Molecular Biology of Plant Pathogens XX

21 September 2009
Somerville College, University of Oxford

Conference organizers

Pari Skamnioti (University of Oxford)
Steve Whisson (SCRI)
Mary Coates (University of Warwick)

Programme and Abstracts

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We are grateful to the following for their financial support:

Information for delegates

Delegates can arrive at Somerville College, Oxford any time after 2 pm of the 20th of September.
Maps and directions can be found here
http://www.some.ox.ac.uk/268/Maps-and-directions.html
Room keys can be collected at the Somerville College Porter's Lodge.
Dinner is not provided but there are excellent pubs/restaurants around (e.g. on Little Clarendon or Walton streets in nearby Jericho, or on St Giles or George streets right in the city centre).
On the 21st of September breakfast is at 7:30 am. Registration starts at 8:30, at the conference venue, the Flora Anderson Hall at Somerville.

Information for speakers

All Powerpoint talks should be brought to the conference on a CD or a USB stick and loaded onto the conference computer by at latest the end of the previous session.
Speakers of the first session should upload their presentations the evening before (please look for Pari Skamnioti) or between 08:00-08:30 on the 21st of September.
It is advisable to compress big static image files. All speakers should make an effort to not exceed their allocated time-slot.

Programme Outline

09:00-9:05	***Welcome***	
Session I	Chair: Pari Skamnioti	
09:05-09:20	Susan Breen	Cloning and recognition of *Phytophthora infestans* RXLR effector Avr2
09:20-09:40	Sue Donovan	Characterising the role of downy mildew pathogenicity effector proteins in Arabidopsis
09:40-09:55	Tom Mentlak	Characterisation of LysM effectors in the rice blast fungus *Magnaporthe oryzae*
09:55-10:15	Liliana Cano	Exploring transcriptome sequencing to study effector evolution in *P. ipomoeae* and *P. mirabilis*
10:15-10:30	Sarat Bimanadham	Deciphering common eukaryotic pathways targeted by *Hyaloperonospora arabidopsidis* effectors
10:30-10:50	Lauren Ryder	A novel mutant of the rhizosphere fungus *Trichoderma hamatum* causes growth promotion in plants
10:50-11:20	***Coffee/Tea***	
Session II	Chair: Petra Boevink	
11:20-11:40	Aziz Mithani	Comparative analysis of metabolic networks provides insight into the evolution of pathogenic and non-pathogenic lifestyles in *Pseudomonas*
11:40-11:55	Muhammad Badaruddin	Investigating the role of glycogen metabolism in *Magnaporthe oryzae*

11:55-12:15	Jens Due Jensen	Targeted gene replacement and overexpression of *Fusarium graminearum* (PH1) (Fg)SKN7 and FgAP-1-like, two transcription factors involved in oxidative stress sensing and regulation.
12:15-12:35	Arantza Rico	Metabolic profiling of the tomato apoplast during infection by *Pseudomonas syringae*
12:35-12:50	Joanna Fyans	The role of protein transport in the physiology and pathogenicity of *Streptomyces* species
12:50- 13:50	***Lunch***	
Session III	Chair: Chris Thornton	
14:00-14:20	Sonia Humphris	*Dickeya* spp. - A new threat to potato production
14:20-14:40	Philip Swarbrick	Molecular tools for the detection of phytoplasmas causing African 'lethal yellowing' diseases of coconut palms
14:40-15:00	Nichola Hawkins	Recent evolution of *Rhynchosporium secalis* populations in response to selection by fungicides
15:00-15:15	Hefni Rusli	Molecular diagnostic tools for detection of oil palm (*Elaeis guineensis*) vascular wilt disease caused by *Fusarium oxysporum* f.sp. *elaeidis*
15:15-15:30	Melanie Tuffen	Molecular and physiological changes induced in *Catharanthus roseus* during phytoplasma infection
15:30-15:50	Caoimhe Fleming-Archibald	Factors which influence the expression of "brown mushroom" symptoms in

		crops infected with Mushroom Virus X
15:50-16:30	***Tea/Coffee***	
Presentations for BSPP PH Gregory Prize		
	Chair: Gary Foster	
16:30-16:45	Helen Lovell	Bacterial evolution by genomic island transfer occurs via DNA transformation *in planta*
16:45-17:00	Helen Fones	Defence spending slashed in the hyperaccumulator economy?
17:00-17:15	Frederico Dorati	Bacterial plant pathogen interactions with predators: survival and spread
17:15-17:30	Christopher Burt	New insights into eyespot resistance
17:30-17:45	Andrew Beacham	A pathogenicity factor enriched micro-region in the *Fusarium graminearum* genome
17:45-18:00	Emily Boys	Resistance to *Pyrenopeziza brassicae* (light leaf spot) in a mapping population of oilseed rape
18:00-18:15	Francesca Stefanato	The ABC-transporter BcatrB protects *Botrytis cinerea* against camalexin and is a virulence factor on Arabidopsis
18:15-18:30	William Truman	Arabidopsis auxin mutants are compromised in systemic acquired resistance and exhibit aberrant accumulation of various indolic compounds

Abstracts for MBPP XX

21st September 2009

Somerville College, University Oxford

Session I
Chair: Pari Skamnioti (University of Oxford)

