2nd Annual Conference of the SUSTAIN Action

15-17 October 2014, Zakopane, Poland

PROGRAM & ABSTRACT BOOK

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PATHOGEN-INFORMED STRATEGIES FOR SUSTAINABLE BROAD-SPECTRUM CROP RESISTANCE

2nd Annual Conference of the SUSTAIN Action

15th – 17th October 2014, Zakopane, Poland

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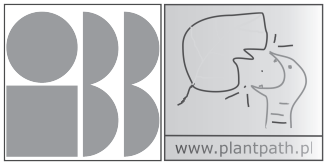

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Ewa Zimnoch-Guzowska – Mlochow Research Centre, Plant Breeding and Acclimatisation Institute, Poland

MEETING OVERVIEW

Wednesday 15th October	Thursday 16th October	Friday 17th October
8.45 – 10.10 **Welcome** **& WG1 presentations**	**8.45 – 9.50** **WG4 presentations**	**8.30 – 10.10** **WG2 presentations**
10.10 – 10.40 Coffee Break	10.00 – 10.30 Coffee Break	10.10 – 10.40 Coffee break
10.40 – 12.00 **WG1 presentations**	**10.30 – 11.50** **WG4 presentations**	**10.40 – 12.20** **WG2 presentations**
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13.40 – 14.40 **WG1 presentations**	**13.30 – 14.30** **WG4 presentations**	14.00 Departure to Krakow
14.40 – 15.25 **WG3 presentations**	**14.30 – 15.30** **Plenary Action meeting**	
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16.00 – 17.40 **WG3 presentations**	**15.30 – 16.45** **Poster session**	
18.30 Dinner	17.00 – 19.00 Zakopane sightseeing	
20.00 – 21.30 **Poster session**	19.00 Dinner & Live Music	

SCIENTIFIC PROGRAMME

TUESDAY October 14th

Participants arrivals
18.00 – 21.00 Dinner

WEDNESDAY October 15th

8.45 – 9.00 WELCOME
Thomas Kroj (Action Chair) and Magda Krzymowska & Marcin Filipecki (Local organizers)

Pathogen effectors and virulence (WG1)
Chairs: Bart Thomma & Jacek Hennig

9.00 – 9.45 *Keynote Lecture*
Jim Beynon (School of Life Sciences and Systems Biology, Warwick University, UK)
Networks in Plant Immunity

9.45 – 10.05 Guido Sessa (Department of Molecular Biology and Ecology of Plants, Tel Aviv University, Israel)
Xanthomonas campestris pv. *vesicatoria* type III effectors that suppress PTI signaling

10.10 – 10.40 COFFEE BREAK

10.40 – 11.00 Gunther Doehlemann (Max Planck Institute for Terrestrial Microbiology, Marburg, Germany)
Identification and functional analysis of Pit2-like protease inhibitors in plant-microbe-interactions

11.00 – 11.20 Mark Banfield (Department of Biological Chemistry, John Innes Centre, Norwich, UK)
Mechanisms of host cell manipulation by filamentous plant pathogen effectors and molecular studies of R-protein function

11.20 – 11.40 Diana Ortiz (INRA/Biology and Genetics of Plant-Pathogen Interactions, Montpellier, France)
The rice NB-LRR protein RGA5 recognizes the *Magnaporthe oryzae* effector AVR-Pia by direct binding

11.40 – 12.00 Charikleia Schoina (Laboratory of Phytopathology, Wageningen University, The Netherlands)
A novel *in vitro* infection system to study *Phytophthora*-host interactions

12.10 – 13.30 LUNCH (CATERING)

13.40 – 14.40 Bauters Lander (Department of Molecular Biotechnology, Faculty of Bioscience Engineering, Ghent University, Belgium)
Chorismate mutase and isochorismatase as possible effectors of the migratory nematode *Hirschmanniella oryzae*

14.00 – 14.20 Changhai Liu (Department of plant and environmental sciences, University of Copenhagen, Denmark)
Identification of effectors in the wheat stripe rust interaction

14.20 – 14.40 Barbara Geric Stare, (Agricultural Institute of Slovenia, Plant Protection Department, Ljubljana, Slovenia)
In planta activity and 3D model structure of effector expansin-like proteins (EXPB2) from plant parasitic nematode *Globodera rostochiensis*

Effector evolution and diversification (WG3)
Chairs: Didier Tharreau & Grzegorz Koczyk

14.40 – 15.25 *Keynote Lecture*
Bruce A. McDonald (Institute of Integrative Biology, ETH Zurich, Switzerland)
Effector evolution and diversification in necrotrophic fungi

15.30 – 16.00 COFFEE BREAK

16.00 – 16.20 Carmen R Beuzón (Instituto de Hortofruticultura Subtropical y Mediterranea, Universidad de Málaga, Consejo Superior de Investigaciones Científicas , Departamento Biología Celular, Genética y Fisiología, Spain)
Suppression of HopZ effector triggered defence responses as analternative force driving host adaptation and evolution on the HopZ effector family

16.20 – 16.40 Stephan Poppe (Max Planck Institute for Terrestrial Microbiology, Marburg, Germany)
Host specialization in the fungal plant pathogen *Zymoseptoria tritici*

16.40 – 17.00 Grzegorz Koczyk (Institute of Plant Genetics, Polish Academy of Sciences, Poznań, Poland)
The distant siblings – investigating the origins of secondary metabolite diversity

17.00 – 17.20 Sophien Kamoun (The Sainsbury Laboratory, Norwich, UK)
Effector specialization following a host jump in a lineage of the Irish potato famine pathogen

17.20 – 17.40 Bart Thomma (Wageningen University, The Netherlands)
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18.30 DINNER in Hyrny Hotel

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THURSDAY October 16th

R genes & host targets for resistance breeding and engineering (WG4)
Vivianne Vleeshouwers & Ewa Zimnoch-Guzowska

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Richard Oliver (Centre for Crop and Disease Management, Curtin University, Perth, Australia)
Exploitation of necrotrophic effectors to improve crop protection

9.30 – 9.50 Laura J. Stevens (Division of Plant Sciences, College of Life Sciences, University of Dundee & The James Hutton Institute, Dundee, UK)
Shuffling resistance genes to protect Solanaceous plants

10.00 – 10.30 COFFEE BREAK

10.30 – 10.50 Erik Slootweg (Laboratory of Nematology, Wageningen University, The Netherlands)
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10.50 – 11.10 Agnieszka Siwoszek (Department of Plant and Environmental Sciences, Faculty of Science, University of Copenhagen, Denmark)
Peptide aptamer-mediated resistance to barley powdery mildew

11.10 – 11.30 Klaas Bouwmeester (Laboratory of Phytopathology, Wageningen University, The Netherlands)
Lectin receptor kinases: sentinels in defense against plant pathogens

11.30 – 11.50 James Cockram (National Institute of Agricultural Botany Cambridge, UK)
Identification of effector sensitivity loci in wheat using highly recombinant populations genotyped with high--density SNP arrays.

