecois fare) and lunch at La Lapin Sauté (all items prepared with rabbit) being the highlights.

The conference was themed: "Biological Interactions and Biological Crossroads" and it was attended primarily by delegates from the Americas. There were seven formal sessions: 1) Biology of Plant Pathogens; 2) Diseases of Plants; 3) Epidemiology / Ecology / Environmental Biology; 4) Molecular / Cellular Plant-Microbe Interactions; 5) Plant Disease Management; 6) Professionalism /Service / Outreach; 7) Mycological Society of America. There was also an enormous poster session, which featured over 800 posters. Additionally, there were numerous field trips offered to the delegates including a trip to Grosse-Île: a small island in the St. Lawrence River which was used a quarantine stop-over for European immigrants to the New World as early as 1832. Grosse-Île has historic connections to the Irish Potato famine as many of the Irish immigrants who fled the famine in 1847, first landed on this island. Other field trips included Forest Pathology and Turf Grass tours.

The Welcome and Plenary Session was the only opportunity for all delegates to listen to the same speakers. It was one of the largest presentation venues that I have experienced and it featured three excellent speakers: Jeff Townsend, Zamir Punja and David Gilchrist. These speakers gave very different presentations:

Jeff Townsend focused upon the technical and statistical challenges associated with the study of transcriptional profiling in fungi; Zamir Punja discussed plant disease management via GM up-regulation of plant defense hormones; David Gilchrist talked about convergent technical and social challenges faced by phytopathologists and in other molecular disciplines.

Being a joint meeting, there were many disciplines represented, but the majority of presentations were about fungal pathogens. However my studies required that I attend talks which featured bacterial pathogens and their molecular interactions with their hosts. In regards to *Xanthomonas* and *Pseudomonas* research, there seems to be a focus on molecular mimicry at the

ideal platform for scientists, practitioners and stake-holders to exchange latest results of scientific research as well as to discuss the implementation of IAS strategies in a Pan-European context .

The emphasis of the 4th Neobiota conference, organized jointly by the Austrian Federal Environment Agency and the German Federal Agency for Nature Conservation, lay on the implementation of IAS strategies, based on sound scientific knowledge focussing on aspects of species ecology through to the conservation of biodiversity in Europe. During the meeting all fields of biological invasions covering pathogens, plants, fungi and animals as invasive organisms in marine, freshwater and terrestrial habitats were addressed with invited key notes as well as offered oral and poster presentation.

Registering for the meeting on wednesday afternoon each participant was presented with a small jar of jam made of Japanese Knotweed (*Fallopia japonica*). Tasting surprisingly nice, this might offer one option to take advantage of the growing infestation by this number one invasive species in Europe originating from the volcanic mountain slopes in Japan! The meeting was then opened by a welcoming note given by Wolfgang Rabitsch from the Austrian Federal Environment Agency which was followed by an invited keynote lecture on "high and low tech success stories to combat IAS" presented by Prof. Daniel Simberloff from the University of Tennessee stimulating much discussion. The schedule for the two subsequent days was packed with oral and poster presentations given that the actual number of delegates had exceeded all expectations. Poster presentations were held during coffee and lunch giving opportunities for discussion amongst delegates as well as to renew old and establish new contacts. My talk addressing the potential for biological control of invasive weeds in Europe contributed the aspect of applied mycology / plant pathology as one strategy against IAS to the conference and was received with lots of interest. Classical biological control, the concept of controlling alien invasive weeds in their introduced ranges using host specific natural enemies, i.e. fungal pathogens and arthropods, from their native range constitutes a safe, environmentally sound and economic method which has been implemented for many years in countries such as Australia, USA, New Zealand and South Africa. In Europe the uptake of this approach has been quite slow, but the level of interest is rising with the issue of invasive weeds such as Japanese Knotweed (*Fallopia japonica*), Ragweed (*Ambrosia artemisiifolia*) and Himalayan Balsam (*Impatiens glandulifera*) becoming more and more pressing.

Apart from the scientific contributions one other highlight of the meeting was definitely the conference dinner held at the Natural History Museum in the centre of Vienna. This was only topped by the opportunity to have a small guided tour which ended on the roof of the building giving the view over the city by night. For me the conference was highly inspiring and I would like to thank the BSPP for the travel award that enabled me to attend this meeting. The 5th Neobiota conference will be held in Prague in 2008.

Marion Seier, CAB International, Silwood Park, Ascot

Alternative control methods

John M Whipps, *Warwick HRI, University of Warwick, Wellesbourne*

Disease control has traditionally relied heavily on the use of chemicals and disease resistant varieties of plants. However, with increasing resistance of pathogens to chemicals, pressure to decrease chemical use in the environment, and reduced availability of active ingredients for disease control, the need for alternative means of disease control has now become even more important. This is especially so for soil-borne pathogens with the loss of the soil sterilant, methyl bromide. Traditional cultural and environmental procedures are still of value and are being adapted to the current situation. These commonly include quarantine, basic hygiene measures, use of crop rotations, soil steaming, solarization, and organic amendments.

Other more specialised techniques such as micropropagation for virus-free stock, thermotherapy for controlling seed-borne pathogens and vector control are also options. In the glasshouse, where environmental control is possible, other techniques such as humidity, temperature and fertigation control can be utilised as well. In addition, microbial inoculants (biological disease control agents) are gradually entering the market with at least 3 viral products, over 30 bacterial products and 50 fungal products available worldwide, including some recently available in the UK. The potential also exists to integrate some of these alternative disease control methods to enhance disease control further. Some specific examples based on the use of *Coniothyrium minitans, Pythium oligandrum and Trichoderma viride* will be discussed.

The pathogen; mechanisms of attack and novel targets

John Mansfield, Marta de Torres, Ian Brown and Murray Grant, *Division of Biology, Imperial College London*

Perhaps the most remarkable discovery in recent years has been that bacteria are able to inject proteins into plant cells. Unravelling the role of the type three secretion system (T3SS) has allowed new perspectives to be developed on mechanisms of innate immunity and their suppression by pathogens. A clear link has been forged between plant and animal pathosystems. The role of effector proteins delivered through the T3SS in the induction of disease and activation of the hypersensitive reaction has provided new insights into the regulation of plant defences.

We have used delivery of proteins through a non-pathogen and also *in planta* expression to examine the impact of potential effectors on plant defence in Arabidopsis. Our focus is on the HopAB family including AvrPtoB. Using the RW60 strain of *Pseudomonas syringae* pv. *phaseolicola* we found that AvrPtoB suppresses basal resistance particularly in the absence of the flagellin (flg22) receptor FLS2. Expression of AvrPtoB in the plant suppressed defences induced by flg22 and also the elf20 peptide, but only if the effector was induced one or two hours before elicitor challenge. The timing of exposure of plant cells to elicitors and effectors has a clear influence on the outcome of interactions. Reduction in callose accumulation was observed and also a reprogramming of the defence transcriptome characterising basal resistance. Disease development and AvrPtoB-induced susceptibility, were associated with increases in abscisic acid (ABA) concentrations and ABA-induced gene expression. The potential and rather unexpected role of plant hormones such as ABA in rapidly modulating the leaf environment to favour pathogenesis will be discussed.

One was an account on the struggle by Ghana to control the disease by eradicating infected trees. This particular presentation was captured by BBC News Front Page on their website of Friday 13th October 2006. The other was on efforts to develop genetic markers for screening CSSV resistant materials, and this is part of my postgraduate work at the University of Reading.

The South East Asian producing countries do not seem to have any major disease problems. Their reports were on pod damage caused by rodents and insects. In fact all other topics discussed at the meeting were equally important but limitation on time and space cannot permit to report on all, and I will like to advise anyone interested to consult the the book of abstract of the 15ICRC for more information.

My final remark is on the round table discussion on chocolate and human health. A four member-panel of experts including Chris Johnson (Dr.), Hagen Schroeter (Dr.) and Fedrick K. Addai (Prof.) explored the health benefits of chocolate and other cocoa products. They highlighted on how health benefits of cocoa flavanols can be accurately measured, and also talked on the cardiovascular effects of the flavanols on health and diseases of humans. These discussions were very revealing to me especially against the background information that chocolate and other cocoa products cause very nasty diseases!

The conference was indeed a learning experience and it has exposed me to the world. Thanks to BSPP for part-sponsoring my participation at the conference that enabled me to make such a huge impact.

Henry Dzahini-Obiatey.
University of Reading/Cocoa Research Institute of Ghana.

Neobiota – From Ecology to Conservation; 4th European Conference on Biological Invasions, Vienna (Austria), 27-29 Sept 2006.

This Neobiota conference held at the Old University Campus in Vienna from 27th-29th Sept was the fourth in a row of biannual meetings addressing the issue of biological invasions by alien species in Europe. Since the first meeting held in Berlin in 2000, the number of participants has grown steadily with over 350 delegates from around 43 countries registered at this year's conference. Representation from countries outside Europe included the USA, Canada and New Zealand as well as Israel, Iran, India, Korea and Japan. The rise in the number of delegates reflects the growing European concern about invasive alien species (IAS) which are considered to be the second most important factor in loss of biodiversity globally after habitat destruction. The Neobiota meetings provide the

15th International Cocoa Research Conference (15ICRC), San Jose, Costa Rica.

Over 250 delegates in diverse disciplines such as research, marketing, industry and media, and from several different countries all over the world attended the conference. More than 100 oral presentations and a similar number of posters were delivered and translated simultaneously into four languages, English, French, Spanish and Portuguese. The speeches were similarly diverse and varied in scope from Cocoa Agro-forestry – encompassing cultural systems, soils, nutrients and physiology; through Genetics – involving quantitative and molecular processes; Crop Protection – comprising phytopathology, entomology, nematodes and rodents problems; Chemistry, Technology & Quality Control; Cocoa by Products Utilisation and New Uses for Cocoa; Environment, Socio Economics and Technology Transfers and Utilisation to a Round Table Discussion on chocolate and human health.