Cloning and recognition of *Phytophthora infestans* RXLR effector Avr2

Susan Breen[1,2], Eleanor M. Gilroy[1], Miles R. Armstrong[2], Juan G. Morales[1], Ingo Hein[1], Emma Douglas[1], Petra C. Boevink[1], Hazel McLellan[2], Eva Randall[1], Zhendong Tian[1], Anna O. Avrova[1], Leighton Pritchard[1], Stephen C. Whisson[1] & Paul R. J. Birch[1,2]
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An important research goal in the fight against potato late blight, caused by the Oomycete *Phytophthora infestans,* is to identify pathogen effector proteins likely to be secreted during infection and translocated into host cells to manipulate host metabolism and defence responses.
AVR3a, the first effector characterized from *P. infestans*, was found to contain an N-terminal RxLR and dEER motif required for transport across the host plasma membrane. Developing genomic resources have allowed large-scale computational screening for this conserved motif to reveal approximately 500 rapidly diverging *P. infestans* effectors.
We have identified the RxLR-EER effector Avr2 from the sequenced isolate t30-4, which is recognised by *R2-like* genes. Cloning *Avr2* from virulent isolates collected around the world has revealed 3 additional alleles. These alleles evade recognition by the *R2-like* genes. The recognition of the C- and N-terminals of Avr2 has also been investigated.
All Avr2 alleles, the C- and N-terminals will be used to identify interacting plant host proteins using the Yeast 2-Hyrid system. The library to be screened was generated from pathogen challenged resistant and susceptible potato cultivars. Once identified, we will be using virus induced gene silencing (VIGS) to examine the function of the identified Avr2 interactors.

Characterising the role of downy mildew pathogenicity effector proteins in Arabidopsis

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The interactions between the oomycete plant pathogen *Hyaloperonospora arabidopsidis* and the host plant *Arabidopsis thaliana* provide a useful model system for the study of disease resistance in plants. *H. arabidopsidis* is an obligate biotroph and causes downy mildew on Arabidopsis and related species cause infection on other members of the crucifer family. It is closely related to highly destructive plant pathogens within the genera of Phytophthora and Pythium. *H. arabidopsidis* proteins with an RXLR motif (amino acid single letter code) and a signal peptide are believed to enter host plant cells where they then target components of the plant immune system and regulators of metabolism. In this project a yeast two-hybrid approach has been used to identify potential host proteins, which are targeted by RXLR motif containing effectors. Good candidates from these screens will be further confirmed using fluorescence microscopy and other techniques to prove the protein-to-protein interactions. Arabidopsis knock-out lines and RXLR overexpression lines will subsequently be used to deduce their effect on plant susceptibility.

Investigating the importance of effectors with lysin motifs of the rice blast fungus *Magnaporthe oryzae*

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Magnaporthe oryzae, the filamentous ascomycete fungus, is the causal agent of rice blast disease. Over three billion people depend on rice as part of their staple diet, yet millions of tonnes of rice yield are lost each year due to *M. oryzae* outbreaks. During biotrophic invasive growth, *M. oryzae* uses a battery of effector proteins in order to evade host defense responses and promote biotrophic growth, although the mechanisms by which this occurs in plant pathogenic fungi as a whole is still largely unknown. Two putative effector proteins from *M. oryzae* containing lysin motifs, MoLYS1 and MoLYS2, were investigated. Both proteins show significant homology to the known virulence factor Ecp6, which is secreted in to the apoplastic space of tomatoes by the leaf mold fungus, *Cladosporium fulvum* (Bolton *et al*., 2008). Targeted gene replacement of *MoLYS1* and *MoLYS2* genes was carried out in order to characterize the potential importance of these genes as virulence factors. In addition, the localization of both MoLYS1 and MoLYS2 was also investigated using translational fusions to green fluorescent protein (GFP) in order to examine their localization *in planta* and to investigate the cellular localization and secretory mechanism of putative apoplastic effectors.

Exploring transcriptome sequencing to study effector evolution in *P. ipomoeae* and *P. mirabilis*

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The highlands of Central Mexico are the center of origin of the potato late blight pathogen *Phytophthora infestans*. They are also home to several species closely related to *P. infestans*, namely *P. mirabilis* on *Mirabilis jalapa* and *P. ipomoeae* on *Ipomoea longipedunculata*. These species are thought to have evolved by host-jump followed by adaptation and specialization on distinct host plants. *P. infestans* secretes a large repertoire of effector proteins that evolve rapidly through birth-and-death evolution and typically exhibit adaptive selection. Our aim is to to identify candidate effectors that show species-level polymorphisms that can be related to host adaptation. We applied Illumina technology to sequence the transcriptomes of *P. mirabilis* and *P. ipomoeae* represented in normalized cDNA libraries constructed from mixed developmental stages including mycelia and germinated cysts. De novo sequence assembly revealed a novel RXLR effector present in both *P. ipomoeae* and *P. mirabilis* species but annotated as a pseudogene in the *P. infestans* T30-4 reference genome due to a 5-bp deletion. We found that transient expression of functional alleles resulted in suppression of plant immunity and we are currently expanding these experiments to the various hosts. Our study highlights the value of comparing transcriptomes from closely related species to identify candidate effectors in species with no prior gene sequence information. We now aim to connect the discovered genes to effector activities to reveal a putative role in host adaptation.