12.00 – 13.20 LUNCH

13.30 – 14.30 Jadwiga Śliwka (Plant Breeding and Acclimatisation Institute – National Research Institute, Młochów Research Centre, Poland)
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13.50 – 14.10 Marek Żurczak (Institute of Biochemistry and Biophysics Polish Academy of Science, Warsaw, Poland)
Nuclear import of N receptor in tobacco is regulated by posttranslational modification of SGT1 protein

14.10 – 14.30 Emmanouil Domazakis (Wageningen UR Plant Breeding, Wageningen University and Research Centre, The Netherlands)
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14.30 – 15.30 Plenary Action meeting

15.30 – 16.30 MC Meeting

15.30 – 16.45 Poster session

17.00 – 19.00 ZAKOPANE sightseeing

19.00 DINNER & LIVE MUSIC in Karczma Sabała

FRIDAY October 17th

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A bacterial tyrosine phosphatase inhibits plant pattern recognition receptor activation

8.50 – 9.10 Hazel McLellan (The Division of Plant Sciences, College of Life Science, University of Dundee & James Hutton Institute; Dundee, UK)
An RxLR effector from *Phytophthora infestans* interacts with a family of potato PP1c phosphatases

9.10 – 9.30 Geert Smant (Laboratory of Nematology, Wageningen University, The Netherlands)
Secreted venom allergen-like proteins of plant-parasitic nematodes modulate defence responses in host plants

9.30 – 9.50 Boris Szurek (Résistance des Plantes aux Bioagresseurs, IRD, Montpellier, France)
Knowledge-informed discovery of *xa40*(t), a new broad-spectrum resistance gene controlling bacterial leaf blight of rice

9.50 – 10.10 Justin Lee (Leibniz Institute of Plant Biochemistry, Halle, Germany)
A bacterial effector protein specifically suppressed MAMP-induced activation of the MAPKs, MPK4 and MPK11

10.10 – 10.40 COFFEE BREAK

10.40 – 11.00 Marek D. Koter (Warsaw University of Life Sciences, Poland)
Small RNA dynamics in the tomato root response to Potato Cyst Nematode

11.00 – 11.20 Tolga O. Bozkurt (Imperial College London, Department of Life Sciences, UK)
Phytophthora infestans RXLR effector PEXRD54 activates plant autophagy by directly binding host ATG8 protein

11.20 – 11.40 Nemo Peeters (Laboratoire des Intéractions Plantes Microorganismes, UMR CNRS-INRA, France)
Functional analysis of the 30 core type III effectors from *Ralstonia solanacaearum*

11.40 – 12.00 Yuanyuan Mei (Department of Molecular Biotechnology, Ghent University, Belgium)
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12.30 – 13.45 LUNCH (CATERING)

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P	2.	Nuno	Almeida	RNA-seq of plant/pathogen interactions as a tool for deciphering effector genes
P	3.	Giuseppe	Andolfo	Tomato genome-wide transcriptional responses to Fusarium wilt and Tomato mosaic virus
P	4.	Balázs	Barna	Heat stress induces changes in possible effector targets and susceptibility of barley to powdery mildew
P	5.	Przemyslaw	Bidzinski	Drought impact on the expression of the plant defence arsenal and fungal aggressiveness in the interaction between *Magnaporthe oryzae* and rice
P	6.	May Bente	Brurberg	Transcriptome analysis of potato tubers during infection with *Phytophthora infestans*
P	7.	Irene	Camboni	Screening the phenome of a transposon tagged gene library in *Fusarium culmorum*, causal agent of Fusarium Head Blight and Fusarium Foot Rot on wheat
P	8.	Emilie	Chanclud	Are cytokinins new effectors from fungi?
P	9.	Anna	Coll	Functional characterisation of an Ethylene Response Factor involved in Potato- PVY Interaction
P	10.	Merete Wiken	Dees	Transcriptome analysis of early stages of potato tuber formation during infection with *streptomyces turgidiscabies*
P	11.	Amalia	Diaz Granados	Elucidating the mechanism of immune suppression by SPRYSEC effectors from potato cyst nematodes
P	12.	Armin	Djamei	*Ustilago bromivora* - Brachypodium, developing a suitable grass-pathogen model system for functional effector studies
P	13.	Abdelnaser	Elashry	RNAseq analysis of the *H. schachtii* transcriptome reveals new putative effectors
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P	16.	Barbara	Franco	Identification and characterisation of *Rhynchosporium commune* pathogenicity factors
P	17.	Barbara	Geric Stare	*In planta* activity and 3D model structure of effector expansin-like proteins (EXPB2) from plant parasitic nematode *Globodera rostochiensis*
P	18.	Fabian	Giska	Phosphorylation of HopQ1, a Type III Effector from *Pseudomonas syringae,* Creates a binding for host 14-3-3 proteins.
P	19.	Cynthia	Gleason	Characterization of novel root-knot nematode effectors
P	20.	Rafał	Hoser	Plant immunity suppression by natural variants of *Pseudomonas syringae* HopQ1 effector
P	21.	Ece Börteçine	Kasapoglu	Comparative transcriptome analysis of three cyst nematode species
P	22.	Ralf	Koebnik	*Xanthomonas translucens* – role of type III effectors in pathogenicity?
P	23.	Philippe	Lecomte	Using SILAC strategy to identify protein effectors in the wheat-Fusarium graminearum pathosystem
P	24.	Jana	Libantova	Antifungal potential of crude protein extract from carnivorous plant *Drosera rotundifolia*
P	25.	Xiao	Lin	Isolation and characterization of apoplastic immune receptors in potato; towards a novel type of durable resistance against late blight
P	26.	Krzysztof	Pawlowski	Bioinformatics prediction of a novel family of pseudokinase effector proteins present in several plant and animal pathogens

P	27.	Carsten	Pedersen	Functional analyses of barley powdery mildew effector candidates
P	28.	Marcin	Piechocki	Functional analysis of homologs of bacterial effector HopQ1
P	29.	Crina	Popa	AWR5 bacterial effector affects TOR signalling pathway in *Saccharomyces cerevisiae*
P	30.	Elżbieta	Różanska	Arabidopsis Sec21 protein is involved in development of syncytium induced by *Heterodera schachtii*
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P	32.	Anja Karine	Ruud	Necrotrophic effectors and sensitivity genes - gene-for-gene interactions in the wheat- *Stagonospora nodorum* pathosystem
P	33.	Remco	Stam	Quantitative transcriptomics of the *P. capsici* – host interaction
P	34.	Doron	Teper	Identification of new type III effectors of *Xanthomonas campestris* pv. *Vesicatoria* by using a machine learning approach
P	35.	Gaetan	Thilliez	Non host resistance in *Solanaceae.* The role of Phytophthora secreted effector in determining the host range
P	36.	Fabienne	Vailleau	The *Ralstonia Solanacearum Secretome*: type III effectors, type III associated proteins and their role in pathogenicity
P	37.	Keke	Wang	Functionnal analysis of Type III effector RipG7 of *Ralstonia solanacearum*
P	38.	Ronja	Wonneberger	Can susceptibility to net blotch in barley be explained by sensitivity to necrotrophic effectors?
P	39.	Elitsur	Yaniv	Fine-mapping of the *Rpt5* net blotch resistance gene region in barley

ABSTRACTS
OF ORAL PRESENTATIONS

Pathogen effectors and virulence (WG1)

Networks in Plant Immunity

Jim Benyon

School of Life Sciences and Systems Biology, Warwick University, Warwick, UK

Global food security is the major challenge for biological sciences. As plants cannot avoid environmental stress they have developed response networks to alleviate their impact. These networks are complex as they involve detection and signaling components as well as many feedback mechanisms to fine tune their output to avoid extreme consequences to the plant. To complicate the issue, at any one time a plant may be exposed to multiple stresses that will alter the output from the stress response networks induced. However, there is likely to be a significant overlap in the gene regulatory networks mediating response to environmental stress. We use two approaches to understanding these networks. We are taking a Systems Biology modeling approach to begin to elucidate the transcriptional response networks that underlie plant stress responses. We have carried out high resolution timecourse microarray analyses on plant responses to biotic (*Pseudomonas syringae, Botrytis cinerea*) and abiotic (high light and drought) stress. Using network inference we have defined, and are validating, a high level network regulating responses to multiple stresses. We are also building a protein-protein interaction network that underpins plant immunity to disease. We have use matrix two hybrid analysis to screen over 200 pathogen effector proteins against 12,000 plant proteins and have identified more than 200 potential interacting proteins. This has revealed that the immune system in plants is highly connected and involved in many different processes. Understanding the function of these networks will be key to developing novel stress resilient crop plants.

Identification of *Xanthomonas Campestris Pv. Vesicatoria* Type III effectors that suppress PTI signaling

Georgy Popov[1], Frederic Brunner[2], Guido Sessa[1]

[1]Department of Molecular Biology and Ecology of Plants, Tel Aviv University, Tel Aviv 69978, Israel.