Despite the theme for the conference "Cocoa productivity, quality, profitability, human health and the environment", disease and pest issues of cocoa production once again dominated discussions at the conference. Pod rot diseases and pod damage by insects and rodents were common phenomena in all the cocoa growing regions of the world, i.e., West and Central Africa, Central and South Americas and the South East Asians. Listening to reports on frosty pod rot (caused by *Monliophthora roreri*) and witches broom (by *Crinipellis perniciosa*) from the Americas was a very sobering and frightening experience. There seems to be no solution to these diseases despite the huge efforts being made to find some cure or control. Efforts being made include the search for resistant materials, bio-control agents and fungicides, yet none had been successful.

On the other hand, these diseases continue to spread and ravage cocoa unceasingly. An account by Dr. Wilbert Phillips-Moran on the spread in space and time of frosty pod from Panama to Mexico was the most unsettling part of the debate. Both frosty pod and witches broom are believed to have co-evolved with cocoa and are endemic to the Americas. – To avoid any accidental transport of the pathogens to Central and West Africa, delegates from these countries were advice not to make any field trips to affected areas!

In West and Central Africa, pod rot disease caused by *Phytophthora megakarya* was the predominant and most devastating pod disease, fortunately, control strategies of breeding for resistance, bio-control and fungicides use seems to be working effectively. *C*acao swollen shoot virus (CSSV) disease, an old but still prevalent and devastating disease of cocoa continues to decimate cocoa fields. The disease has completely annihilated cocoa fields in Togo, whiles Ghana continues to struggle with it. Now, Cote D'Ivoire, the world's largest producer of cocoa has just declared the devastating effect of the disease in their most productive cocoa fields at the 15ICRC. Ironically, there were only three papers on this disease, two of which I presented.

The host: Resistance gene isolation and realising the potential

Kim Hammond-Kosack, *Rothamsted Research*

Most plants are resistant to most pathogens. This is because plants have evolved sophisticated systems for the recognition of non-self that in turn leads to the activation of both local and systemic plant defence responses. Over the past twelve years many novel genes, proteins and molecules have been discovered as a result of investigating both compatible and incompatible plant-pathogen interactions. We now recognise that both interaction outcomes involve dramatic cellular reprogramming events in plant tissues and that some parallels exist between plant defence and the animal innate immune response.

This presentation will focus on the different classes of resistance genes, how they were isolated, how they are thought to function in pathogen perception and defence activation and how novel functionalities may be evolving at resistance gene loci. Many fundamental questions remain stubbornly unanswered. Where research breakthroughs are urgently required these will be pin-pointed and the value of post-genomics approaches reviewed.

Most of the knowledge gained on resistance genes / proteins and the defence responses activated arose from investigations on model plant-pathogen interactions. Unfortunately most attempts to harness this new knowledge to engineer improved disease resistance in crops have so far failed even though good gene efficacy has been shown. Currently underway is a shift in emphasis towards strategies to enhance marker-assisted breeding and the use of vectors containing highly regulated transgenes that confer resistance in several distinct ways.

Acknowledgements
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Public understanding of plant pathology; what do the public know and what shapes what they know?

Tony Gilland, *Institute of Ideas, London*

"As the earth's population expands, and global climate changes, increasing demands are made on our limited cropping area. Ever present pest and pathogen populations continue to cause serious crop losses and, on a world scale, crop protection remains one of man's principal challenges."
The BSPP and Education, A Career in Plant Pathology?, The British Society for Plant Pathology web site.

However important the work of plant pathologists may be it seems fair to say that the intricacies of plant pathology are not high in the public's mind. Whilst many a keen gardener may be familiar with some of the challenges posed by an array of plant pathogens, and some of the options available to combat them, the science of plant pathology is simply not a major issue of public concern. That said, what has become a far greater issue of public concern is the way we farm our food. From pesticide residues to genetic modification and organic farming, debates have raged across the media and elsewhere about this issue and have undeniably had an impact on public perceptions of what science has to contribute to improving the way we grow our crops. How plant pathologists should respond to this situation is an important question for debate.

In addressing this question this paper will focus on two key problems. First, the drawbacks of our media dominated society where whatever does or does not get reported in the pages of the national papers is seen to have a defining impact on public life. Whilst the power of the media is important to recognise there is a real danger of underestimating the wide range of factors that shape public debate – not least of which is the clarity and sense of purpose that any community of professionals has about their work. This leads to the second key problem, defensiveness. Scientists are constantly being berated about the need to engage the public with evermore prescriptive guidance about the best way to do this. But the bigger problem is not so much how to communicate but what. Science has been on the back foot for far too long. The way to address this is not to worry about what the public does or does not know or think, but to insist firmly on the significant contribution science has to make to society and to alter the terms of the debate – at least that way 'the public' stand a fair chance of hearing more balanced and genuine debates about the issues from which they can make up their own minds.

communication of scientific research to the public. The traditional theme of pathology in relation to the production of alcoholic beverages was incorporated, and was followed by beer and wine tasting for all the delegates. This year, SGM generously provided a prize for the best talk given by a doctoral student. This was awarded to Michael Ravensdale, SCRI, whose work is described above. The runners up in the competition, with "workshop" awards, were Lauren Ryder (Exeter) and Mary Illes (Oxford), who work on *Trichoderma hamatum* and *Magnaporthe grisea* respectively.

We would like to thank the organisers of this year's conference, John Jones (SCRI) and Chris Thornton (Exeter) for arranging a smooth-running programme, and the chairs of each of the sessions for such stimulating discussion. Generous sponsorship of the meeting was provided by The Gatsby Charitable Foundation, The British Society for Plant Pathology, The Society for General Microbiology, The Society for Applied Microbiology, Syngenta, Cogeme and The British Mycological Society.

Mary Illes and Solange A. Mateo Montalcini

Conference delegates, photo by Sarah Gurr

via the application of exogenous cAMP, suggesting that CUTE also has a role in perception and signal transduction.

In his talk Michael Ravensdale (SCRI) suggested that considering *Erwinia carotovora* subsp *atroseptica* solely a "brute force" pathogen might be an oversimplification and introduce several aspects of the "stealth" interaction *Eca* is believed to have with the host. A gene cluster similar to that encoding the phytotoxin coronatine in *Pseudomonas syringae* has recently been identified in *Eca*. Despite lacking some of the genes thought to be involved in the cluster's regulation and phytotoxin biosynthesis, coronafacic acid mutants showed reduced virulence and were readily rescued via expression of the native genes *in-trans.* Furthermore, regulation of the gene cluster appears to involve quorum sensing.

The second day culminated with an excellent light-hearted lecture given by Chris Ridout, "Beer today champagne tomorrow", which nevertheless delivered a poignant message about the importance of

Student talk prize winners; from left: Mary Illes, Michael Ravensdale and Lauren Ryder, Photo by Sonia Humphris

Foresight: plant pathology in a global disease context

Jeff Waage, *Imperial College, London*

The UK Office of Science and Innovation has completed this year a Foresight project on Detection and Identification of Infectious Diseases. This 18 month study considered the future risks of infectious disease *across* human, animal and plant sectors. The study was focused on both the UK and sub-Saharan Africa. Across these sectors and regions, experts identified the same three future risks as priorities: new pathogens/strains arising through natural genetic change, geographical extension of pathogen range and increased pathogen resistance to microbiocides.

Parallel to risk studies, the project considered future scientific advances affecting our capacity to detect, identify and monitor infectious diseases. Four areas of technology were seen to converge in future on systems for rapid, pre-symptomatic disease monitoring: gene technology, sensing technology, electronic miniaturization and information technology. In UK and Africa, these future visions of risk and technology were put to practitioners responsible for disease prevention and management, and four "systems" emerged for future development: novel information technology for capture and analysis of disease-related data; tools to detect and characterize new diseases based on genomics and postgenomics; hand held point-of-care devices for rapid disease diagnosis; and high throughput, non-invasive disease detection systems for use in ports, airports. Throughout this project, plant disease perspectives were integrated with those on animal and human diseases. As plant diseases hold a comparatively lesser place in political priorities for infectious disease, such engagement with advances in human and animal disease diagnosis and prevention should be of future value.

A message from the first president — notes on the origin of the BSPP

At the International Congress of Botany, Edinburgh, 1964, I met again W C Snyder (Berkeley, California, where I had been a Commonwealth Fund Fellow 1950-51), and J G Horsfall (New Haven, Connecticut, where I had been a Research Fellow 1956-57). Over a few drinks we agreed that plant pathology had been represented poorly at the Congress. Somewhat impetuously I proposed that we should have our own Congress. This was received with some enthusiasm by WCS and JGH and then by others, especially S D Garrett and P H Gregory. One thing led to another and, as they say, the rest is history, well recorded in the PAM, Vol 48, p 225-253.

Now I had been Secretary of the Association of Applied Biologists (AAB) for some years and on the Plant Diseases Committee (as Chairman, I believe) of the British Mycological Society (BMS). By 1964 I had recognised the anomaly that plant pathology was no more than a part of AAB and only a minor part of the BMS. Encouraged by the response to my idea of a new Congress I also proposed an independent society for plant pathology for the UK. This, too, was well received by a group of UK plant pathologists at Edinburgh, who then commissioned me to develop the proposal. The AAB and BMS soon learned about it and at once strongly resisted the formation of a new and independent society for plant pathology in the UK. They appointed a committee to recommend an alternative with me as Chairman. The outcome was a weak compromise, the Federation of British Plant Pathologists to be financed by subventions from the AAB and the BMS. I soon realised that I should not have accepted the Chairmanship and the recommendations of the Committee. I should have resisted the AAB and BMS and continued to fight, albeit forlornly, for an independent society.

Thereafter, I had little to do with the Federation. Instead I occupied myself with organising the First International Congress of Plant Pathology, 1968, at Imperial College, magnificently supported by Bryan Wheeler as Treasurer and (hedge fund manager manqué!), June Cheston, my secretary. And later as Founder President of the International Society for Plant Pathology, again superbly supported by the Founder Secretary, J G ten Honten, a leader of Plant Pathology in the Netherlands. I did not dwell too much on my failure in the UK. However, some years into the reign of the Federation I learned that a small group of younger plant pathologists were on the way to establishing an independent society. I found ways of assuring the group of my total support but I warned it not to associate in any way my name with their efforts. These were rewarded with success on a scale that I might have hoped for but with no chance of achieving. I contributed nothing to their success. Their diligence and that of many others who followed, has given us BSPP with its highest standard of professional science, a leading international journal, administrative efficiency and financial probity, and a high position among the best of the national societies for plant pathology. And there it will remain.