Deciphering common eukaryotic pathways targeted by *Hyaloperonospora arabidopsidis* effectors

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The family oomycetes, harbours some of the most devastating plant pathogens that cause severe damage to plants important in agriculture, forestry and natural ecosystems. Arabidopsis-*Hyaloperonospora arabidopsidis* interaction is an excellent model system for the elucidation of molecular and cellular bases of compatibility/incompatibility in the course of plant-parasite co-evolution. Recent cloning of effectors in several oomycete species including *H. arabidopsidis* revealed the presence of an RXLR-EER motif that has subsequently been shown to be necessary for translocation of the protein into the host cell. My project aims to investigate the roles of these RXLR-EER motif containing *H. arabidopsidis* candidate effectors inside the host cell and characterise the mechanisms that enable the pathogen to evade host cell immune system. I am utilising the yeast (*Saccharomyces cerevisiae*) system to screen a repertoire of these effectors containing the RXLR-EER motif. A high-throughput screen of these effectors has revealed some putative candidates causing yeast growth inhibition. I am performing an additional screen with these effectors to identify candidates that might perturb mouse BAX induced

cell death in yeast. The candidate effectors identified from the screens are being further studied by localisation and co-localisation experiments in yeast to identify their associated targets. The candidate effectors identified in yeast will then be studied in plants to determine whether any common eukaryotic factors targeted by these effectors have a biological significance in pathogen virulence and plant immunity.

A novel mutant of the rhizosphere fungus *Trichoderma hamatum* causes growth promotion in plants

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The human population is projected to reach ten billion people by the year 2050, and global grain demand is projected to double. This will place unsustainable demands on agriculture to meet global food requirements. Improvements in crop yields that have traditionally been driven by nitrogenous fertilizer applications are unsustainable due to the spiraling costs of fossil fuels used in the energy-intensive Haber-Bosch process. Soils contain substantial amounts of nitrogen sequestered in recalcitrant organic polymers such as chitin, and exploitation of this natural resource in nutrient-poor soils by beneficial fungi represents a sustainable means of nutrient supply. We examined the role of hexosaminidase, a key chitinolytic enzyme implicated in nitrogen release and supply in ectomycorrhizal-plant interactions, in the promotion of plant growth by the free-living soil fungus *Trichoderma hamatum*. We report the phenotypic analysis of a Δ*Thhex::HYG* mutant and demonstrate that, contrary to expectations, disruption of enzyme activity through targeted gene deletion of the *HEX1* gene resulted in an amplification of plant-growth-promotion by the fungus. Furthermore, we show that this amplification is generated by the hyper-secretion of diffusible metabolites that stimulate plant growth, as well as improving the biocontrol capabilities of *T. hamatum* against the destructive rice infecting pathogen *Magnaporthe oryzae*. Thus, the work shows that significant improvements can be made to crop yields in low fertility soils without exogenous plant nutrients, by simple genetic modification of a ubiquitous soil fungus.

Session II
Chair: Petra Boevink (Scottish Crop Research Institute)

Comparative analysis of metabolic networks provides insight into the evolution of pathogenic and non-pathogenic lifestyles in *Pseudomonas*

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Bacteria in the genus *Pseudomonas* are ubiquitous inhabitants of the plant environment. However, individual strains and species adopt diverse lifestyles and colonise different plant-associated environments. The association of different species of *Pseudomonas* with different environments and different host interactions indicates that each species possesses distinct environment and lifestyle specific adaptations. In this study we have used the metabolic pathway prediction tool Rahnuma and complementary bioinformatic and phylogenetic analyses to assess the metabolic similarity across nine genome-sequenced *Pseudomonas* strains belonging to five distinct species by comparing the distribution and function of metabolic reactions annotated in the KEGG database. The results show that strains from the two pathogenic species, the opportunistic human pathogen *Pseudomonas aeruginosa* and the plant pathogen *Pseudomonas syringae* display a relatively high level of intra-species similarity compared to strains from the non-pathogenic species *Pseudomonas fluorescens,* which is consistent with experimental analyses of nutrient utilisation by these bacteria. We use parsimony analysis and reaction neighbourhood analysis to model the evolution of metabolic pathways in *Pseudomonas* and to infer the pathways and reactions present in the common ancestor of *Pseudomonas*.

Investigating the role of glycogen metabolism during infection-related development in *Magnaporthe oryzae*

Muhammad Badaruddin, Lucy J. Holcombe, Zheng-Yi Wang, Darren M. Soanes and Nicholas J. Talbot.
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The rice blast fungus *Magnaporthe grisea* infects its host by elaborating a specialised infection structure known as an appressorium. This single-celled structure forms in response to the hard, hydrophobic rice leaf surface and brings about infection by generation of hydrostatic pressure. *M. grisea* appressoria are melanin-pigmented cells with a thickened cell wall that allows turgor to develop within the cell due to accumulation of glycerol and the subsequent influx of water. We are investigating the mechanism by which appressoria generate high turgor, which is necessary for breaching the host plant cuticle. Turgor generation involves accumulation of glycerol in the appressorium and we are studying the origin of glycerol, its biosynthetic pathway during appressorium formation and the genetic regulation of turgor generation and appressorium maturation. In particular the role of glycogen metabolism is being investigated. We have generated targeted gene deletion mutants in genes encoding amyloglucosidase, the glycogen de-branching enzyme (*AGL1*) and glycogen phosphorylase (*GPH1*), which are collectively responsible for degradation and mobilisation of glycogen reserves. Both *Δagl1* and *Δgph1* mutants show reductions in blast symptoms, while a double *Δagl1 Δgph1* mutant shows a severe reduction in virulence. Consistent with this, we also observed delayed mobilisation of glycogen reserves from conidia and appressoria during infection-

related development. The metabolism of glycogen in *M. oryzae* and its genetic regulation will be discussed.