[2]Department of Biochemistry, Centre for Plant Molecular Biology, Eberhard Karls University, Auf der Morgenstelle 5, D-72076 Tübingen, Germany.

The Gram-negative bacterium *Xanthomonas campestris* pv. *vesicatoria* (*Xcv*), is the causal agent of spot disease in pepper and tomato plants. *Xcv* pathogenicity depends on a type III secretion system (T3S) that translocates effector proteins into the plant cell. Collectively T3S effectors suppress plant PAMP-triggered immunity (PTI), alter metabolism and gene expression for the benefit of the pathogen. About thirty T3S effectors are encoded in the genome of the *Xcv* strain 85-10 and nine of them were previously implicated in PTI suppression. However, little is known about their molecular functions and plant targets. We used an *Arabidopsis thaliana* pathogen-free protoplast assay to identify a comprehensive set of *Xcv* 85-10 effectors that manipulate early PTI response signaling. Of 34 tested effectors, 18 suppressed flg22-dependent activation of a reporter gene under control of a typical PAMPinducible promoter (*pFRK1-Luc*). Among them, seven effectors also suppressed ABAdependent activation of a reporter gene driven by an ABA-inducible promoter, while eleven effectors were specific PTI suppressors. PTI signaling includes activation of mitogen-activated protein kinase (MAPK) pathways and phosphorylation of the MPK3 and MPK6 MAP kinases. Interestingly, expression of effectors with PTI-suppressing activity did not affect flg22 – mediated phosphorylation of MPK3 and MPK6 indicating that all eighteen effectors interfere with PTI signaling downstream to MAPK3/6.

Identification and functional analysis of Pit2-like protease inhibitors in plant-microbe-interactions

André N. Mueller[1], Marlen Breuer[1], Stefanie Glaeser[2], Peter Kämpfer[2], Gunther Doehlemann[1]

[1]Max Planck Institute for Terrestrial Microbiology, Karl-von-Frisch Str. 10, D-35043 Marburg, Germany

[2]Institut für Angewandte Mikrobiologie, Heinrich-Buff-Ring 26, D- 35392 Gießen, Germany

Ustilago maydis, the causative agent of corn smut disease, is a biotrophic plant pathogen that relies on living plant tissue to fulfill its life cycle. This lifestyle requires an efficient suppression of the plant's immune system which is accomplished by the secretion of effector proteins. In a previous study, the secreted effector protein Pit2 (Protein important for tumors 2) was found to be essential for tumor formation in infected plants [1].

A combination of yeast-two-hybrid and protease activity assays showed that Pit2 acts as an inhibitor of a set of defense related apoplastic plant cysteine proteases. Sequence comparison with Pit2 orthologs from related smuts identified a conserved 14 amino acid motif (PID14), which is required for protease inhibition and interaction. This domain is essential for virulence of *U*. *maydis* [2].

To elucidate what determines the target-specificity of Pit2 and its inhibitor-domain, Pit2- orthologs and chimeric Pit2--versions are tested for functionality in the *U*. *maydis*-maizepathosystem. Interestingly, we could identify a highly conserved PID14-motif in Streptomycetes. To identify putative protease inhibitors from Streptomycetes and other root colonizing bacteria, maize endophytice bacteria have been isolated for a functional screen on PID14-activity.

[1] Doehlemann *et al*. 2011. *Mol. Micobiol.* 81: 751–766
[2] Mueller *et al.* 2013. *PLoS Pathog 9: e1003177*

Mechanisms of host cell manipulation by filamentous plant pathogen effectors and molecular studies of R-protein function

Mark J. Banfield

Department of Biological Chemistry, John Innes Centre, Norwich Research Park, Norwich, NR4 7UH, UK

A mechanistic understanding of how plant pathogens re-program their hosts to enable colonisation, and how plants respond to attack, may provide novel genetic or chemical opportunities to interfere with the development of disease. Bacterial and filamentous eukaryotic plant pathogens secrete effector proteins both outside of and into plant cells to suppress host defences and manipulate other cellular processes. Structural studies to describe how filamentous eukaryotic plant pathogen effectors directly interact with their host targets *inside cells* are currently lacking. Such knowledge is critical for understanding the molecular mechanisms underlying the *in planta* activities of these proteins, and for developing genetic approaches to managing disease. Yeast-2-hybrid and co-immunoprecipitation approaches have identified host proteins that interact with specific effectors from *P. infestans* and are of relevance to plant immunity. We have been exploiting biophysical, structural and *in planta* approaches to study these interactions. Also, pathogen effectors, or their activities, can be recognised by intracellular host immune receptors (NB-LRR or Rproteins) leading to restriction of pathogen growth. We have recently obtained structural information for certain domains of these receptors from crop plants that are predicted to be involved in recognition and molecular switching. We are currently following up on these studies. In this presentation I will give updates on our progress in these areas.

The rice NB-LRR protein RGA5 recognizes the *Magnaporthe oryzae* effector AVR-Pia by direct binding

Diana Ortiz[1], Karine de Guillen[2], Véronique Chalvon[1], Andre Padilla[2], Thomas Kroj[1]

[1] INRA – UMR BGPI, Campus International de Baillarguet TA A54/K 34398 Montpellier cedex 5, France

[2] CNRS-INSERM, Centre of Structural Biology, 29 rue de Navacelles, 34090 Montpellier

Plant immunity relies on direct or indirect recognition of pathogen effectors by plant resistance (R) proteins. This recognition activates disease-resistance signaling pathways leading to the inhibition of pathogen growth and the induction of a localized programmed cell death called the hypersensitive response (HR). To gain a better understanding of the molecular mechanisms governing effector recognition in plants, we study the translocated effector Avr-Pia from the rice blast fungus *Magnaporthe oryzae* and its recognition by the rice nucleotide-binding and leucine-rich repeat domain (NB-LRR) proteins RGA4 and RGA5. Yeast two-hybrid and co-immunoprecipitation experiments revealed physical interaction of AVR-Pia to an unconventional domain in RGA5 related to the yeast copper chaperone ATX1 (RATX1 domain). This suggests that AVR-Pia recognition occurs by direct binding and that RGA5 acts as an effector receptor. This hypothesis is supported by the finding that a polymorphic site, present in a natural allele of AVR-Pia, abolishes Avr activity and RGA5-binding. A three dimensional structure model of AVR-Pia was generated using nuclear magnetic resonance spectroscopy (NMR) and the polymorphic site was mapped on the model identifying a region which may be important for RGA5- binding and resistance induction. Point mutations were introduced in this region and elsewhere in AVR-Pia by site-directed mutagenesis and analyzed by yeast two hybrid and transient expression in *Nicothiana benthamiana*. By this, six additional amino acids, crucial for interaction with RGA5 and activation of defense responses were identified. They are located either in the previously identified region or in other sites of the molecule suggesting that different parts of AVR-Pia are engaged in physical interaction with RGA5.

A novel *in vitro* infection system to study *Phytophthora*-host interactions.

Schoina, C., Govers, F., & Bouwmeester K.

Laboratory of Phytopathology, Wageningen University, Wageningen, 6708 PB, The Netherlands. klaas.bouwmeester@wur.nl

One of the most devastating plant diseases world wide is late blight on potato and tomato caused by the oomycete pathogen *Phytophthora infestans*. During the early biotrophic phase of infection, *Phytophthora* penetrates host tissue and thereafter forms specialized feeding structures called haustoria. Here, effectors produced by the pathogen, are transferred into the host cells to manipulate the host cell machinery thereby suppressing plant defense. Therefore, studying the interface between the host and the pathogen at the early stages of infection is of great interest. An important drawback when studying the *Phytophthora*-host interaction in leaves is the lack of synchronization of the infection process. For this purpose, a new *in vitro* infection system was established, in which MsK8 tomato cell suspensions were challenged with zoospores of different *Phytophthora* species. Here we show that *P. infestans* infects MsK8 cells in a similar fashion as tomato leaf tissue. In contrast, other *Phytophthora* species that are not pathogenic on tomato could not penetrate the MsK8 cells. Expression analyses of *Phytophthora* effector and tomato defense genes and various histological assays were performed to monitor *Phytophthora*-MsK8 interactions in more detail. In addition, multi-omic datasets were genereated (i.e., transcriptome, metabolome, proteome), and are currently integretated and analyzed in a systems biology approach to elucidate the essential processes during *Phytophthora*-MsK8 interaction. The use of this novel infection system allows simplification and synchronization of the infection process, and is expected to provide a more detailed insight into *Phytophthora*-host interactions.