PhD student Elise Lambeth (University of Exeter) is investigating the NADPH oxidase 3 gene (*NOX3*) in *Magnaporthe grisea*, building on previous work by Martin Egan. The group have already found that Δ*nox1,* Δ*nox2* and Δ*nox1*Δ*nox2* mutants cannot infect plant tissue and cause disease symptoms, although appressoria are formed normally. Future work will focus on characterising the *NOX3* gene and identifying a possible regulator of the *NOX* gene family. Diane Saunders (University of Exeter) is building on previous work by Claire Veneault-Fourrey, which focuses on the role of specific cell cycle check-points in the appressorium development of *Magnaporthe grisea*. Treatment with hydroxyurea at different stages of the cell cycle suggests that nuclear divisions within 6 hours of inoculation are key for appressoria development.

Pari Skamnioti, University of Oxford, discussed the role of cutinases in *Magnaporthe grisea*. *M. grisea* has traditionally been considered to penetrate host leaves via mechanical force, yet 16 cutinase genes have been identified in its genome. *CUTE*, which is upregulated during leafbuilding penetration, was knocked out resulting in reduced pathogenicity and the formation of multiple, aberrant appressoria on inductive surfaces. *ΔcutE* was readily rescued

View of Birnam Woods, photo by Sonia Humphris

considerably and be considerably more sustainable - buton this aspect there was no meeting of minds. Where there was more consensus was on the need for sustainability in future farming of whatever colour – when the oil eventually runs out where will we get our feedstock for nitrate production, for example?

A lively ceilidh after the Conference Dinner was participated in enthusiastically by delegates irrespective of philosophical, theological or scientific persuasion. All in all it was a most stimulating conference. Was I any wiser at the end? I was certainly not convinced that if all conventional farmers suddenly saw the light and became organic that it would solve world hunger, but there is also no doubt in my mind that we will have to learn to farm in a more sustainable manner and there are certainly things that the conventional sector can learn from the organic sector.

I am very grateful to the BSPP for their travel grant. Papers on which both talks and posters were based are published in *Aspects of Applied Biology* **79**, available from the AAB.

Peter Mercer
Applied Plant Science Division, Agri-Food & Biosciences Institute, Belfast

Molecular Biology of Plant Pathogens (MBPP) 2006, Birnam 18-20th September

Seventy scientists, drawn from 12 research groups met in Birnam, Perthshire, for the 2006 MBPP annual meeting. There were 30 research talks given, principally by doctorate students and post doctorates. Research on *Magnaporthe, Erwinia, Phytophthora* and nematodes featured heavily in the talks this year, and presentations we found particularly interesting are outlined below.

David Cooke from SCRI described the work of the Late Potato Blight Network for Europe, Eucablight. This is a collaborative effort that has established a database (www.eucablight.org, hosted at DIAS in Denmark) to store simple sequence repeats (SSRs) in order to track *Phytophthora infestans* population diversity across Europe. Currently, host, pathogen and SSR information has been entered for 13000 *P. infestans* isolates. As further data is acquired, it is hoped factors driving population change will become more evident. There are plans to extend the database to include the US and South America. One trend that has already emerged is the spread of the A2 mating type of *P. infestans* westwards through Europe, where historically the A1 mating type has dominated. This will have implications for disease control, as A2 resistance to metalaxyl is increasing.

A final and reflective note. An independent UK society was conceived at Edinburgh in 1968. There was no birth. Thirty years later the seventh and one of the most successful of our International Congresses was held in Edinburgh, in 1998. It was superbly and felicitously organised by BSPP.

R K S Wood

THE BSPP: A RETROSPECTIVE

The British Society of Plant Pathology was founded on 8 April 1981, with charitable status. Prior to its creation, the interests of British plant pathologists were catered for by the Federation of British Plant Pathologists (created jointly by the British Mycological Society and Association of Applied Biologists in 1966). With its formation, the new society entered into a publishing agreement with Blackwells to edit *Plant Pathology*, formerly owned by the Ministry of Agriculture, Fisheries and Food. The Society started with eighty-nine Founder Members. On 5 June 1998, the Society became a company limited by guarantee and on 10 September of the same year the company became a registered charity, this time with about 600 members from over 54 countries.

Some of the key events in the history of the Society included the purchase of *Plant Pathology* from Blackwells on 1 January 1987; the publication in 2000 of a new Web-based publication, *New Disease Reports*. This replaced the popular "New and unusual disease records", which ceased to be part of the journal in 1994. 2000 also saw the publication of *Molecular Plant Pathology,* initially trialled as a web-based publication in the mid-1990s, but as a venture that was probably ahead of its time. *Molecular Plant Pathology* now has an impact factor of 3.3, with *Plant Pathology* at 1.7. Both excellent achievements, brought about through the hard work of their respective editors and editorial boards, keen to maintain high scientific standards in the papers published.

The Society made an unsuccessful bid to host the 1993 International Congress of Plant Pathology, which went to Canada. However, the experience gained lead to success with our bid for the 7th International Congress. This was held in Edinburgh in 1998, where we played host to about 2500 scientists and accompanied persons. It was the start of the era of web-based registration and on-line submissions, which gave the organising committee and our professional conference organisers some major headaches, but we came through relatively unscathed. The Congress was opened by the Princess Royal and, despite being concurrent with the Festival Fringe and the Edinburgh Military Tattoo, the scientific programme retained the vast majority of delegates in the conference centres!

The ISPP meets the ICPP98 at the RBGE

The Princess Royal greeted by David Ingram at ICPP98

The Society is still in its infancy, compared with the Association of Applied Biologists (1904), the British Mycological Society (1896) and even the American Phytopathological Society (1908), but it has come of age and could be said to have matured well beyond its years. It now provides its members with two journals, a newsletter, scientific meetings, a members' database and an active web-site. The Society supports members by providing funds for attendance at conferences in the UK or overseas; study visits abroad; visiting fellowships for members to undertake study at another institute; student vacation and MSc bursaries for research projects in appropriate laboratories and a promotion fund to support new initiatives with potential to further the promotion of plant pathology in the UK and elsewhere. All this has been achieved through an ever-changing group of dedicated members that constitute the Board.

Nigel Hardwick

the Soil Association at one end of the spectrum where organic agriculture is an article of faith to people such as myself at the other, who see organic agriculture as a niche market, worthy of commercial exploitation, although possibly also teaching us something about sustainability.

The Keynote talk was given by Rev Dr. D. Atkinson, one time employee of SAC and now translated to the cloth in St. Andrews Cathedral, Aberdeen. Although I myself am an ordained minister of the Methodist Church I have never given a scientific talk in clerical garb, but it was certainly an interesting start to the Conference, with an overview of the current opportunities for organic farming in relation to the present state of farming economics and the current expectations of society, considering both food production and the delivery of environmental services paid for from the public purse. In the question time afterwards I suggested that it might be a good idea to promote the environmental benefits of organic farming rather than the apparent food quality benefits, which generally lacked any scientific basis.

The result of this intervention was that in the coffee break I was waylaid by a very animated young lady from the Soil Association who tried to put me right. I have to admit, however, that after half an hour of argument I remained unconvinced.

Most of the talks were twenty-minute presentations and were generally well put together and delivered. The subject matter ranged from the dispassionate "Strategies for profitability: product, management and misinformation" by Gareth Edwards-Jones of The University of Bangor, to the experimental "Evolutionary breeding of healthy wheat from plot to farm" by a team under Martin Wolfe of Elm Farm Research, to the practical "Constraints to the sustainability of a stockless arable rotation" by Bill Cormack of ADAS Terington, to the observational "Behaviour of dairy cows on organic and non-organic farms" from a team at SAC, Edinburgh – apparently, rather counter-intuitively, the organic cows are a much more badly behaved crowd than the non-organic, possibly because they are hungrier.

There was a large number of posters which were arranged in groups and each participant was invited to give a very short talk – this was possibly the least satisfactory part of the Conference as the venue was cluttered and noisy and it was hard to see the poster and hear the speaker at the same time.

There were a couple of visits to organic farms on one of the afternoons. These were very informative and practical and showed just how difficult and costly organic farming can be at the sharp end *e.g.* literally burning down potato tops.

On the first evening there was a public debate on "Can organic farming take the lead in a post fossil carbon society?" Perhaps there was more heat than light in the discussion, especially as the debate got somewhat sidetracked into an argument on GM in organic farming – never could understand the opposition of the organic movement to this technology which has the potential to cut pesticide use

Alabouvette described the production and formulation of biocontrol agents, post release, control and risk assessment and Warrior Prem from Valent BioSciences, the commercial perspectives of biorational pesticides. Now owned by the Sumitomo Chemical Company, formerly part of Abbott Laboratories, this company launched *Bacillus thuringiensis* (Bt) as well as other products. Session 8 covered non-target effects with Linda Thomashow describing the effect of genetically modified bacterial biocontrol agents on the fungal rhizosphere microflora and John Whipps the effects of *Coniothyrium minitans* on microbial communities following introduction into soil. In the final Session 9, on monitoring biosafety, Brian Federici covered the scientific basis and data for the safety of transgenic crops to humans and other organisms – so far no major problems.

It is a pity that a lack of space does not allow me to describe all the presentations as many of those on the biocontrol of animal and weed pests were highly relevant to our understanding of biocontrol prompting a cross fertilization of ideas, as well as a salutary warning of what can go wrong, for example when cane toads were unwisely introduced to Australia to eat beetles they could not reach in the sugarcane!

Roland Fox, The University of Reading

Association of Applied Biologists Conference, Edinburgh : What will organic farming deliver?