Targeted gene replacement and overexpression of *Fusarium graminearum* (PH1) (*Fg*)SKN7 and *Fg*AP-1-like, two transcription factors involved in oxidative stress sensing and regulation.

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Fusarium graminearum (*Fg*) is an ascomycetous fungal pathogen that mainly infects cereals, causing the disease Fusarium head blight (FHB) on barley and wheat as well as stalk and ear rot disease on maize. This disease not only causes significant crop yield losses, but also causes severe quality losses by contamination of the grain with mycotoxins, making the products unhealthy for food or feed.
Environmental stress factors, including oxidative stress, are reported to increase mycotoxin production of fungi, but the molecular regulation is not well understood. During infection, *Fg* mycotoxin biosynthesis may by modulated by host defence responses (*i.e*. oxidative burst). *In vitro* studies of *Fg* cultures stressed by H_2O_2 showed increased accumulation of deoxynivalenol (DON) (Ponts *et al.* 2006 & 2007). However, it has also been shown that injection of DON into wheat leaves resulted in H_2O_2 accumulation (Desmond *et al.* 2008), implying that *Fg* also needs to be able to deal with oxidative stress during infection.
In yeast (*Saccharomyces cerevisae*), three conserved signalling pathways have been identified in response to oxidative stress: the stress-responsive MAP kinase cascade, the multistep phosphorelay and the AP-1-like transcription factor (Ikner and Shiozaki 2005). Deletion of the AP-1-like transcription factor gene (CHAP1), a redox-regulated bZIP protein in *Cochliobolus heterostrophus,* resulted in increased sensitivity to oxidative stress caused by H_2O_2 and menadione (Lev *et al.* 2005). Likewise, deletion of SKN7, a response regulator in the multistep phosphorelay signaling pathway of *Aspergillus parasiticus* resulted in increased sensitivity to oxidative stress as well as early reactive oxygen species (ROS) formation and aflatoxin biosynthesis (Reverberi *et al.* 2008). The presence of putative binding sites in the promotor region of (aflR), a regulatory gene of aflatoxin biosynthesis, suggests that SKN7 has a role in regulation of secondary metabolism.
In *Fg,* it has been shown that H_2O_2 triggers toxin production and that toxin accumulation may be a part of a global response against and/or part of an adaptation to ROS. However, the signalling pathways which respond to H_2O_2 and lead to the toxin biosynthesis have yet to be elucidated. To investigate this, targeted gene replacement and overexpression of *Fusarium graminearum Fg*SKN7 and *Fg*AP-1-like have been constructed using USER-friendly cloning (Frandsen *et al.* 2008) and genes under transcriptional regulation of the two targeted genes will be identified under oxidative stress as well as *in planta* during infection.

Desmond *et al.* 2008. Molecular Plant Pathology 9(4):435-445; Frandsen *et al.* 2008. BMC Mol Biol 9:70; Ikner and Shiozaki 2005. Mutation Research 569:13-27; Lev *et al.* 2005. Eukaryotic Cell 4(2):443-454; Ponts *et al.* 2006. FEMS Microbiol Lett 258:102-107; Ponts *et al*. 2007. FEBS Lett 581:443-447; Reverberi *et al*. 2008. Eukaryotic Cell 7(6):988-1000.

Metabolic profiling of the tomato apoplast during infection by *Pseudomonas syringae*

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The plant pathogenic bacterium *Pseudomonas syringae* spends most of its parasitic life in the plant apoplast. *P. syringae* is able to colonize this niche by suppressing and manipulating plant defences through the secretion of proteins and toxins. Genomic analyses and nutritional assays indicate that the tomato pathogen *P. syringae* pv. tomato DC3000 is well adapted for growth using the nutrients present in the tomato leaf apoplast and shows nutritional specialization for growth in this environment relative to non-plant pathogenic *Pseudomonas.* This supports the hypothesis that some of the keys for successful colonisation of a susceptible host reside in bacterial adaptation to and modulation of the nutritional and physiological characteristics of the plant apoplast. However, the nutritional component of plant pathogenesis is relatively unexplored. We have taken a metabolomics approach to study the metabolic preferences of *P. syringae* in apoplast extracts *in vitro* and to investigate the changes that occur in apoplast composition during infection. Our ultimate goal is to identify, characterise and understand the metabolic interface between plants and *P. syringae* in the context of a susceptible interaction.

The role of protein transport in the physiology and pathogenicity of *Streptomyces* species

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Streptomyces scabies is a Gram-positive bacteria that causes common scab of potatoes but can also infect the roots of other plants. Its genome sequence has revealed that it is closely related to the non-pathogenic, soil-dwelling, saprophyte *Streptomyces coelicolor* but that it produces additional proteins involved in the infection process. Very little is known about how this pathogen delivers these

virulence factors into the host tissue. The twin-arginine translocation (Tat) pathway has been shown as a major route of protein secretion in *S. coelicolor* and, more recently, in *S. scabies*, and many Tat-secreted substrates are thought to contribute significantly to the virulence of *S. scabies*. The role played by the Tat system in this virulence is being investigated through a combination of gene knockouts and fluorescence microscopy. This latter approach has shown that a strain deficient in Tat transport is delayed in infection, and future work will attempt to elucidate the role of individual Tat substrates by investigating their localisation *in planta*. More recently, another protein export pathway, the Type VII secretion system, has been discovered in Gram positive bacteria. This system is essential for full virulence of both *Mycobacterium tuberculosis* and *Staphylococcus aureus*. However, non-pathogens also possess this system, which carries out a range of functions, including conjugal DNA transfer and iron / zinc homeostasis. Despite carrying gene orthologues, the type VII secretion system in the streptomycetes is completely uncharacterised. A proteomics approach is being taken to identify type VII substrates in *S. coelicolor* and *S. scabies* with a view to elucidating its physiological role and, in the case of the latter, its role in virulence.