Chorismate mutase and isochorismatase as possible effectors of the migratory nematode *Hirschmanniella oryzae*

Bauters Lander, Kyndt Tina, Gheysen Godelieve

Department of Molecular Biotechnology, Faculty of Bioscience engineering, Ghent University, Ghent, Belgium

The rice root nematode *Hirschmanniella oryzae* is the most abundant plant-parasitic nematode in flooded rice fields and is distributed world-wide. Although it is economically less important than sedentary nematodes, it can cause severe yield reductions and economic losses in specific environmental conditions. The recently sequenced transcriptome of this nematode can give us some more insights into the interactions of this pathogen with its host plant. The transcriptome data was used to identify putative effector proteins. One of the identified genes encodes a chorismate mutase (*Hocm*), an enzyme involved in the production of some amino acids and a large range of secondary metabolites, among which salicylic acid. The full sequence was cloned, the gene structure was investigated and activity was assessed by performing a complementation assay in *E. coli*. Transgenic rice plants overexpressing *Hocm* were generated. qPCR results indicate that the defense mechanisms of these transgenic plants are disturbed. Next to *Hocm*, another gene involved in the salicylic acid pathway was detected in the transcriptome: isochorismatase (*Hoicm1*). The gene structure of *Hoicm1* was determined and its activity was assessed. qPCR on transgenic rice plants overexpressing *Hoicm1* showed that this gene is also able to interfere with defense pathways of the host. Research is ongoing to unravel the mechanisms by which these two possible effector genes act on the host.

Identification of effectors in the wheat stripe rust interaction

Changhai Liu, Carsten Pedersen, Hans Thordal-Christensen

Department of plant and environmental sciences, University of Copenhagen, Denmark

Stripe rust, caused by the biotrophic fungus, *P. striiformis* f. sp. *tritici* (*Pst*), is globally the most prevalent and damaging disease on wheat. It is well known that pathogens employ effectors to interfere with host defence. However, no effector from stripe rust has been identified until now but genome and transcriptome sequencing has revealed many effector candidates (Cantu et al. 2013 and Garnica et al. 2013).

We selected some effector candidates, which are highly expressed in haustoria and tested their function in tobacco (*Nicotiana benthamiana*) and wheat (*Triticum aestivum*) by delivering effectors into plant cells using the type three secretion system (T3SS) of *Pseudomonas fluorescence*. In *N. benthamiana*, REFC6 (Rust EFfector Candidate 6) significantly suppressed the ROS accumulation, ion leakage and callose deposition induced by *Pseudomonas fluorescence*. Similarly, REFC6 also compromised defence in wheat and enhanced susceptibility to stripe rust. Localization analysis showed that five out of six effector candidates localized in nuclear and cytoplasm in wheat leaves after transient expression by particle bombardment. We also used VIGS (virus induced gene silencing) to study effectors function. Hyphal length and branching significantly reduced when we knock down rust effector RTP1, which is shown playing roles in fibril formation (Kemen et al. 2013).

We speculate that REFC6 might be working in the PTI signalling pathway. The next challenge is to find out what REFC6 targets in plants. We will try immunoprecipitation and mass spectrometry, which has been used by other groups in finding protein targets *in planta.*

In planta activity and 3D model structure of effector expansin-like proteins (EXPB2) from plant parasitic nematode *Globodera rostochiensis*

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Infective larvae (J2) of potato cyst nematodes secrete effector proteins which facilitate successful infection of the host plants. Expansins are one group of the effectors, which help in degradation of plant cell wall by loosening non-covalent interactions between components of the plant cell wall. Molecular variability of *exp*B2 gene was shown in diverse populations of the *Globodera rostochiensis.* In this study we have shown the *in planta* expression and activity of GrEXPB2 protein. Systemic expression of GrEXPB2 type protein (CAC84564.1) induced chlorosis and dwarfing in *Nicotiana benthamiana* while variant EXPB2 proteins although expressed had no effect. Further GrEXPB2 type protein induced cell death in tomato and potato, but no symptoms in *N. tabacum.* Variant proteins induced no visible symptoms in tomato. Model of 3D structure for GrEXPB2 type protein was determined based on previously determined crystal structure of EXPB1 from maize (PDB: 2hczX). All three AA changed in the variant proteins are positioned on the outer surface of the protein model. Small changes (1 or 2 AA change compared to type protein GrEXPB2) resulted in inactive EXPB2 protein variants. These changes probably affect the interaction with polymers of the cell wall and the proteins function as well as the 3D structure of the protein (one variant possibly affecting a disulphide bond).

Effector evolution and diversification (WG3)

Effector evolution and diversification in necrotrophic fungi

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The first population genetic analysis of a plant pathogen effector was conducted with the NIP1 protein of *Rhynchosporium commune* and published in 2004. This work established the global significance of both non-synonymous substitutions and deletions as important mechanisms for avoiding detection by host R-genes at the field scale. The NIP2 and NIP3 effectors of *R. commune* have also been well characterized in global populations and global diversity for three host-specific toxins in *Parastagonospora nodorum* (ToxA, Tox1, Tox3) has also been established. All six of these effectors have unique features, but all also share some general patterns of diversity. For example, deletions occur for all six effectors in natural field populations, but the frequency of the deletion varies according to the effector and the field population analyzed. It is clear that each effector is operating under evolutionary constraints likely due to fitness costs associated with different mutations. There is also compelling evidence for horizontal gene transfer for several of these effectors. We are now identifying effectors in *Zymoseptoria tritici* using a combination of RNAseq, QTL mapping and population genomics. Several candidate effectors have been identified and one has been functionally validated. Several more effector candidates are under functional investigation. We anticipate conducting detailed population genetic analyses of these effectors and also expect to track their evolutionary history based on comparisons with the wild sister species *Z. pseudotritici* and *Z. ardabiliae*.

Suppression of Hopz – Effector triggered defence responses as an alternative force driving host adaptation and evolution on the Hopz Effector Family

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The majority of the Type III Effectors (T3E) investigated suppress plant defence responses. Owing to this, the zig-zag model proposed by Jones and Dangl in 2006, has become universally accepted to be driving evolution of bacterial effectors and plant resistance. Nevertheless, evolution of effector families is rarely explained taking into account that the selective pressure against Effector-Triggered Immunity (ETI) in a given host may be compensated by an ETIsuppressing effector encoded by the same strain.

The *Pseudomonas syringae* HopZ effector family, one of the best characterized effector families from an evolutionary point of view, is one of the most diverse, displaying the highest rate of horizontal transfer among pathovars. Changes in the sequence of the different members of the HopZ family have been proposed to prevent detection within their respective hosts, without losing out on their virulence activities, in a process called pathoadaptation (Ma et al., 2006).

Our results show that different HopZ effectors do not trigger immunity in their respective pathosystems, as their mutation usually lead to a small decrease in growth within the plant. However, expression of these effectors from heterologous strains does trigger immunity within the same hosts, indicating that the hosts can detect their presence and activate a defence response. Thus, the strains naturally encoding the HopZ effectors prevent HopZ-triggered immunity likely through the action of a suppressing effector. We identify the effectors suppressing HopZ3 triggered immunity, and propose cross-suppression of ETI as an alternative force driving evolution of the HopZ family.