Edinburgh, September 2006
Although I have been working on the production of organic cereals for about four years, and have attended various COST Action and focus group meetings, this was the first scientific conference on the subject at which I was present. I was really looking forward the occasion to see what group of people would be assembled, as organic agriculture (as against "inorganic" agriculture?) is a subject that causes many a raised eyebrow and disapproving look from scientists, particularly from those of us who have worked in the conventional sector for many years. However, it is clear that there are pressures on us to become more "sustainable" – indeed I often summarise my 25 years with the Department of Agriculture in N. Ireland as saying my first 15 years were spent telling farmers to apply more fungicides to their cereals and the last ten years telling them to stop! But is organic agriculture in all its fullness the answer? Would I be any wiser by the end of the Conference?

Those attending the Conference came from a variety of backgrounds from

25 years for BSPP is an achievement to be proud of — from your current president, Peter Mills.

Twenty five years for BSPP is an achievement to be proud of. Following a somewhat awkward and stressful birth (see page) the Society has grown to become one of the strongest plant pathology societies in the world representing and supporting pathologists from more than 50 countries. But what have we achieved and what has changed since those exciting early days in 1981?

Looking backwards

From a research perspective, the early 1980s were an exciting time with PCs appearing on our desks for the first time (how did we manage before that? – 'out of office' was a note sellotaped to your door!), recombinant DNA technologies were being used effectively (we all became expert bacteriologists and learned how to handle plasmids) and perhaps arguably the most important leap forward was the development of PCR (never had we had so much DNA to play with). As with many disciplines in biology, the impact of all of these technologies on plant pathology research has been outstanding. Sensitive pathogen detection and discrimination, the molecular basis of host/pathogen interactions, mapping and characterisation of resistance genes, ecology, population biology and forecasting have all developed from 'jam tomorrow' to reality. We've all been a part of these developments and similar ones in one way or another. It would be foolish of me to attempt to name those who have taken forward our discipline over the last 25 years but the speakers on the programme for our Anniversary meeting represent a tiny proportion of the leaders in their field within our Society. The programme also demonstrates the breadth of expertise within the Society and it would be hard to identify any topic where a BSPP member is not making a significant contribution. This brings me neatly on to the subject of skills and strength within the discipline.

Where do we stand today?

There is a concern voiced fairly frequently that training and job opportunities are declining and that by extrapolation we will soon face an era where Universities will no longer teach plant pathology and that the ability of students of the subject to recognise a disease will be lost. I'm not sure that I totally subscribe to this analysis. The Society certainly acknowledges these concerns and we are currently attempting, as one would expect of well trained scientists, to collect data that will test the hypothesis. There is conflicting data available at present. On the one hand, there appear to be fewer University Departments offering plant pathology as an option in the third year of an undergraduate degree course and as we know there have been redundancies in UK research organisations resulting in job losses in our discipline. To counter this, the membership numbers within BSPP seem to be holding up well. As technologies, funding body requirements and fashions change, the scientific 'label' that people give themselves change also. I'm convinced that there are many plant pathologists currently working under the labels of molecular biologist, environmental biologist or biotechnologist whose work is still delivering improvements in pathology.

For those who have been involved in research for the last 25 years it is obvious that there is a cycle in scientific fashion within the main sponsors of research. As a snap-shot of the situation in 2006 it is worth recording that research on the pathology of crop plants is less well supported than has been the case in previous decades. Support is shifting towards environmental issues and model species. Although many of our members may have views on this strategy the reality is that our skills as pathologists are still being employed. We are still progressing the science of plant pathology and making contributions at national and international levels.

Where's it all going?

So what about the next 25 years? Well I'm reluctant to fall into the trap of predicting the future (I don't want this article to be reproduced in 2031 and bench-marked against reality) - but I'm going to anyway. I think that I'm on relatively safe ground to say that some of our challenges over the next 25 years are fairly self-evident. Climate change is a massive opportunity to us as a discipline. Not only new pathogens and food crops to get to grips with but also the prospect of huge increases in biomass and alternative crops all of which will need our skills to allow them to be grown efficiently. Who, for example, amongst our current group of post-graduate students will want to be the Director of the Alternative Bio fuels Research Institute in, say, Oxford in 2031? Global food markets will bring exotic pathogens to our doorstep and as we look more closely at the relationship between natural and agro ecosystems we will find interesting new systems to study. Will chemical control measures still be needed and will the legislative authorities be disbanded or, more likely, will they be spending all their time dealing with biological control agents? But above all the priority over the next 25 years surely must be to exploit the increasing amount of information being generated from genome sequencing projects of both host plants and pathogens. This *has* to deliver robust disease resistance in major crop plants. Looking back at where we started in 1981 and where we are now I am convinced that we will easily achieve that - and much more.

What has the Society done for you?

There are some very tangible outputs from 25 years of BSPP. We started our journey in 1981 with *Plant Pathology*, a minor journal that has been transformed by a succession of Editors into one of *the* major pathology journals generating substantial income that fuels the Society's activities. To add to this, we have created a sister journal *Molecular Plant Pathology* and between them our two journals rival the very best plant pathology journals anywhere in the world. Our income has been spent on sponsoring at least 100 scientific conferences (including hosting a very successful Congress in Edinburgh) and, at a rough estimate, funding 1000 plant pathologists to attend conferences at home and abroad. We have funded Fellowships for senior researchers but more critically sponsored large numbers of student bursaries to encourage young people into our profession. We should be immensely proud of what we have achieved over the last 25 years and acknowledge the foresight of those who were bold enough to get us started on the journey.It is inconceivable that the skills we have will ever be superfluous to plant production and the Society's network of members across the world will enhance our ability to deal with many of the issues mentioned in this article. I look forward to being part of the next 25 years of BSPP!

NATO Advanced Study Institute Workshop on Novel biotechnologies for biocontrol agent enhancement and management.

Organised by Jonathan Gressel (Professor Emeritus of Plant Sciences (Plant Sciences Weizmann Institute of Science, Rehovot, Israel) and Maurizio Vurro (Istituto di Scienze delle Produzioni Alimentari, Bari – Italy) held at Gualdo Tadino (Perugia) Umbria, Italy, 8-19 September 2006. As this was the first NATO ASI Workshop that I had attended, I wondered what to expect. In particular were these NATO workshops run in a formal almost military fashion in some bleak remote camp? Fortunately this was far from the case and the tenor of this meeting was extremely jovial and relaxed in a delightful medieval stone farm house on an idyllic estate. The only minor disparity between the established expert "Teachers" and the "Students" was largely confined to question time. Jonny Gressel enlivened the meeting with many of his apparently inexhaustible collection of largely appropriate cartoons and David Sands read some of his equally thoughtful and apposite poems.

Like many people I previously considered NATO to be limited to the North Atlantic countries. While the lecturers were predominately Westerners, there were more participants from Russia and the Ukraine than the UK. Parts of Africa and Asia were also well represented.

The main theme of this workshop was "Why we need enhanced biocontrol". In the first session "Who is being controlled by whom?", Gary Harman gave a stimulating review of the current uses and capabilities of fungal biocontrol agents, with an emphasis on *Tríchoderma* spp. while others reviewed the situations for weed, insect and rabbit control. The second session ably chaired by Ray St Leger concentrated on "Finding and utilizing weaknesses in the pests". For pathogen biocontrol, Claude Alabouvette followed by F. L'Haridon and S. Moretti covered 'The biocontrol capacity of *Fusarium oxysporum*', what do we know today?

Matteo Lorito discussed the molecules activating and stimulating biocontrol process in *Trichoderma* and other beneficial fungi. There were a number of practical sessions, for example Session 3 covered mixing organisms with organisms or products in which I revealed our success in controlling *Armillaria mellea* with a combination of a *Trichoderma* spp. with fosetyl-Al. Session 4 covered choice of biocontrol agents, genetics and enhancement. Session 5 covered finding genes to enhance biocontrol – using omics.

Session 6 covered engineering enhanced biocontrol in which Angharad Gatehouse discussed the role of biotechnology in crop protection and Kathryn Brocklehurst outlined protein science at Syngenta. Session 7 covered getting agents to work in the field, where Linda Thomashow described the detection of antibiotics produced by soil and rhizosphere microbes in situ, Claude

Disease spread mostly occurs in areas contiguous to infected areas – connectivity between forest patches was seen as important in facilitating disease spread. However, long distance spread initiating new infections has occurred in California. He said that identification of the circumstances under which this take place would be key in managing large scale spread of the disease.

Population genetics

Apart from the key paper presented by Kelly Ivors (discussed above), Nik Grunwald and co-authors discussed the independent assortment of alleles in the three clonal lineages of Pr using SNP (single nucleotide polymorphism) microarray data and suggested that sex was occurring at the putative centres) of origin of Pr.

Additional items of interest

New Fungal Databases: Erica Cline talked about the development of new fungal databases which provide information on nomenclature (including synonyms), distribution, substrate or hosts, supporting literature etc. *Phytophthora* was one of the genera available on the database at http://nt.ars-grin.gov/fungaldatabases.

Forestry field trip and diseases of interest to the UK: On the forest field trip we were shown a number of interesting diseases and tree resistance breeding programmes. *Septoria* leaf spot and canker disease is an European Commission (EC) quarantine listed disease (i.e. not yet found in Europe) caused by *S. musiva* (teleomorph: *Mycosphaerella populorum*). Stem breakage causes tree losses in plantations in Canada. The leaf spot phase has also recently been found on *Salix lucida* spp. *lucida*. *Cytospora* sp. and *Phomopsis* sp. are secondary invaders colonising cankers and overtaking the primary pathogen masking its presence.

A new disease in Québec was Mycosphaerella needle cast of larch. Discovered in 2003, disease severity was high on European larch (*Larix decidua*) and Japanese larch (*Larix kaempferi*). The causal organism is *Mycosphaerella laricina* (anamorph: *Pseudocercospora* sp.) which has not yet been recorded in the UK. Initial symptoms of the disease were yellowing of the needles following by needle necrosis, defoliation and tree death. Frequently *Cytospora* sp. acted as a secondary invader of lower branches of infected trees and caused stem cankers.