Session III
Chair: Chris Thornton (University of Exeter)

Dickeya spp. - A new threat to potato production

Sonia Humphris[1], Emma Douglas[1], John Elphinstone[2], Neil Parkinson[2], Gerry Saddler[3] and Ian Toth[1]
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Erwinia chrysanthemi has been a problem for potato production in some European countries for over 40 years and in England for almost 20 years. Over the last 5 years, however, losses in some European countries due to these pathogens have increased by as much as five-fold. Although there has been many years of research carried out on *Dickeya dadantii* (strain 3937), this species is not responsible for potato disease in Europe. Instead, this appears to be caused by *D. dianthicola* and, more recently, a new group of *Dickeya* called DUC-1, for which little is known. This talk will discuss the current situation in Europe and highlight some of our recent research findings.

Molecular tools for the detection of phytoplasmas causing African 'lethal yellowing' diseases of coconut palms

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Lethal yellowing-type diseases of coconut are caused by phytoplasmas (bacteria with minimal genomes that lack cell walls, infect host phloem and are spread by sap-sucking insect vectors). They cause yellowing and death of fronds, and can kill palms within weeks of symptom development. Thus, they cause significant socio-economic problems in parts of East Africa, such as Tanzania and Mozambique, and in several regions of Western Africa, including Ghana. According to molecular analyses each of these three countries is infested with a distinct strain of lethal yellowing-type phytoplasma. Field trials are underway in Ghana to evaluate the performance of coconut cultivars reported to be resistant to 'Cape St. Paul Wilt' lethal yellowing-type disease, or tolerant of infection. These palms are being monitored for symptom development and for the presence of phytoplasmas. We have developed a sensitive molecular diagnostic technique for detection of African lethal yellowing pathogens based on nested PCR of the bacterial 23S ribosomal gene or the *secA* gene, followed by RFLP analysis. Preliminary data show that while many Ghanaian palms are infected with Cape St. Paul Wilt disease (some of which have succumbed and died, but several are tolerant of infection), a number of tested palms appear to contain DNA that corresponds to that of the Tanzanian lethal yellowing. Use of such diagnostics is valuable for phytoplasma research, and possible multiple infections by lethal yellowing pathogens would have implications for the evaluation of disease tolerance in coconut palms.

Recent evolution of *Rhynchosporium secalis* populations in response to selection by fungicides

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Barley leaf blotch or scald, caused by the fungus *Rhynchosporium secalis*, is a highly economically damaging foliar disease of barley in the UK, causing annual yield losses estimated at £4.8 million in 2005. Effective control relies on the combined use of more resistant barley varieties and fungicide applications, but host resistance can be overcome, and fungicide use can exert a strong selective pressure for resistance. A reduction in sensitivity to some triazole fungicides has been found in the field, and strobilurin resistance was reported for the first time in *R. secalis* at a site in Northern France in 2008.
Understanding the genetic basis of fungicide resistance enables improved detection through molecular diagnostics, facilitating a better understanding of the spread and management of resistance. This project aims to identify genetic changes responsible for reduced fungicide sensitivity in *R. secalis*, and to study their occurrence and spread in populations.
A fungicide sensitivity bioassay has been developed for *R. secalis*. This has revealed a 100-fold reduction in *in vitro* sensitivity to some triazoles over the last 10-15

years, but the resistance mechanism is not yet known. *R. secalis* has two copies of the gene, *Cyp51*, encoding the triazole fungicide target site. Investigations into the possible role of these genes in triazole sensitivity will be presented.

Molecular diagnostic tools for detection of oil palm (*Elaeis guineensis*) vascular wilt disease caused *by Fusarium oxysporum* f.sp. *elaeidis*

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Fusarium oxysporum f.sp. *elaeidis* (*Foe*) invades the host xylem and causes a devastating vascular wilt disease of oil palm in West and Central Africa and parts of South America. So far this disease has not been reported in South East Asia in spite of long term importation for breeding purposes of African seed and pollen, now known to be contaminated with *Foe*. Malaysia is the largest palm oil producer in the world and *Foe* remains a major threat to this industry. Therefore, this study is being conducted in order to help Malaysia avoid and/or be prepared for this potential problem. Molecular diagnostic tools are being developed for rapid detection and quantification of *Foe* in diseased plant tissue, soil, seed and pollen. These tools can be used for quarantine purposes of any imported materials and to test infection of putative resistant palm genotypes. It is not straightforward to develop molecular diagnostic tools for this disease because *Fusarium* is a very complex genus. Traditionally, at species level *Fusarium* has been distinguished based on morphological criteria such as colony morphology, formation of macro and micro conidia and production of chlamydospores. A forma specialis (f.sp.) is often assumed to have a single common ancestor from which all vegetative compatibility groups (VCGs) arise (monophyly). However, multiple VCGs and races within a given f.sp. could have multiple independent origins, with pathogenicity and virulence evolving more than once through mutation and/or transposition or spreading to distantly related strains through parasexuality or horizontal gene transfer. Thus, it is difficult to design specific primers to identify *Fusarium* species complex members as they may share high DNA sequence similarity in the aligned region. Nevertheless, we have managed to develop *Fusarium* genus-specific PCR assays based on the internal transcribed spacer (ITS) region of rDNA and *F. oxysporum*-specific primers within the translation elongation factor 1-α (TEF) gene. The specificity of the *Fusarium* genus-specific primers (Fusf1 and Fusr1) was assessed against forty-two *Fusarium spp.* isolates from various hosts and origins and seven closest out-groups to the genus *Fusarium*. Different isolates of *Fusarium* species, irrespective of formae speciales and race, were amplified with this primer and excluded all the out-groups. Furthermore, the species-specific primer pair Foxy F2/ EF2 was able to amplify a DNA fragment of all *F. oxysporum* spp. isolates tested and no amplification was observed for *F. redolens* and *F. foetens* as these species are phylogenetically in the same section with *F. oxysporum*. Currently, we are working on *Foe*-specific primers using the intergenic spacer region (IGS).