Host specialization in the fungal plant pathogen *Zymoseptoria tritici*

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Positive selection is one crucial feature of genes involved in the interaction of plant pathogenic fungi and their hosts according to the arms race that has been described for this interaction. Here we show that genome comparison and evolutionary analyses of the very closely related hemibiotrophic, ascomycete plant pathogen species *Zymoseptoria tritici* (synonym: *Mycosphaerella graminicola*), *Zymoseptoria pseudotritici* and *Zymoseptoria ardabiliae* can be used to identify positively selected genes putatively participating in this interaction. Despite the recent speciation of *Z. tritici* and *Z. pseudotritici* that happened 10.500 years ago in the Fertile Crescent they are adapted to different hosts and environments. While *Z. tritici* evolved in an agroecosystem and is specialized to wheat, both ancestral species infect a variety of wild grass species. The close relationship of the pathogens and the availability of the genomes are making this a unique model system to study the evolutionary processes behind the speciation and specialization of plant pathogens.

In this study we deleted three genes in *Z. tritici* which we identified in a comparative genome study as they show signatures of positive selection between the three pathogen species. Two deletion strains showed reduced virulence on wheat while the deletion of the third gene led to a hypervirulent phenotype and all three genes are expressed only during the infectious phase of the pathogen. The most intriguing result however is the generation of a signal peptide in one of the candidate genes in *Z. tritici* that is absent in the wild grass pathogens suggesting a new function of the according protein in *Z. tritici* in comparison to the wild grass pathogens. This could be one possible method of a pathogen to adapt and specialize to its host plant.

The distant siblings – investigating the origins of secondary metabolite diversity

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Diverse secondary metabolites play important roles as effectors driving the interactions between plant-associated fungi and other species (whether symbiotic or pathogenic). However, the available resources aiming to put large data sets (polyketide and terpene synthases, major families of accessory genes) in context of evolutionary origins of their diversity (e.g. duplication, speciation, loss and horizontal transfer) are still scarce.

We show results of inquiries into core biosynthetic gene phylogenies, resulting from their reconciliation with species phylogeny inferred from a conserved set of single-copy housekeeping gene orthologues. This allowed for batch annotation of evolutionary events (duplication, speciation, transfer and loss) contributing to the extant biosynthetic gene families in over 140 model fungal genomes. For the purpose of detecting monophyletic patterns in synteny, domain architecture and gene structure, we devised and assessed a statistical approach to validating and pinpointing conserved traits positively correlated with tree topology. Thus we were able to both provide points of origin for trait gain/loss (strongest correlations with monophyletic clades), as well as postulate chains of inferred evolutionary events and their relative (topological) dating. Our results highlight the emergence and changes of ancestral traits within the respective biosynthetic gene families (e.g. biosynthesis of orsellinic acid derivatives, acquisition of beta-lactamase termination mechanism for C6-C11 polyketide cyclisation), as well as indicate that maximally parsimonious reconciliations allow consistent prediction of likely horizontal transfer events.

Effector specialization following a host jump in a lineage of the Irish potato famine pathogen

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Accelerated gene evolution is a hallmark of pathogen adaptation following a host jump. However, the biochemical basis of adaptation and specialization to new hosts remains largely unknown. We describe functional specialization of a plant pathogen effector following a host jump. Orthologous protease inhibitor effectors from the Irish potato famine pathogen *Phytophthora infestans* and its sister species *Phytophthora mirabilis* are better adapted to protease targets from their respective host plants potato and *Mirabilis jalapa*. Single amino acid polymorphisms in the inhibitors and their target proteases largely underpin biochemical specialization. These results provide a molecular framework for how antagonistic pleiotropy drives effector specialization in a plant pathogen ultimately resulting in diversification and speciation.

Evolution and biological function of effectors of the vascular wilt pathogen *Verticillium dahliae*

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Verticillium dahliae, causal agent of vascular wilt disease, is one of the most notorious plant pathogens of tomato. By comparative population genomics, we previously identified the race 1 specific effector that activates tomato immunity mediated by the cell surface receptor Ve1. Further comparative genomics revealed extensive genomic rearrangements between individual *V. dahliae* isolates that could be implicated in the occurrence of lineage-specific regions involved in niche adaptation and virulence. Remarkably, genes that reside in lineage-specific regions are over-represented during *in planta* expression. Moreover, in contrast to candidate effector genes that reside in the core genome, targeted disruption of several lineage-specific effector genes resulted in compromised virulence. One of the lineage-specific effectors that could be implicated in virulence on tomato is a LysM effector, a homolog of the Ecp6 effector of the foliar tomato pathogen *Cladosporium fulvum* that functions in suppression of chitin-triggered host immunity. Structural analysis revealed a novel mechanism for chitin binding by Ecp6 through intrachain LysM dimerization, leading to a composite binding site that binds chitin with ultra-high affinity.

Considering the importance of lineage-specific regions for *V. dahliae* aggressiveness, detailed genomic information on the recombination sites is required to understand how lineage-specific regions arise. To this end, we re-sequenced a *V. dahliae* genome with PacBio technology, leading to a gapless assembly of eight complete chromosomes. Our efforts to identify genomic signatures at the recombination sites and the molecular mechanism(s) that establish chromosomal rearrangements will be discussed.

R genes & host targets for resistance breeding and engineering (WG4)

Exploitation of necrotrophic effectors to improve crop protection

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The role of necrotrophic effectors in promoting virulence can be exploited as a way to select more resistant germplasm. Resistance to necrotrophic diseases was typically found to be partial, in contrast to the major gene resistance noted in some cases for biotrophic pathogens. This has meant that breeding for disease resistance is much more demanding and explains why necrotrophic pathogens have grown in importance whilst progress in controlling biotrophic diseases was often rapid (until the emergence of the next mutant pathogen race). However the identification and production of cloned and expressed effectors of necrotrophic pathogens allows breeders to select introgressions that are insensitive. Effectors from both Parastagonospora nodorum and Pyrenophora tritici-repentis have been ex-pressed in microbial systems and used to identify germplasm that is insensitive to the effector Thus, in the case of multi-effector systems like P. tritici-repentisand P. nodorum, selection of cultivars insensitive to each effector promises to assists breeders improve disease resistance in an incremental, step-wise fashion. Progress and complications in this process will be discussed and reviewed.

Shuffling resistance genes to protect Solanaceous plants

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The potato resistance protein R3a recognises the essential effector Avr3a_KI from the oomycete pathogen *Phytophthora infestans*, causing a hypersensitive response (HR) in the host which prevents further spread of infection. However, the product of the closely related virulent allele *avr3a_EM*, which is homozygous in 72% of *P. infestans* isolates in the Toluca Valley, Mexico, evades recognition and is able to cause disease on plants expressing R3a.

Gain-of-function variants of R3a (R3a*) were engineered by an iterative process of random mutagenesis and PCR shuffling. Studies with the best-performing R3a* variants in the Solanaceous model host *Nicotiana benthamiana* reveal a gain--of-recognition of avr3a_EM as well as enhanced disease resistance towards *P. infestans* isolates.

Recognition of Avr3a_KI by wild-type R3a occurs in the host cytoplasm and both proteins subsequently re-localise to pre-vacuolar compartments (PVCs). avr3a_EM does not cause this relocalisation. Confocal microscopy has revealed that R3a* variants re-localise to PVCs upon coinfiltration with either form of Avr3a, indicating a shared mode-of-recognition between wt R3a and R3a* variants. It is hoped that a shuffled *R3a** gene, deployed with other *R* genes in potato, would confer durable resistance to late blight disease.