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Sandra Denman, Forest Research

25 years of plant pathogen interactions

- from your next president, Richard Cooper

The author 25 years ago.

In 1981 I went on sabbatical in the USA to work on fireblight, *Erwinia amylovora*. This was my first hands-on experience with a bacterial pathogen and I soon realised why the wealth of information at that time on interactions had come from fungal diseases. In short one could track fungal infections by light microscopy, antimicrobial compounds from plants were generally more active against fungi, spores as inocula were easily counted and fungi seemed more amenable to bioassays. Now, thanks to molecular genetic techniques with bacteria, the situation is reversed, but not entirely. I think it would surprise many current molecular-trained and -driven young scientists how much was known twenty five years ago. That knowledge of the biochemistry, physiology and ultrastructure of interactions still provides some of the basis for teaching, but major gaps there certainly were, as I will attempt to outline. Much of the contrast can be found by comparing the state of play in two multiauthored textbooks: Biochemical Plant Pathology 1983 edited by J. A Callow (Wiley) and Plant Pathogen Interactions 2004 edited by N. J. Talbot (Blackwell). I will attempt to bring the subject more or less up to date, but it is not meant to be comprehensive. Some key references of more recent work are included in the text for those that may wish to explore more deeply.

What was known then?

Host defences centred on the hypersensitive response (HR) for which there was excellent information on ultrastructure, sequence and timing of pathogen inhibition, and associated triggering and localisation of phytoalexins. The long list of phytoalexins from most families, their chemistry and toxicity was being added to with preformed compounds or phytoanticipins. Physical changes linked with defence were largely cell wall based. Pathogenesis-related (PR) proteins had been found in viral diseases but functions were unknown. Non-host resistance was ignored by most as too obscure; we knew little enough above host resistance based on major genes, let alone that conferred by minor genes, and in the "wrong" host. Triggers of defence were well recognised as elicitors from fungi, such as glucan and chitin oligomers and LPS from bacteria. No serious claims for race-specific triggering of HR existed.

Pathogenicity was well represented with extensive information on toxins. Much was available for their chemistry, role and in a few cases, mode of action. Classical genetic information on host and pathogen underlined some Cochliobolus toxins as prerequisite for disease and rare explanations of specificity. Barrier-degrading enzymes (cutinases, pectinases, hemicellulases and cellulases) were well characterised and seemed to be involved in many diverse diseases involving necrotrophs and some hemibiotrophs. The highly destructive action of pectin hydrolases and lyases on primary walls along with their ability to kill plant cells, justifiably, gave them the spotlight. This mechanism is perhaps the most common means of cell killing by necrotrophs, but remarkably, is still unknown. Later we implicated fungal xylanases rather than pectinases in pathogenicity to graminaceous monocots as an adaptation to the xylan-rich primary wall matrix of cereals.

Bacterial extracellular polysaccharides (EPS) also appeared to be key in some diseases. Siderophores and phytohormones were under investigation. Fungal biotrophy was well described ultrastructurally, such as the haustorial-host membrane interface, and in terms of nutrient uptake following the isolation of haustorial complexes and analysis of carbohydrates therein. Suppressors of recognition or of host defences were anticipated, but not identified. In spite of all this wealth of information, the bottom line of genetic proof was mostly lacking. Evidence often seemed overwhelming, yet remained circumstantial.

Host physiology. Much was known about the deleterious effects of disease on respiration, photosynthesis, phytohormone balance, nitrogen metabolism and water stress. It was realised eventually however that such information was not going to reveal the early critical events in interactions that we were seeking. These changes largely reflected downstream cascade effects. Efforts to study total biochemistry were thankfully waning. One might bear this in mind with regard to the current upsurge in metabolomics.

What we did not know then?

Host-pathogen specificity was a black box (other than for a handful of interactions involving host-selective toxins, for which host-pathogen genetics does not fit the usual pattern for most diseases). How are pathogens recognised (or not) and by what? Speculation included claims for differential synthesis or release of elicitors and existence of cultivar-specific elicitors. According to transmission electron microscopy bacteria bound to cell walls and there followed claims for specific binding involving host lectins and pathogen oligosaccharides. The binding later appeared to be artefactual as infiltrated water from bacterial suspensions is withdrawn from the apoplast. Resistance genes were being used to study the expression of defences, but this told us little of the early critical events. Pathogen avirulence genes clearly existed, but why and what were they? How did they interact with products of R genes? There were many models proposed but no molecules. With hindsight, we had no facile model systems to study the bigger picture. We knew little or nothing of the genes behind pathogen virulence and host defences. Also signalling molecules and pathways in hosts and pathogens were another barren area.

So what's new and why?

Transposons, cloning, site directed mutagenesis. Around this time, molecular genetics was established in model bacterial systems and being adapted for plant pathogens. Transposons with selectable markers were being used to induce mutations in *Rhizobium* and *Pseudomonas syringae*, and DNA cloning systems were becoming routine. Labs such as Staskawicz's started the search for avirulence and virulence genes. Perhaps the first example of a bacterial virulence factor came from Comai and Kosuge in 1982 cloning tryptophan 2-monoxygenase and introducing this into a plasmid-cured IAA minus strains of *P. syringae* pv *savastanoi* to restore virulence (but is was not essential for infection, just degree of symptoms). Many groups in the 1980s such as labs of Keen and Collmer investigated the multiple pectinases of soft rot erwinias with their relatively simple *E. coli*-type genetics. This was only possible with site-directed mutagenesis to remove stepwise the 4-6 genes to evaluate the individual or combined roles of the iso-enzymes. Here hangs a tale: pathogens turned out to be far more versatile and complex than might

indeed a quandary for plant health regulators. Furthermore, this discovery challenges our thoughts on the ecological relationship between these Phytophthoras and their hosts. It is important to elucidate the biological factors influencing the asymptomatic condition and environmental circumstances under which these asymptomatic infections and sporulation events occur, to better detect and manage disease. Information on the incubation period of these pathogens is also vital to understanding the asymptomatic condition.

A number of other new developments on the biology, epidemiology and population genetics of *P. ramorum* were also presented at the meeting.

Biology and epidemiology

Interesting work on germination of- and root infection by- *P. ramorum* chlamydospores was presented in posters and talks. Chlamydospores are formed in infected plant tissues (leaves) and are released into soil upon decay of leaf debris. Evidence from two laboratories showed that Pr chlamydospores germinate by producing sporangia (Fitchner *et al*. and Shishkoff *et al*.). This is an important contribution to our understanding of the behaviour of the different propagules in the life cycle of Pr and has considerable implications for disease epidemiology in different environments. The production of sporangia from chlamydospores in leaf litter is not only relevant to spread in forest situations but also to the nursery industry. Water running off infested leaf litter and contaminated potting mix could also be a source of zoospore inoculum especially if untreated re-circulated water is used.

According to Glenn Colburn roots of rhododendron planted in soils infested with chlamydospores became infected (presumably by zoospores released from sporangia developing from chlamydospores). Nina Shishkoff showed that with Pr higher levels of infection of viburnum roots occurred at a lower temperature (10°C) than at high temperatures (15–25°C). Similarly sporulation was more abundant at 10 and 15°C decreasing to negligible at 25°C. The significance of root infection needs to be fully elucidated but it might be important in disseminating this pathogen through the nursery trade.

A number of posters presented by Jennifer Parke detailed the occurrence of Pr in the sapwood (xylem) of tan oaks and demonstrated that the sap flow in infected trees was reduced. In the UK Brown and Brasier (2006) have recently demonstrated colonisation of secondary xylem on trees infected with *P. ramorum*, *P. kernoviae*, *P. cambivora*, *P. citricola*, *P. cinnamomi* and other *Phytophthora* spp. Using scanning electron microscopy Parke *et al*. showed hyphae, chlamydospores and tyloses in the xylem vessels of infected tanoaks. These results may have consequences for the movement of wood and wood products from Pr and Pk infested areas if it can be shown that chlamydospores or hyphae in wood remain viable and pose a threat in any way.

In monitoring and predicting the spread of the epidemic in the USA at the landscape level, Ross Meentemyer discussed the mathematical models he used to predict high-risk areas of native forest not yet infected in California.

In the UK so far thousands of rhododendrons, about 60 beech trees and English oaks (*Quercus robur*) have been infected. The pathogen penetrates the dead suberised outer bark layers and attacks the inner bark (phloem), cambium and even the xylem as recently shown by Brown and Brasier (2006). The resulting stem necroses force tree fluids, known as tarry exudates, to ooze through to the stem to the exterior, a condition known as bleeding cankers. Apart from beech trees and rhododendrons members of the Magnoliaceae are also attacked, the leaves, flowers and fruits become blotched and necrotic. At some heritage sites in England valuable species of the National Magnolia Collection could be under threat by Pk. This pathogen has only been found in two nurseries in the UK and until 2006 it was only found in England and Wales, but has now also been found at two sites in New Zealand (http://www.maf.govt.nz/mafnet/press/240306fungus.htm).

The biology and epidemiology of both these Phytophthoras is complex. There are many similarities between the two organisms but also a number of differences. *P. ramorum* is heterothallic and there are clear genotypic and phenotypic differences in the populations. At the meeting Kelly Ivors presented a paper in which the populations were defined using AFLP and Microsatellite SSR methods. The European population (EU) appears to have a single lineage and is of the A1 mating type (with only one A2 isolate found in that lineage). Following Ivors *et al*. (2006), this population should now be referred to as *P. ramorum* EU lineage 1. In America three lineages have been detected. The EU lineage 1 (A1 mating type) has been found in a restricted number of nurseries in Washington state and is thought to be introduced from Europe (Hansen *et al*., 2003; Ivors *et al*., 2006). In the USA, the so-called 'original' American population, NA lineage 1, and a more recently discovered group, NA lineage 2 *fide* Ivors *et al*., 2006, occur. Forest populations of Pr in the US are comprised uniquely of NA lineage 1, while in nurseries both NA lineage 1 and NA lineage 2 occur (Ivors *et al*., 2006). Both NA lineage 1 and 2 are exclusively of the A2 mating type. At APS data was presented to show that NA lineage 2 is equally pathogenic on coast live oak seedlings, but has different colony morphology, growth rate and temperature preferences regarding growth on V8A than NA lineage 1.