Molecular and Physiological Changes Induced in *Catharanthus roseus* During Phytoplasma Infection

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Phytoplasmas are specialised plant pathogenic bacteria. They are spread by insect vectors and within the plant are limited to the phloem. Phytoplasmas are responsible for many economically important diseases. Typical symptoms caused by phytoplasma diseases include phyllody (the replacement of flowers with leaf like structures) loss of apical dominance with increased branching and dramatically reduced leaf size. Very little is known about the molecular pathology of phytoplasma diseases.

Catharanthus roseus (Madagascan periwinkle) can be used as an experimental host for many phytoplasmas and gives a good range of representative symptoms, including phyllody. Infected plants may produce both fully phyllodous flowers and partially converted flowers. The epidermal characteristics of healthy flowers, partially converted flowers and fully phyllodous flowers were investigated using light microscopy and it was found that fully phyllodous flowers have an epidermal structure very similar to leaves.

Methylation sensitive AFLP was used to detect global changes in DNA methylation between healthy and phytoplasma-infected *C. roseus*. Two restriction enzymes were used to successfully detect different methylation status of plastid sequences between infected and healthy plants.

The Xspecies microarray technique allows genechips to be used with transcripts from species they were not originally designed for. Probe pairs are selected by assessing their hybridisation efficiency to the genomic DNA of the target organism, and data are removed from probes that fail to hybridise efficiently. The *Arabidopsis* ATH1 gene chip was used to analyse gene expression in *C. roseus* plants infected with Sweet potato little leaf phytoplasma. Sixty-six differentially regulated genes were identified using this technique. This included the down regulation of several photosynthesis-related genes, which fits with previous findings that phytoplasmas affect photosystem II. Up regulation of auxin-related genes was also seen, and such findings could help to uncover the method of symptom development in phytoplasma infected plants.

Factors which influence the expression of "brown mushroom" symptoms in crops infected with Mushroom Virus X

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Mushroom Virus X (MVX) is a syndrome which affects the output from crops of the button mushroom, *Agaricus bisporus*, by inducing symptoms such as crop delay, poor quality mushrooms, malformed pins and brown mushrooms in white strain crops. Symptoms were first observed on English farms in the mid nineties, by the late 90's Irish and Dutch farmers were also reporting symptoms. Double-stranded (ds) RNAs were associated with symptomatic samples, indicating a viral aetiology.
In Ireland the most commonly observed symptom is the presence of brown mushrooms in white strain crops. Brown mushroom symptoms have been found to be consistently associated with the presence of a complex of lower molecular weight dsRNAs. The expression of these 'browns' in crops infected with MVX is transient and inconsistent. Symptoms may appear in one growing room on a farm and not in others, or in one flush of a crop but not in the next.
Our current research focuses on identifying factors which influence the expression of symptoms. We are developing and modifying existing methods to detect the presence of the dsRNAs in both mushroom and compost samples, we have also introduced a chromameter as means of quantifying brownness.
Experiments are being carried out to examine the hypothesis that symptom expression is influenced by agronomic and/or environmental factors, including time of introduction of infective material to a crop. Our results to date, from several experiments, indicate that infection just prior to applying casing to the crop, produces significantly more brown mushroom symptoms compared to infection at other times. Anecdotal reports suggest that brown mushroom symptoms may be the result of adverse or stressful growth conditions. Results of an experiment under different agronomic and environmental conditions, in the absence of a source MVX inoculum, produced poor quality mushrooms but no brown symptoms were observed and the associated dsRNAs were not detected.

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Bacterial evolution by genomic island transfer occurs via DNA transformation *in planta*

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We have previously reported that the loss of the 106kb genomic island, PPHGI-1 from the bean pathogen *Pseudonomas syringae* pv. *phaseolicola* (*Pph*) strain 1302A

is driven by exposure to the stress imposed by the plants resistance reaction, the hypersensitive response (HR). We now show that this genomic island is able to horizontally transfer between strains of *Pph* by transformation during co-inoculation *in planta*. Transformation occurs at high frequency in the resistant host, bean cultivar Tendergreen (TG) but is reduced in susceptible hosts and extremely limited outside of the host. Transfer has also been achieved in the non-host plants, Tobacco and Arabidopsis. Limited transformation has been observed *in vitro*, under various conditions including at very low temperatures and in apoplastic fluid, the highest frequency being observed in apoplastic fluid extracted from TG leaves undergoing the HR. Our results suggest that both excision of the island and competence for transformation are enhanced within the microenvironment of the plant compared to culture. Transfer of PPHGI-1 leads to a change in virulence of the recipient strain and could have other implications to its fitness as PPHGI-1 contains genes clusters encoding type IV pilli, photosensory and chemotactic proteins and enzymes involved in DNA repair a recombination. These findings show pathogen evolution can occur by the simplest process of horizontal gene transfer and is particularly prevalent during host defence when the pathogen is in greatest need to acquire potentially new genetic traits to alleviate the antimicrobial stress imposed by the host.