Deconstructing a molecular switch; structure-function analysis of the CC-NB-LRR proteins Rx1 and Gpa2

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Many plant and animal immune receptors have a modular nucleotide-binding-leucine-rich repeat (NB-LRR) architecture in which a nucleotide-binding switch domain, NB-ARC, is tethered to a coiled coil (CC) and a LRR sensor domain. The cooperation between the CC, switch and sensor domains, which regulates the activation of these proteins, is poorly understood. Through targeted mutagenesis, interaction studies and structural modelling we attempt to investigate the way the domains of the potato resistance proteins Rx1 or Gpa2 work together. We demonstrated that the correct cooperation between the CC-NB-ARC and the LRR is focussed on a region in the ARC2 subdomain of the NB-ARC and the N-terminal repeats of the LRR. A mismatch leads either to autoactivation or loss-of-function. Complementary charged surface patches in the ARC2 and LRR are key determinants of the physical interaction between the domains, but do not appear to be involved in signalling. Furthermore alanine-substitution of specific aromatic residues in the CC domain shows that distinct surfaces are required for the interaction of the CC with the NBARC – LRR or with the protein RanGAP2. The CC domain of Rx1 consists of 4 helices and mutagenesis of the hydrophobic residues required for their interaction revealed that distinct parts of the CC are involved in either the cell death or PVX resistance signaling by Rx1. Currently we try to characterise how the binding of RanGAP2 and the recognition of specific elicitors affects these interdomain interactions and subcellular localisation of the resistance protein in the cell.

Peptide aptamer-mediated resistance to barley powdery mildew

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Barley powdery mildew, *Blumeria graminis* f. sp. *hordei* (*Bgh*) is a biotrophic fungus that develops feeding structures (haustoria) in the living plant cells. Around each haustorium, the host cell generates an extrahaustorial membrane (EHM). In order to control the host plant, fungi secrete effector proteins that are believed to inhibit host defence mechanisms. Approximately 500 barley powdery mildew effector candidate proteins (CSEPs) have been identified, showing no homology to any known proteins outside powdery mildew fungi (Pedersen et al., 2012). The only common feature of 63% of those putative effectors is YxC-motif in the N-terminal of the mature protein (Godfrey et al., 2010). This feature makes it a very interesting target in engineering disease resistance. Peptide aptamers are an excellent tool for this purpose, because of their small size, high recognition specificity and high binding affinity to their target.

In this study, we applied yeast two-hybrid screen to identify peptide aptamers targeting the YxC motif of CSEPs. This interaction was confirmed by bimolecular fluorescence complementation assay in *Nicotiana benthamiana*. Analysis of peptide aptamer-CSEPs interactions based on point mutations within the motif showed it to be YxC-motif specific, which suggests that the identified peptide aptamers should be able to target the majority of CSEPs. Localization studies showed that both peptide aptamers and CSEPs are localized to the plant cytosol and nucleus. Transient overexpression of selected peptide aptamers in barley epidermal cells resulted in lower susceptibility to barley powdery mildew. *Arabidopsis thaliana Bgh*-susceptible mutants were used to create overexpression lines of selected peptide aptamers. Those plants will be challenged with barley powdery mildew to verify disease resistance mediated by peptide aptamers.

Lectin receptor kinases: sentinels in defense against plant pathogens

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Plant breeders continuously face the challenge to obtain new cultivars with adequate levels of resistance to a variety of pathogens. In recent years, we identified a novel type of immune receptors that could be employed as disease resistance components towards both *Phytophthora* and bacterial pathogens. This resistance is mediated by cell surface receptors belonging to the family of L-type lectin receptor kinases (LecRKs). LecRKs are wide-spread in plants, and justifies exploitation of *LecRKs* as novel sources of crop resistance. The *LecRK* multi-gene family in Arabidopsis consists of 45 members, and their individual role in defense was determined in a genome-wide phenotypic analysis of T-DNA insertion mutants in infection assays. We found that multiple LecRKs play a role in resistance to a variety of plant pathogens, and that overexpression of various LecRKs enhances disease resistance. In addition, we screened for LecRK-Interacting-Proteins (LIPs) usingmass spectrometry. LecRK interaction was confirmed for one of the candidates, LIP1, in co-immunoprecipation assays. Further functional analysis showed that Arabidopsis *Lip1* mutants are compromised in *Phytophthora* resistance in a similar fashion as *LecRK* mutant lines. Understanding how LecRK-mediated resistance is functioning is crucial to design novel resistance in crops against *Phytophthora* and bacterial plant pathogens.

Identification of effector sensitivity loci in wheat using highly recombinant populations genotyped with high-density SNP arrays

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Molecular markers have arguably had limited impact on wheat resistance breeding because diseases are generally controlled by multiple, weak QTL for which markers are hard to define and deploy. The identification of necrotic effectors (NEs, proteins secreted by the fungal pathogen that can cause disease symptoms) in SNB, TS and other pathogens represents a paradigm shift within the field of phytopathology. Effector sensitivities are Mendelian traits, and so can be easily deployed by breeders and researchers. This complexity reduction allows advances in wheat genomics and biological resources to be applied for the rapid stacking of wheat resistance alleles.

Using association mapping (AM) and Multi-parent Advanced Generation Inter-Cross (MAGIC) populations genotyped with the Illumina iSelect 90k SNP array, we have used purified protein effectors from SNB and TS to identify wheat host sensitivity loci using seedling test assays. Proof of concept using TOXA (SNB/TS) identifies peak SNPs 2 genes away from the cloned *Tsn1* locus. Subsequent experiments using TOX1 and TOX3 (SNB) identify the wheat *Snn1* and *Snn3* resistance loci on chromosomes 1B and 5B, respectively. Available genomic resources are being deployed for map-based cloning of *Snn3*.

Fine mapping of the *Rpi-rzc1* gene for resistance to potato late blight

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The *Rpi-rzc1* gene originates from *Solanum ruiz-ceballosii*, a wild diploid relative of the cultivated potato. It provides high levels of resistance to late blight both in detached leaflet and in tuber slice tests. The gene has been mapped to potato chromosome X using 114 progeny from the interspecific cross between dihaploid of *S. tuberosum* and *S. ruiz-ceballosii* as a mapping population and Diversity Array Technology (DArT) markers. The *Rpi-rzc1* is linked to a potentially useful phenotypic marker, that is violet flower colour, conferred by the locus *F*. Genetic distance between *Rpi-rzc1* gene and locus F is 3.4 cM, while on the other side the gene is flanked by marker T1521 located in the distance of 6.1 cM.

To increase the precision of the genetic map we expanded the mapping population with another 240 individuals originating from the same cross. These individuals were assessed for resistance to *Phytophthora infestans* in detached leaflet tests and their flower colours were evaluated. Ten sequence-specific PCR markers were scored in the enlarged mapping population: two were derived directly from the NBS-LRR homologs from the potato genome sequence, two were identified by the Resistance Gene Enrichment Sequencing (RenSeq) approach and the remaining six – from other mapping studies. On the resulting map the *Rpi-rzc1* gene is flanked by markers located 0.3 and 0.7 cM from it. Narrowing down the chromosome sector containing the *Rpi-rzc1* gene to 1 cM will improve the efficiency of marker-assisted selection and will be useful for studies aiming at cloning the gene.

Nuclear import of N receptor in tobacco is regulated by posttranslational modification of SGT1 protein

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Resistance to Tobacco Mosaic Virus is mediated by the tobacco N protein, which belongs to the TIR-NB-LRR class of R proteins. NB-LRR receptors recognize in the cytoplasm proteins delivered by pathogens into plant cells, and this event initiates downstream signaling pathways. Subsequently, R proteins are transported to the nucleus where they play a key role in regulation of defense-related gene expression. However, little is known how nucleocytoplasmic transport of those receptors is regulated.

Most of NB-LRR receptors, including N, require the SGT1 protein to execute their function. SGT1, HSP90 and RAR1 form a molecular chaperone complex, which maintains R proteins in an inactive but signal-competent state.

Our studies revealed that SGT1 is specifically phosphorylated by MAP kinase. Plants expressing SGT1 phospho-null or phospho-mimic mutants in an endogenous SGT1-silenced background are more susceptible to pathogen attack. The phosphorylation state of SGT1 affects its distribution within cells, i.e. 50% of cells expressing phosphomimic variant, compared to 25% of cells expressing phospho-null or wild-type SGT1, display nucleocytoplasmic localization of SGT1.

Here, we show that forced nuclear import of SGT1 leads to increased nuclear accumulation of N protein. Co-localization studies of SGT1 with N derived domains revealed that SGT1 may regulate LRR nuclear import and export, whereas localization of TIR and NB domains was unaffected. We propose a model in which nucleocytoplasmic partitioning of N protein during TMV-triggered defense response is modulated by SGT1 phosphorylation.