P. kernoviae on the other hand is homothallic and to date AFLP data show little variation suggesting a single introduction into the UK (K. Hughes, CSL, pers. comm.). With both these pathogens the epidemics they cause are driven by the asexual cycle in which deciduous sporangia and zoospores are formed. Sporulation takes place on infected foliage and shoots (but not on tree trunks) of ornamental and understorey species, therefore foliage infections are crucial in driving disease outbreaks. Epidemics caused by *Phytophthora kernoviae* and *P. ramorum* exemplify the dangers that introduced aggressive, broad-spectrum pathogens have on native ecosystems as well as on valuable exotic species, and the role that infected nursery plants play in dissemination. Thus the news that both *Phytophthora* spp. can reside asymptomatically for at least 8 days in infected tissue, and can sporulate on symptom free leaves and fruits, is

have been predicted (or were we just desperate for answers by then?). Many gene knockouts of putative pathogenicity factors had no effect on phenotype. For example most wall degrading enzymes are represented by several genes such that deletion of one seems to be compensated by others (e.g. Bindschedler *et al*. 2003, Fung. Genet. Biol. 38, 43). Walton's group have deleted many genes such as xylanases and glucanases from *Cochliobolus carbonum* with no alteration to pathogenicity. Detoxification of antimicrobial compounds can occur by different routes. Multiple genes, previously silent genes and functional redundancy has severely limited the number of clear answers given by this approach. There are clear links established however for some factors as diverse as avenacinase of *Gaeumannomyces graminis* (to detoxify the oat phytoanticipin avenacin) and HC toxin synthase of *C. carbonum.*

Forget the past; random mutants reveal hrp genes. A more progressive approach was to create random mutants and screen for non-pathogenicity or reduced virulence. Genes responsible could then be located, cloned and complemented back to restore normal function. A classic example and truly major advance came from a search by random mutagenesis for bacterial genes linked with HR. Many HR-defective mutants were readily obtained; unexpectedly and at that time confusingly, most mutants also lost pathogenicity. How could avirulence and pathogenicity be linked? These so called HRP genes (*HR* and *P*athogenicity) mostly coded for type III secretion apparatus, known from animal pathogens like *Salmonella* and *Yersinia* to be responsible for secretion of virulence proteins. We now know that a long, filamentous HRP pilus transcends the plant cell wall and delivers HRP effectors directly to host cytoplasm or nucleus; the proteins are required for HR in incompatible interactions and pathogenicity in compatible responses and some are the once elusive avirulence gene products. Mutagenesis of fungi remains more problematic. REMI (restriction enzyme mediated integration) provided hope but has been surpassed by *Agrobacterium*-mediated transformation, used now routinely on a wide range of pathogens for insertional and targeted mutagenesis, as its T-DNA integrates on chromosomes usually as a single copy.

***mRNA and proteins* in planta**. Other open-minded routes to unravel pathogenicity factors include: seeking *in planta* pathogen transcripts, such as revealed *mpg1* coding for the hydrophobin of *Magnaporthe grisea* on rice leaf surface; or isolation of pathogen proteins *in planta* which led to identification of avirulence proteins from *Cladosporium fulvum* and *Fusarium oxysporum* in tomato leaf apoplast and xylem fluids respectively; development specific transcripts or proteins showed wall-degrading enzymes in infection structures and hexose and amino acid transporters of haustoria of *Uromyces viciae faba*, confirmation of their role alluded to above as feeding structures. Flax rust provided the original gene for gene concept of plant-pathogen interaction. Now the molecules involved are being revealed. AvrL567 rust genes were identified by map-based cloning and the small, secreted proteins are recognised by the L5, L6 anor L7 flax resistance proteins (Dodds *et al*. 2004, Plant Cell 16, 755). The *avr* genes are expressed in haustoria and another 20 haustorial expressed proteins were found from an isolated haustorium cDNA library; at least d/two are avr genes (avrM and avrP4) and their transient expression triggered HR in

plants containing the corresponding R genes M or P4. Notably, even relatively intractable fungi such as obligate biotrophs and Oomycetes are open to these approaches. The transcriptome of surface structures of *Blumeria graminis* on barley revealed some cDNAs homologous to fungal pathogenicity and virulence genes; other unidentified homologues in this cluster might be proved "guilty by association". This biotroph appears able to carry out most primary metabolic processes and it appears unlikely that auxotrophy will explain its obligate nature; reliance on host cues is more likely (Both *et al.* 2005. Mol. Plant-Microbe Interact. 18, 125). For *Phytophthora* spp. there are intensive inputs as sequencing projects from interaction transcriptomes. For example, novel necrosis-inducing proteins *crn1* and *crn2* have been revealed from *P. infestans*. Also serine protease inhibitors were found, of which EP11 interacts with tomato P69 subtilisin-type proteases suggesting defence-counter defence crosstalk. EP11 also protects another protease inhibitor from plant proteases in intercellular fluids. The knowledge of pathogen (and related saprotroph) genomes is often required for identification of what are sometimes gene fragments (expression sequence tags or ESTs), which are being generated in large numbers from infection or starvation libraries. Soames & Talbot (2006. Mol. Plant Pathol. 7, 61) describe a comparative genomic analysis of fungi from ESTs including differences between free-living yeasts, more complex saprotrophic filamentous fungi and pathogenic fungi. One revelation was that *B. graminis* with its biotrophic lifestyle requires many gene products not found in even hemibiotrophs and possesses many unisequences of unknown function (68%) of which some are expressed during infection.

Pathogen genomes. The first plant pathogenic bacterial genome sequence was *Xylella fastidiosa* from citrus in 2000. Now there are around 80 completed or ongoing projects, including many fungi, as sequencing centres can now handle their larger genomes of the range 30-50Mb for ascomycetes and <250 Mb for Oomycetes. The list includes: *Agrobacterium, Clavibacter* subspp., *Erwinia* spp., *Ralstonia, Pseudomonas syringae* pvs., *Fusarium spp., Magnaporthe, Phytophthora spp., Puccinia, Stagonospora, Streptomyces.* Other than functional genomics, which interaction libraries are revealing, comparative genomics provides much fundamental information. In bacteria genomic islands differing in GC content and codon usage pattern from the rest of the genome reveal horizontal gene transfer. The regions are often pathogenicity islands containing type III secretion and associated effector protein genes, or toxins, and show the potential for rapid evolution to new pathogenicity. For example *Xylella* is a highly specialised, xylem-invading citrus pathogen introduced by an insect vector. Coincidentally and unusually, it lacks type III secretion and the ability to deal with lipid metabolism, yet its carbohydrate utilisation is evident. In contrast *Ralstonia* has much greater flexibility coincident with its mode of pathogenicity and need for survival outside the host. In particular, many of its genes control attachment, polysaccharide production and type III system and effectors (Salanoubat *et al.* 2002, Nature 415, 497). Who would have predicted that the archetypal necrotrophic, pectinase producing, cell macerating *Pectobacterium* (syn. *Erwinia*) *atroseptica* uses type III effectors, fixes nitrogen and needs the toxin coronatine (Toth & Birch 2005. Curr. Opin. Plant Biol. 8, 424)? This type of information really makes one think again and discard dogmas.

Selected highlights on *Phytophthora ramorum*, *P. kernoviae* and other items of interest presented at the 2006 APS meeting

This year (2006), the American Phytopathological Society (APS) annual meeting was combined with the Canadian Phytopathological Society (CPS) and the Mycological Society of America (MSA) meetings, to make a tripartite congress held in Quebec city, Canada from 31 July to 3 August. There were 1700 attendees and the underlying theme was 'Biological interactions and biological crossroads'.

Thanks are extended to the BSPP and Forest Research for funding my trip. At the congress I presented a paper describing discovery of asymptomatic infection of plant tissue, and sporulation on infected, symptom free leaves and fruit by two newly introduced *Phytophthora* species, *P. kernoviae* and *P. ramorum*, that are causing disease epidemics in the UK and elsewhere.

Phytophthora ramorum (Pr) is well know for causing sudden oak death in native coastal forests in California and parts of Oregon in the USA, but also affects foliage of many understorey species such as the Californian bay laurel (*Umbellularia californica*). In Europe in natural and semi-natural situations such as woodlands and heritage gardens it primarily attacks rhododendrons, *Pieris* and *Viburnum* causing leaf necrosis and shoot tip dieback. Trees are also affected and deaths occur because Pr kills the inner bark (phloem), girdling trees; or secondary invaders e.g. *Armillaria* may finally cause weakened trees to die. In the UK the most susceptible tree species is European beech (*Fagus sylvatica*) but others are also attacked (E.g. *Acer pseudoplatanus* (sycamore); *Aesculus hippocastanum* (horse chestnut) and *Quercus rubra* (red oak) see http://rapra.csl.gov.uk. In many cases if the foliage of a plant species is susceptible to Pr then the inner bark on stems and branches will be resistant, and vice versa. In both Europe and the USA *P. ramorum* is also a very important nursery pathogen affecting a wide range of plants causing leaf necrosis and shoot tip dieback primarily.

Phytophthora kernoviae (Pk), another recently described species (Brasier *et al*., 2005), is essentially a woodland pathogen with the key components of the disease system being foliage infections of *Rhododendron ponticum* and lethal bleeding cankers on beech trees. In spring and early summer (March – May) primarily the terminal buds on the rhododendrons are attacked and Pk migrates downwards through the shoot and into the petioles of the newly flushed leaves. Buds, shoots and petioles become blackened and necrotic and the entire shoot tip and its foliage wilt. Later in the season secondary infections affect mature rhododendron foliage, causing margin, leaf and drip tip necrosis. Trees in close proximity to infected rhododendrons become infected presumably when inoculum is available and conditions suitable for infection.