Defence spending slashed in the hyperaccumulator economy?

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Thlaspi caerulescens is a 'metal hyperaccumulator'; a plant which takes up metals from its environment and stores them in its aerial tissues at surprisingly high concentrations, which would normally be expected to be toxic. The explanation for the evolution of this trait is uncertain, but the most popular theory proposes that hyperaccumulated metal smay act as a defence against pathogens or herbivores. We have tested the possibility of an 'elemental defence' against the pathogen *Pseudomonas syringae* and found evidence that this does occur in *T. caerulescens*. To further understand the nature of anti-pathogen defences in this plant, we have investigated many of the basal defences found in its relative, *Arabidopsis*. Surprisingly, we have found evidence that most of these defences are severely attenuated in *Thlaspi.* We are now comparing the levels of these defenses across a range of *Thlaspi* populations which vary in their levels of metal hyperaccumulation. We are also investigating the ability of *P. syringae* and *P. syringae* mutants with altered metal tolerance, as well as bacteria isolated from *Thlaspi* in the field, to grow in plants of each of these populations. Thus we hope to build up a clearer picture of which defences are most important to this unusual plant.

Bacterial plant pathogen interactions with predators: survival and spread

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Understanding the molecular mechanisms underpinning the ecological success of plant pathogens is key for developing strategies for the control of diseases and protection of crops. Recent observations have shown that plant pathogenic bacteria are not just interacting with the plant itself, but with a series of potential predators present in the soil, such as nematodes, insect larvae and amoebae. Bacteria are therefore under strong selective pressure to avoid or survive predation and we hypothesise that bacteria have evolved mechanisms to escape predation. The aim of this project is to identify the gene systems that contribute to the ecological success of the plant pathogen, *Pseudomonas syringae*, with a specific focus on anti-predation and pathogenicity mechanisms. A Rapid Virulence Annotation (RVA) screening was developed to identify which mechanisms allow the pathogen to cope against predators. This assay was developed to identify *P. syringae* cosmids that enhanced *Escherichia coli* survival in the presence of predators (nematodes, amoeba) and insect larvae. Approximately 2000 cosmids were screened against each predator and 150 found that reduced bacterial killing or killed the insect larvae of *Galleria mellonella*. Sample end-sequencing of ten cosmids was carried out. Bioinformatics analysis was used to identify the genes encoded on each cosmid, a task that was aided by the availability of the complete genome sequence. We identified a number of genes that could be contributing to anti-predation including Type 6 secretion system, hemolysin and insecticidal toxins. A comparison of the genes conferring resistance within others *P. syringae* strains showed differences in the organization of these clusters, indicating differential evolution. These cosmids will be mutagenized using a transposon and the contribution of each individual gene will be evaluated. Furthermore, gene expression *in vivo* will be identified by IVET (*In vivo expression technology*) screens. These data, will provide an important contribution to understanding how gene expression is controlled during the different phases of the bacterial life and to give insight to the ecological function of the genes.

New insights into eyespot resistance

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Eyespot is an important stem-base disease of wheat caused by two fungal species; *Oculimacula yallundae* and *O. acuformis.* The durable resistance exhibited by the variety Cappelle Desprez has previously been attributed to the partial resistance gene *Pch2* on chromosome 7A, and to an adult plant resistance on chromosome 5A. Our work provides new insights into the efficacy and genetic location of these resistances.

We found *Pch2* to be effective against *O. acuformis*, but to be ineffective against penetration from *O. yallundae*. We mapped *Pch2* resistance to *O. acuformis* to the distal end of chromosome 7AL and identified closely linked SSR markers. However, it was not possible to map any resistance to *O. yallundae* on chromosome 7A. This confirms that *Pch2* operates specifically against *O. acuformis* and has important implications for its use in commercial varieties. In addition, we have gained a further insight into *Pch2* through cDNA-AFLP expression studies, and have identified candidate genes involved with the resistance response.
We have found the adult plant resistance on chromosome 5A of Cappelle Desprez to also be effective in seedlings. Furthermore, we showed that it provides protection against both *O. yallundae* and *O. acuformis*. We have mapped the resistance as a single major QTL on chromosome 5AL, and have identified closely linked SSR markers. This resistance is a potentially useful resource to plant breeders and we propose it should be named *Pch4*.
In conclusion, our research provides a greater understanding into the function of these eyespot resistance genes and informs their use by plant breeders. In addition, the tools that we have developed enable them to be tracked by marker-assisted selection in breeding programmes.