Currently we are studying if oligomers formed by N's splice variants affect their nucleocytoplasmic transport.

Potato Sobir1 and Sobir1 – like interact with the elicitin – response receptor (ELR) of potato and are involved in the response to INF1 elicitin of *Phytophthora Infestans*

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Late blight, caused by the oomycete *Phytophthora infestans*, is the most threatening disease of potato (*Solanum tuberosum*, *St*). So far, plant breeding for late blight resistance was focused on the introduction of resistance (*R*) genes, which encode cytoplasmic receptors of the NB-LRR family. However, these introduced resistances have been defeated by the pathogen rather quickly. Currently, we are investigating whether immunity that is triggered upon perception of apoplastic pathogen elicitors can enhance late blight resistance. Previous work in our group led to the identification of ELR, a pattern recognition receptor (PRR) that recognizes several elicitins, a highly conserved family of secreted effectors of *Phytophthora* and *Pythium* spp. *ELR* encodes a receptor-like protein (RLP) and provides broad spectrum recognition of various elicitins in potato. Since RLPs do not have a kinase domain, they were anticipated to require a co-regulatory receptor-like kinase (RLK) that does carry a kinase domain to initiate cytoplasmic signaling. Indeed, it was recently described that several RLPs form a constitutive complex with the RLK SOBIR1. To study whether ELR associates with SOBIR1, we cloned several *StSOBIR1* and *StSOBIR1*-like homologues from wild potato accessions. We found that these genes are highly conserved in *Solanum* spp. and coimmunoprecipitation experiments showed that all StSOBIR1 variants physically interact with ELR. Virus-induced gene silencing (VIGS) studies of the *Nicotiana benthamiana SOBIR1* homologues resulted in a compromised response to INF1 of *P. infestans* in *N. benthamiana*. Together, our results suggest that StSOBIR1 and StSOBIR1-like are involved in the INF1 response by associating with ELR.

Plant proteins and processes targeted by effectors (WG2)

A bacterial tyrosine phosphatase inhibits plant pattern recognition receptor activation

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The first active layer of plant innate immunity relies on the perception of pathogen-associated molecular patterns (PAMPs) by surface-localised pattern-recognition receptors (PRRs). Many known plant PRRs are receptor kinases, which are annotated as serine/threonine kinases. However, the exact phosphorylation events that lead to receptor activation and initiation of PAMP-triggered immune signaling remain unknown. Here, we report that the Arabidopsis receptor kinase EFR, which perceives bacterial EF-Tu (or the derived peptide elf18), is phosphorylated on tyrosine residues and that this modification is critical for EFR activation upon ligand binding. We identify a single tyrosine residue required for EFR activation, downstream responses and immunity to the phytopathogenic bacterium *Pseudomonas syringae*. Pathogenic bacteria employ type--III secreted effectors to suppress PAMP-triggered immunity and cause disease. The effector HopAO1 is important for the virulence of *Pseudomonas syringae* and possesses tyrosine phosphatase catalytic activity, but its plant targets were still unknown. We found that HopAO1 directly interacts with EFR and FLS2, and blocks elf18-induced EFR activation and immune responses, revealing that HopOA1 targets tyrosine phosphorylation of plant PRR to block their activation. Our results shed light on a novel regulatory mechanism controlling plant immune signaling and highlight a host-pathogen battle to take control of PRR tyrosine phosphorylation that is critical for anti-bacterial immunity.

An RxLR effector from *Phytophthora infestans* interacts with a family of potato PP1c phosphatases

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Phytophthora infestans is an economically important pathogen of both potato and tomato crops. This pathogen secretes a large number of effector proteins into its host in order to manipulate plant defences and metabolism to benefit its growth and reproduction. However, relatively little is known about what these effectors do to modulate their host targets. We have identified a *P. infestans* RxLR effector Pi04314 which is up-regulated during infection. When this effector was screened in Y2H it was found to interact with several phosphatase isoforms belonging to the PP1c family, this interaction has been confirmed *in planta* by pulldown assays. Using confocal microscopy, fluorescent protein fusions of Pi04314 and StPP1c both localise individually to the nucleus and the nucleolus. However, in the presence of Pi04314 most but not all StPP1c

isoforms are no longer detected in the nucleolus. In addition, expression of Pi04314 *in planta* was found to strongly enhance *Phytophthora* colonisation suggesting a role in virulence for this effector. A myristolated form of this effector which no longer accumulates in the nucleus was no longer able to enhance *P. infestans* colonisation demonstrating the importance of effector sub-cellular localisation for virulence.

Secreted venom allergen-like proteins of plant-parasitic nematodes modulate defence responses in host plants

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The venom allergen-like proteins form a family of effectors that seems to be conserved among all parasitic nematodes of plants and animals studied to date. Recently, we have shown that the venom allergen-like protein of the potato cyst nematode *Globodera rostochiensis* Gr-VAP1 interacts with the apoplastic cysteine papain-like proteases Rcr3pim of *Solanum pimpinellifolium*. Gr-VAP1 and Rcr3pim are both required to activate defencerelated programmed cell death and resistance to nematodes mediated by the extracellular plant immune receptor Cf-2 in tomato. Thus, Gr-VAP1 is able to trigger defence responses in a host plant of *G. rostochiensis*, but the virulence function of Gr-VAP1 or of any other venom allergen-like protein of an animal- and plant-parasitic nematode is not known. A specific knock-down of *Gr-VAP1* expression in *G. rostochiensis* showed that the effector is indeed important for virulence of infective juveniles in host plants. Similarly, the ectopic expression of venom allergen-like proteins in transgenic plants alters their response to nematodes and other plant pathogens. RNAseq analysis of these transgenic plants has shed light on the molecular mechanisms underlying the virulence function of venom allergen-like protein of plant-parasitic nematodes in plants.

Knowledge-informed discovery of *xa40*(t), a new broad-spectrum resistance gene controlling bacterial leaf blight of rice

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Plant disease-susceptibility (*S*) genes are pathogen-induced host genes that are required for pathogen survival and proliferation. Altered host susceptibility alleles, also viewed as recessive disease-resistance alleles, provide new sources of resistance for durable control of plant diseases. *OsSWEET14* which is a major *S* gene for Bacterial Leaf Blight of rice, is transcriptionally induced by *Xanthomonas oryza* pv. oryzae (*Xoo*) Transcription Activator-Like (TAL) effectors through their specific binding to the *OsSWEET14* promoter. We screened a collection of wild and cultivated rice accessions for polymorphism in the *OsSWEET14* promoter region, and identified an allele with a deletion of 18 bp predicted to alter the DNA binding element of several major virulence TALs from strains of various geographic origins and lineages. We show that *Xoo* strains relying on these TALs for virulence are “disarmed” when inoculated on plants harboring the *OsSWEET14* promoter variant. This is the first report of successful knowledge-based molecular screen for a new plant resistance gene which we call *xa40*(t).

A bacterial effector protein specifically suppressed MAMP-induced activation of the MAPKs, MPK4 and MPK11

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Mitogen-activated protein kinases (MAPKs) regulate multiple cellular signalling pathways, including developmental processes and stress responses in plants. Exposure to pathogens or microbe-associated molecular patterns (MAMPs) induces the rapid activation of at least four MAPKs (namely MPK3, MPK6, MPK4 and MPK11). These MAPKs target various downstream substrates to dictate the appropriate defence reactions against the pathogenic invasion. MPK3 and MPK6 are associated with the signalling pathway dealing with positive regulation of the pathogen response. MPK4, on the other hand, acts in a second MAPK signalling branch that is linked to negative control of SA-regulated defence. MPK11 is highly homologous to MPK4, with some partially overlapping functions to MPK4. Several pathogen effector proteins have been shown to subvert antimicrobial defence responses by inactivating MAPKs. An example is the *Pseudomonas syringae* HopAI1, a phosphothreonine lyase, which irreversibly modifies the activation loop of MAPKs, thus preventing their phosphorylation and activation. However, such effectors can target all four MAMPactivated, as well as other, MAPKs. We present here another *P. syringae* effector that specifically suppresses the MAMP-induced MPK4/MPK11 activation but not the MPK3/MPK6 pathway. The suppressive effect is dependent on the effector enzymatic activity. Thus, this presumably represents a virulence function of this effector and would imply that the MPK4/MPK11 must have positive signalling functions for defence activation. In view of the lethality or developmental defects of *mpk3 mpk6* or *mpk4 mpk11* double mutants, understanding the molecular mechanisms of such effectors may aid the design of tools for interfering with specific MAPK pathways.