Another interesting talk and of considerable value to me was delivered by Paivi Parikka of Finland on "*Fusarium* infection and mycotoxin contents of oats under different tillage treatments." The presentation covered a less researched crop in terms of *Fusarium* infection and mycotoxin production - oats, and a recently identified *Fusarium* species (*F. langsethiae*), the fungal species I am researching on. This fungus is unique compared to other fusarium head blight (FHB) and mycotoxin producing species in small cereal crops. This created some curiosity among the audience, some of whom had never heard of it before. This led to a short but a very informative discussion on the subject. *F. langsethiae* has been found to produce more HT-2 and T-2 mycotoxins in oat than in any other cereal crop in Europe.

Another very important characteristic of this fungus is that it causes no visible disease symptoms on the infected crop making field assessment as would normally be done for other FHB causing fungi difficult. This is not good news for farmers and cereal food/feed processors where accept/ reject decisions have been based on an easy method of visual assessment of harvested grains. From her research, the speaker found that *F. langsethiae* (a HT-2 and T-2 producer) could be isolated in the early stages of panicle emergence and gradually decreased towards harvest when deoxynivalenol-producing *Fusarium* species dominated - which kind of explains why it is difficult to isolate it from dry grains. In connection with this presentation, there was a lot of interest in my poster presentation which talked about *F. langsethiae* infection and mycotoxin production in oats, particularly from Scandinavian researchers who have identified high levels of *Fusarium* mycotoxins in oats.

Besides scientific presentations, participants had ample time for discussions and some socialising which made the conference more fun. Delegates were treated to a very colourful dinner on a luxury boat on the river Rhine. Various delicacies were served and I could see people spoilt for choice. Live music on the boat kept scientists entertained as they enjoyed one drink after another.

I am delighted for having had an opportunity to attend this conference which gave me an opportunity to meet and chat with researchers whom I had only known as names in journal articles, and others I had never met before. The seminar was a huge success, thanks to the organisers.

For more information and to view abstracts presented in this seminar, please visit: http://www.efs9.com

I would like to thank the British Society for Plant Pathology for kindly contributing towards my conference expenses.

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Likewise, why does *M. grisea* carry nine putative cutinases when the fungus is well known for generating enormous turgor pressure to penetrate its hosts mechanically? Perhaps one should not be surprised when fungi such as *Fusarium* are predicted to produce 350-450 secreted degradative enzymes, from pectinases to nucleases. Nitric oxide is now known as an animal and plant signalling molecule but for what purpose does *M. grisea* contain four NO synthases? Mimicry of host molecules to subvert key host defences is evident from coronatine production by *Pectobacterium* and by *P. syringae*, as coronatine structurally and functionally mimics methyl jasmonate, a plant defence signalling molecule. Also Boucher's group showed that *Ralstonia solanacearum* produces GALA effectors. These seem to mimic plant F-box proteins and target plant cellular components for proteolysis through the ubiquitin ligase-mediated pathway.

Interspecific gene transfer has recently been shown in fungi. The genome sequence of *Stagonospora nodorum* has a predicted gene with 99.7% similarity to ToxA of *Pyrenophora*. Mixed infection of wheat leaves is common and a population of *P. tritici-repentis* with significantly enhanced virulence appears to have arisen around 1941 and spread "tanspot" worldwide (Friesen *et al.* 2006 Nature Genetics 38, 953). Evidence for longer term evolution to pathogenicity comes from genome comparisons of *Phytophthora sojae* and *P. ramorum* with stramenophile photosynthetic algal ancestors. The expansion and diversification of protein families is linked with infection of plants, notably hydrolases such as cutinases, ABC transporters, toxins, proteinase inhibitors, and especially a superfamily of putative avirulence genes; data mining of the genomes and from functional genomics has identified >200 genes likely to code for secreted effectors. Secreted proteins have evolved significantly more rapidly than the overall proteome (Tyler *et al*. 2006. Science 313, 1261). Many bacterial virulence traits are controlled by quorum sensing in which bacterial cell populations act *via* diffusible signals molecules, such as homoserine lactones (OHHL) in *E. carotovora* and *P. syringae*. Other than the well established control of wall degrading enzymes and EPS, another gene activated or repressed by OHHL is *nip*. The Nip protein induces necrosis and is homologous to various necrosis-inducing toxins from *Phytophthora*, some true fungi and a streptomycete (Pemberton *et al*. 2005. Mol. Plant-Microb. Interact. 18, 343). Unculturable phytoplasmas have remained poorly understood despite their importance, until the recent sequencing of three of their genomes. AY-WB (Asters Yellows strain Witches' Broom) encodes >58 secreted proteins of which several are predicted to target cell nuclei as confirmed by fluorescent protein fusions; also gene transcripts were detected in infected plants (Bai *et al*. 2006. J. Bacteriol. 188, 3682).

Model systems, essential, but mind the gaps. *Arabidopsis* and its range of pathogens have enabled major advances in understanding interactions. All will be aware of the reasons, such as rapid life cycle, sequenced genome, tagged mutations and small size. We should though bear in mind its limitations. It does not harbour *Rhizobium* or mycorrhizas. Some pathogens appear to me forced rather than representing true diseases; these include most necrotrophic fungi and vascular fungi. For example *Fusarium oxysporum* invasion of leaves hardly mimics xylem invasion. *Medicago truncatula* as a legume model covers some of these deficiencies and is being groomed accordingly.

Avirulence genes and other suppressors of defences. Type III effectors are double-edged swords with the capacity to induce and suppress host defences. Their predicted function is to inhibit PAMP (see below)-induced defences. Most *avr* genes are likely to be fundamental to fitness or virulence or they would be shed to avoid recognition by R genes. *P. syringae* delivers 20-50 effector proteins into plant cells and *R. solanacearum* has >40 predicted effectors. Some but not all enhance virulence. Seeking their functions is a priority area and ongoing research is revealing their targets. Many comprise novel sequences that do not allow prediction of their function. Many host targets for effector proteins from mammalian pathogenic bacteria are described and involve subversion of host defences, but much less is known for plant pathogens. Programmed cell death, cell wall-based defences, hormone signalling, expression of defence genes and other basal defences are some putative targets of plant pathogen effectors (see Abramovitch & Martin 2004. Curr. Opin. Plant Biol. 7, 356). Much less is known about fungal and Oomycete *avr* genes although the search is on. Even Avr genes from powdery (*B. graminis*) and downy (*Hyaloperonospora parasitica*) mildews are being uncovered. Bioinformatics, through synteny allows prediction of such effectors. Oomycete avr genes share two motifs including RXLR near the N terminus. Both turn up as a superfamily in genomes of *P. sojae* and *P. ramorum* "*avh*" (*avr* homologues) genes. RXLR is conserved in the malarial parasite *Plasmodium* to transport proteins to the cytoplasm of human erythrocytes. RXLR has over 60 representatives and they are likely to be cytoplasmic effectors; these include AVR3a from *P. infestans* that triggers HR in R3a plants and suppresses HR induced by INF1 elicitin. Delivery is likely to be *via* infection vesicles and haustoria. Apoplastic effectors are often small and cysteine-rich, such as serine and cysteine protease inhibitors (Birch *et al.* 2006. Trends Microbiol.14, 8). Suppression of recognition is another likely role; for example *avr4* from *C. fulvum* encodes a chitin-binding protein that may mask fungal chitin from recognition and from host chitinases. Evolution being what it is, this protein is recognised by a host R gene *Cf-4*, but the pathogen has countered this by mutating a disulphide bridge, which maintain function but avoids detection. Other fungi such as *Colletotrichum* and *Uromyces* also seem to have shielded their chitin, possibly by developmentally regulated chitin deacetylases. *Colletotrichum* also surrounds its infection vesicle with a matrix proline-rich protein resembling that found in plant cell walls, possibly avoiding detection by this means; the gene is switched off at the onset of necrotrophy. Current work from my lab is showing how EPSs from a wide range of bacterial pathogens block signalling and defence gene expression. Previously the abundant high molecular weight polymers were assumed to be merely protective from dehydration and UV. In fact their polyanionic nature confers another key binding property of the signalling cation calcium. Viral pathogens limit host defences by suppressing RNA silencing used by plants to target and degrade viral RNA (Baulcombe 2002, Trends Microbiol 10, 306.). Virus induced gene silencing (VIGS) is now used to study function of defence genes; the viral vector is engineered to contain part of the plant gene in the antisense orientation. The produced complementary RNA will bind to the corresponding RNA in the plant cell forming d/s RNA. This causes production of small interfering RNA molecules that target their specific sequence for

CONFERENCE REPORTS

The 9th European Fusarium Seminar (EFS9), Wageningen, The Netherlands, Sept 2006

Wageningen is a small quiet city many people refer to as a Friendly City, a University City and/or a Liberation City. Residents love cycling, something no one fails to notice the moment they arrive. The weather during the conference was simply excellent, bright sunny and warm, creating a perfect atmosphere for the event.

The conference was attended by 185 scientists from all over the world, and was officially opened by a presentation from Martin Kroff, Dean of Wageningen University and Research Centre. His talk touched on the importance of providing safe food and ensuring food security in both developed and developing countries. His speech emphasised the need for more research to tackle the problems associated with *Fusarium* fungi which impacts negatively on the availability and safety of foods and feeds. He noted that problems associated with *Fusarium* such as reduced crop yield, quality and the potential contamination of food/feed with mycotoxins did not only affect Europe but every part of the world. This point was made clear by the high number of conference participants from outside Europe. His parting comment was that the scientists working on *Fusarium* research should keep the fire burning to alleviate problems posed by the genus.

By bringing together scientists with a common goal, the seminar provided a first hand opportunity for all to listen and discuss on a variety of subjects related to *Fusarium*. The subjects ranged from various crop infections and disease incidences on crops, mycotoxins and their toxicity to humans and animals, disease control and forecasting as well as the past and the present molecular techniques for fungal detection and fungal biomass quantification.