A pathogenicity factor enriched micro-region in the *Fusarium graminearum* genome

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Using a novel bioinformatics approach, we have identified a genomic micro-region that appears to be enriched for verified pathogenicity genes in the important crop pathogen *Fusarium graminearum*. Analysis of this micro-region by a combination of bioinformatic and reverse genetics approaches is helping to ascertain its role in the pathogenicity of this species with the aim of locating novel virulence determinants.
Comparative genomics has been used to investigate conservation of the micro-region in other closely and less closely related species. Targeted deletion of nine separate genes has allowed the determination of the role of the micro-region in *F. graminearum* pathogenicity.
Deletion of the neutral trehalase gene *NTH1* appears to slow infection of wheat ears, while deletion of the *SNF1* protein kinase or *PKAR* cAMP-dependent protein kinase regulatory subunit inhibits pathogen spread from the inoculated spikelet. Targeted deletion of unannotated genes in this region has then allowed the identification of a novel virulence determinant, *FGSG_09907*.
This micro-region appears to be distinctly different from the virulence-associated biosynthetic and secreted protein clusters identified so far in pathogenic fungi. Further investigation will reveal more about the properties of this small genomic region.

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Resistance to *Pyrenopeziza brassicae* (light leaf spot) in a mapping population of oilseed rape

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Pyrenopeziza brassicae causes light leaf spot on winter oilseed rape (*Brassica napus*), one of the most important diseases of oilseed rape in the UK and northern Europe. Disease severity is currently reduced through a combination of (costly) foliar fungicide applications and cultivars with quantitative (polygenic) resistance. There has been one report of major gene-mediated resistance to *P. brassicae* (Bradburne *et al.*, 1999) and the oilseed rape cultivar Imola was bred from resistant material used in that study. This project aims to investigate the resistant phenotype of cv. Imola and to map the major resistance gene(s) against *P. brassicae* within the context of a linkage map constructed using the N26 doubled haploid (DH) population (derived from a cross between cv. Imola and 218-11, a susceptible breeding line).

Results presented here show that the N26 DH population is segregating for resistance to *P. brassicae* in a ratio of approximately 1:1 resistant:susceptible, suggesting that it is controlled by a single major gene. Susceptible DH lines show typical light leaf spot symptoms (asexual sporulation) when inoculated with *P. brassicae* conidia in both controlled environment and field experiments. Resistant DH lines show no asexual sporulation, but instead show a "black flecking" phenotype. Scanning electron microscopy reveals that this "black flecking" is associated with the collapse of groups of epidermal cells. This resistance phenotype is unusual because although *P. brassicae* cannot undergo asexual sporulation on resistant plants, there is still limited growth of mycelium and sexual sporulation (producing ascospores) has been observed on senescent debris of artificially-inoculated resistant plants.

We hope to provide breeders with markers linked to the resistance gene to make it easier to incorporate this major gene-mediated resistance against *P. brassicae* into new elite oilseed rape cultivars. A greater understanding of the mechanisms associated with the resistant phenotype should provide insights into the durability of the resistance, if it is incorporated into cultivars grown on a commercial scale.

Bradburne R, Majer D, Magrath R, Werner CP, Lewis B and Mithen R (1999) Winter oilseed rape with high levels of resistance to *Pyrenopeziza brassicae* derived from wild *Brassica* species. Plant Pathology 48: 550-558

The ABC-transporter BcatrB protects *Botrytis cinerea* against camalexin and is a virulence factor on Arabidopsis

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Arabidopsis thaliana produces the phytoalexin camalexin in response to abiotic and biotic stress. Here we studied the mechanisms of tolerance to camalexin in the fungus *Botrytis cinerea,* a necrotrophic pathogen on *A. thaliana*.
B. cinerea inoculated on WT *A. thaliana* plants yields smaller lesions than on camalexin-deficient mutants. Exposure of *B. cinerea* to camalexin induces the expression of several transporter genes, among which BcatrB, an ABC transporter that functions in efflux of fungitoxic compounds. The accumulation of the known BcatrB substrate ^{14}C-fludioxonil is increased by treatment with camalexin, indicating that the two compounds compete for active efflux and that BcatrB can transport camalexin. A *B. cinerea* strain lacking functional BcatrB is more sensitive to camalexin *in vitro* and less virulent on WT plants but is still fully virulent on camalexin mutants. Pre-treatment of *A. thaliana* with UV-C leads to increased camalexin accumulation and substantial resistance to *B. cinerea.* UV-C-induced resistance does not occur in camalexin-deficient *A. thaliana* mutants.
This is the first time that an ABC transporter is demonstrated to be a virulence factor by increasing tolerance of the pathogen towards a phytoalexin, combined with complete restoration of virulence on host plants lacking this phytoalexin.

Arabidopsis auxin mutants are compromised in systemic acquired resistance and exhibit aberrant accumulation of various indolic compounds

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Systemic acquired resistance is a widespread phenomenon in the plant kingdom that confers heightened and often enduring immunity to a range of diverse pathogens. Systemic immunity develops through activation of plant disease resistance protein signalling networks following local infection with an incompatible pathogen. The accumulation of the phytohormone salicylic acid in systemically responding tissues occurs within days after a local immunizing infection and is essential for systemic resistance. However, our knowledge of the signaling components underpinning signal perception and establishment of

systemic immunity are rudimentary. We have previously observed that an early, and transient, increase in jasmonic acid in distal responding tissues is central to effective establishment of systemic immunity. Based upon predicted transcriptional networks induced in systemic responding tissues we show that a variety of auxin mutants compromise the establishment of systemic immunity. Linking transcriptional and targeted metabolite studies our data provide evidence for a role of indole derived compounds, but not auxin itself, in establishment and maintenance of systemic immunity.