The mycotoxins in food and feed chain talk presented by Johanna Fink-Gremmels was very interesting. It focussed on the effect some *Fusarium* mycotoxins have on humans and animals. It gave an insight on their toxicity, and the advancement in the understanding of the molecular mechanisms underlying effects caused by them. She pointed out that prediction of safe levels and long-term exposure are important as it is not possible to produce *Fusarium*-mycotoxin-free foods under current agricultural practices, and that new mycotoxins continue to be identified. In Ulf Thrane's talk, he demonstrated how important *Fusarium* and its mycotoxins are by giving an example of a two word search on a scientific database ("*Fusarium* AND Mycotoxins") which generates thousands of hits showing how much research has been and is being done on the subject, meaning the battle of man and *Fusarium* is not yet over.

destruction. VIGS characterisation of genes associated with powdery mildew resistance of barley is described by Hein *et al*. (2005. Plant Physiol. 138, 2155).

Resistance genes are eventually identified The five main groups comprise: [1] Pto from tomato-a serine threonine kinase which interacts directly in the plasma membrane with AvrPto from *P. syringae* pv. *tomato.* [2] Proteins with leucine rich repeats (LRR), a nucleotide binding site (NBS) and a putative leucine zipper (CC). LRR motifs are implicated in protein-protein interactions. From rice blast resistance protein *Pi-ta* interacts with AvrPi-ta from *Magnaporthe.* [3] Proteins which lack CC domain but possess Toll (receptor protein homologue from *Drosophila*) and interleukin-1 receptors. [4] Cf proteins targetting *Cladosporium fulvum* in tomato possess a transmembrane domain and an extracellular LRR region. This implies receptor activity outside the cell coincident with the apoplastic location of the pathogen. The first three classes all appear to be cytoplasmically localised, relevant to intracellular effectors of bacteria. [5] Resistance to the rice pathogen *Xanthomonas oryzae* is given by Xa21 which contains extracellular LRR and intracellular serine-threonine kinase domain. R genes are ubiquitous in plants; Arabidopsis has *ca.* 150 putative R genes, based on NBB-LRR homology. Comparison of mutant and naturally occurring alleles of R genes with loss of function or different specificities is revealing regions controlling specificity. With the two exceptions above there is a lack of evidence for direct interactions between R proteins and corresponding AVR proteins. That would be too simple. Space prevents detailed discussion here of complex issues involving complexes but see Martin *et al*. (2003) Annu. Rev. Plant Biol. 54, 23. The *guard hypothesis* proposes that effector proteins target host proteins, which regulate host defences, but plants possess guard proteins that prevent or recognise such interactions and trigger HR. In other words, R proteins may undertake surveillance of key physiological processes targeted by pathogens. For example in *Arabidopsis* the protein RIN4 is a negative regulator of R genes RPM1 and RPS2 and seems to play the role of a broad spectrum, molecular switch regulating at least two, probably three R protein-mediated defence pathways. These are activated when *P. syringae* AvrRpt2 targets and cleaves RIN4 (Day *et al*. 2006. Plant Cell 18, 2782).

Pathogen perception and defence-related genes. Following R-AVR interactions leading to HR, or perception of elicitors (now more often termed PAMPs or *P*athogen *A*ssociated *M*olecular *P*atterns) in hosts or in non-hosts, a high proportion of host genes are up- or down-regulated. PAMPs continue to be uncovered, notably bacterial flagellin and elongation factor (EF-Tu); both are major conserved proteins that are recognised and cannot be readily altered (Kunze *et al*. 2004 Plant Cell 16, 3496). However, some pathogens (*X. campestris, R. solanacearum, E. carotovora, A. tumefaciens*) have evolved independently inactive flagellin and evade that detection system. A surprising PAMP addition is fungal xylanase. Defence elicitors can also derive from the host; oligogalacturonides are released on cell damage or by pathogen pectinases and are potent inducers of defence. Host inhibitory wall proteins PGIPs might limit polygalacturonase activity such that eliciting oligosaccharides generated from wall cleavage are not further degraded to inactive forms. Host surveillance molecules fit into two categories: Toll like receptors (TLRs) are transmembrane proteins with extracellular LRRs and NB-LRR proteins; Arabidopsis has >400 TLRs and 100 NB-LRRs.

However, only the receptors for flagellin and xylanase have been characterised. Many host genes are clearly linked to defence and include enzymes involved in biosynthesis of phenolics and phytoalexins, enzymes that modify and strengthen cell walls, PR proteins with enzyme activity against microbial cell walls or peptides that are directly antimicrobial. For example, PR1 in tobacco is induced up to 1000-fold and reaches 2% of leaf protein. Techniques allow these to be tracked with time and are revealing other numerous and diverse genes associated with defence. Expression profiling becomes even more sophisticated when combined with *Arabidopsis* genetics and genomics methods. Mutations that affect salicylic acid and jasmonic acid (JA) signalling in defence linked pathways) and synthesis of the phytoalexin camalexin have been used. Even non-hosts and non-model species can be analysed for potential defences; using cDNA-AFLP we described many new defence-related genes new to cassava (Kemp *et al*. 2005. Mol. Plant Pathol. 6, 113).

Non-host resistance. Non-host resistance accounts for the majority of disease resistance in natural situations. We knew from the 1980s that rust fungi could fail on the "wrong" host at any stage from germination to haustorium establishment-so called "switching points" of Heath. Clearly, constitutive and inducible responses are involved. Activation of the latter is probably brought about by PAMPs, somewhat analogous to activation of innate immunity in animals. PAMPS, also avirulent pathogens, mycorrhizal fungi and other molecules like SA, establish systemic protection against subsequent infection with virulent pathogens. This so called "priming" is reviewed by Mauch Mani *et al*. (2006. Mol. Plant Microbe Interact. 19, 1062). Non-host resistance is now being dissected by various means. Mackey's group have shown resistance of Arabidopsis to the non-pathogen *P. syringae* pv. *phaseolicola* is based on at least three pathways involving "basal defences", but not HR. When all are inhibited, growth reaches levels similar to that of compatible *P. syringae* pv. *tomato*. Forward genetic screens for *Arabidopsis* mutants with impaired penetration by barley powdery mildew *B. graminis* have revealed some novel loci. One (*pen1*) encodes syntaxin, which belongs to the superfamily of SNARE, proteins that mediate membrane fusion during vesicle trafficking. *Pen1* mutants have delayed deposition of cell wall appositions on attempted penetration. The actin cytoskeleton and cell wall-plasma membrane interconnection seem to be important preformed but responsive elements as revealed in *Arabidopsis* defence to wheat powdery mildew and to rusts . Analysis of mutants impaired in the hormones JA, SA and ethylene show that they not only play key roles in cultivar-specific resistance but in non-host resistance. Inducible defence responses in non-hosts include synthesis and accumulation of phytoalexins. Searching for antimicrobial compounds is no longer a mainline activity, but by chance we discovered the first (and still only) inorganic phytoalexin. Elemental sulphur (S^0) is produced as a component of induced defence against xylem-invading vascular pathogens in diverse families including tomato, tobacco, cotton, *Phaseolus* bean and the species in which it was first discovered, *Theobroma cacao*. S^0 is of course highly fungitoxic and was localised by SEM-EDX to xylem cells. In *Arabidopsis* leaves S^0 is constitutive (Cooper & Williams, 2004. J. Exp.Bot. 55, 1947). According to Nurnberger & Lipka (2005 Mol. Plant Pathol 6, 335), non race-specific and race-specific defences should be

considered as distinct but evolutionarily interrelated and together constitute plant innate immunity. Analogies with animal innate immunity include the FLS2 receptor of flagellin in *Arabidopsis* that is related to animal TLR receptors; also nitric oxide and MAPK cascades are key elements in both defence systems.

Applications of resistance? Many defence-related genes have been tried as transgenes to enhance resistance. However, engineering resistance to diseases has proved much more recalcitrant than to insects and viruses. Genes such as those coding for hydrolases are best used in combination but there are few commercial examples to date. Most hopes rest with R genes, but they cannot be readily moved between taxa. They may however be accessible to domain and specificity alterations. Placing R and cognate avr genes under an infection-inducible promoter in the same plant is an exciting strategy that could provide resistance to all pathogens that trigger that promoter. It has been shown to succeed in tomato expressing both Cf9 and avr9. Conserved domains in R genes allows searches in crop species to clone gene fragments known as resistance gene analogues or RGAs, and using them as RFLP markers or in genetic mapping. Tight genetic linkage between RGAs and R genes has been found in monocots and dicots. The approach may be especially useful for species relatively intractable for resistance screening and breeding, such as oil palm and cassava. Even where R genes are well known they can be especially vulnerable to pathogen adaptability, never more so than with potato late blight. Map based cloning with LT-PCR has provided a major R gene RB from *Solanum bulbocastanum*, a diploid, wild relative, highly resistant to all known *P. infestans* races; RB gives broad spectrum resistance to *P. infestans* and should therefore be durable. Certain potato varieties are favoured by the processing industry, but backcrossing *S. bulbocastanum* derived germplasm may not be efficient and acceptable thanks to the tetraploid and heterogeneous potato genome. Favourites such as blight susceptible Russet Burbank may be rendered resistant by engineering the RB gene (Song *et al*. 2003. PNAS, 100, 9128). Lack of durability of R genes has been their Achilles' heel, but some have proved long lasting. Durability can in some cases be predicted by the fitness penalty that loss of the corresponding *avr* gene imposes. All of these possibilities await acceptability of transgenic technology in UK and some other crop production systems.

In Conclusion.

The field of plant-pathogen interactions is at a truly exciting stage. This is not only because of the technologies that can now be used, but because there is real awareness that one has to operate on both sides of the fence. This is epitomised by the questions raised when considering pathogen Avr products (what are they?) targeting plant defence pathways (what is targeted?) and suppressing PAMPS perception (which ones are produced *in planta*?), but plants with cognate R genes guard their targets (how?) and consequently can perceive PAMPS (what are the receptors? what then results?). In the past too many researchers were focussed on the host or the pathogen. However, mechanisms of pathogenicity and defence are inextricably intertwined as they have undoubtedly co-evolved. This is why I have chosen the theme of "Attack and Defence" for my Presidential Meeting to be held in Bath in September 2007. At the current rate of progress there will be many more conceptual breakthroughs to discuss then.