Small RNA dynamics in the tomato root response to Potato Cyst Nematode

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Plant cyst nematodes (PCN) infect roots and induce formation of multinuclear syncytium, a specialized structure becoming a sole food source for developing larvae and adults. The formation of syncytium is accompanied with active suppression of defense response as well as substantial reprogramming of development and metabolism of incorporated cells. Such process are likely to engage different mechanisms of gene expression regulation including those mediated by small RNAs. Since down-regulation of genes upon plant parasitic nematode infection is a common phenomenon concerning a third part of regulated genes we decided to purify and sequence the sRNA fraction of infected root transcriptome.

The tomato roots were grown in vitro and infected with PCN. Root fragments with syncytia were collected and RNA was isolated and fractionated. The indexed sRNA libraries were sequenced using Illumina MiSeq genome sequencer. Resulting sequences were analyzed using UEA sWorbench 3.1 and potential target genes were identified using psRNATarget server. The results show many changes in composition of sRNA profiles. 54 known miRNAs have been found with 6 of them manifesting over 2-fold induction/suppression as compareds to control samples. Their predicted target genes in tomato genome are transcription factors (GRAS, MYB, NAM), proteins involved in signal transduction (glucose/ribitol dehydrogenase, phosphatidate cytidyltransferase) and LRR receptor-like serine/threonine-protein kinase. Substantial portion of target candidates are likely to participate in other stress responses. Several miRNA homologues identified, such as mir1446, mir164 and mir399 are up-regulated in infected tissues showing the potential mechanism of plant response suppression by cyst nematodes.

Phytophthora Infestans RXLR effector PEXRD54 activates plant autophagy by directly binding host ATG8 protein

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Autophagy is a bulk process of degrading dysfunctional cellular components for survival under starvation conditions. In animal immune systems, selective autophagy plays key roles in degrading invading intracellular bacteria to prevent infection. In response, some bacterial pathogens secrete effector proteins that inhibit autophagy to enable parasitic infection. However, the role of autophagy in plant microbe interactions, and the extent to which it mediates focal immunity remain poorly known. We discovered that the Irish potato famine pathogen *Phytophthora infestans* deploys a host-translocated effector, PexRD54, a member of the RXLR-WY family, which accumulates at the haustorial interface and activates a selective autophagy process. PexRD54 specifically targets and stabilizes ATG8, a key component for regulation of autophagy in eukaryotic organisms. Remarkably, PexRD54 binds ATG8 both *in vivo* and *in vitro* via its C-terminal ATG8 interacting motif (AIM). Moreover, PexRD54 colocalizes with ATG8 at autophagosomes and enhances both the number and size of these endomembrane compartments. Consistent with these observations, an AIM mutant of PexRD54 failed to interact with and stabilize ATG8 and was inefficient in activating autophagy. Finally, both ATG8 and PexRD54 are recruited at the haustorial interface, indicating deployment of an autophagy pathway towards haustoria. Our work provides insight into the biogenesis of the extrahaustorial membrane and reveals an effector targeted host autophagy process that is recruited at the haustorial interface.

Functional analysis of the 30 core type III effectors from *Ralstonia solanacaearum*

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By pan-genomic analysis we identified a core set of 30 type III effectors in the plant pathogenic bacterium *Ralstonia solanacearum* [1]. We have recently embarked in the systematic functional analysis of these type III effectors in order to gain insight in their contribution to the pathogenicity of this bacterium on tomato.

I will present our current advancement on the functional characterization of these type III effectors by presenting our data on yeast-2-hybrid screening of a tomato root library; plant sub-cellular localization and contribution to virulence of type III effector mutant and overexpressing *R. solanacearum* strains.

1. Peeters N, Carrère S, Anisimova M, Plener L, Cazalé A-C, Genin S: Repertoire, unified nomenclature and evolution of the Type III effector gene set in the Ralstonia solanacearum species complex. *BMC Genomics* 2013, 14:859.

Insight into the functions of *Globodera pallida* SPRYSEC proteins

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The potato cyst nematodes, *Globodera spp.*, are the most damaging nematodes in the UK. They impose an annual cost in excess of £50 million to growers. Due to a lack of major resistance genes and public concerns about nematicides, it has become vital to understand the molecular basis of the interactions between this pathogen and its host plant in order to develop novel sustainable control strategies.

G.pallida is a biotrophic pathogen. The interaction between this nematode and its host is mediated by effector proteins that are synthesized in nematode oesophageal glands and injected into host cell cytoplasm through the stylet. The genome

sequencing project of this nematode has identified a large gene family encoding SPRYSEC proteins that includes more than 300 members. Many *SPRYSEC* genes are expressed in the dorsal gland cells of parasitic stage juveniles (J2) indicating their potential roles in plant parasitism. Potential host virulence targets of SPRYSEC proteins have been identified by yeast two-hybrid screens and these include a carotenoid cleavage dioxygenase 4 (CCD4) protein. RNAi silencing of this target dramatically increases the susceptibility of potato to *G. pallida*. Previous work has shown that a potato variety that has naturally lower *CCD4* gene expression and higher carotenoids content is also more susceptible to *G. pallida*. Our data may indicate that a SPRYSEC protein is involved in manipulating host carotenoids either to fulfil the dietary requirements of the nematode or to change levels of precursors of various plant hormones such as abscisic acid (ABA).

ABSTRACTS
OF POSTER PRESENTATIONS

P1 Mahinur Akkaya

Effectors and microRNA like small RNAs in pathogen- host interaction

Bayantes Dagvadorj[1], A. Caglar Ozketen[2], Ayse Andac[2], Kubra Narci[3], Burak Demiralay[4], Zemran Mustafa[2], Adnan Yaramis[1], Evrim Elcin[1], Mahinur S. Akkaya[1,2,3,4]

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Our study is focused on two basic routes in understanding the pathogen plant interaction. One of which is to investigate the function of the candidate effectors detected computationally based on the public pathogen genome sequence data. Toward this end, candidate effectors of *Puccinia striiformis* f. sp. *tritici* (Pst) known to be expressed either during germination of spores or in the host are cloned and preliminary experiments of their subcellular localizations were conducted. We are also carrying out bioinformatics based clustering analysis for better categorized Pst effectors. The other major focus of our study is to identify microRNA like small RNAsof *Blumeria graminis* f. sp. *hordei* and *Priformaspora indica* targeting host gene messages to promote or suppress disease formation, respectively. Number of known and (mirLN) miRNA like novel RNAs are (mirLN) detected based on small RNA sequencing data we obtained. Preliminary data will be presented at the meeting.

P2 Nuno Almeida

RNA-seq of plant/pathogen interactions as a tool for deciphering effector genes

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Rusts are among the most important plant diseases. As biotrophic fungi, there is a requirement to sustain infected host cells alive for their development, increasing the need for an efficient cross-talk between host and pathogen. Rusts are also known for the frequent breakdown of resistance due to the rapid evolution of these pathogens. RNA-seq can provide valuable information about plant/pathogen interactions, allowing the identification and quantification of expressed sequences potentially involved in plant resistance and in the pathogen attack. Using this approach on resistant and susceptible *Lathyrus* accessions, we identified the potential effectors involved in the *Uromyces pisi*/*Lathyrus sativus* and *U. pisi*/*L. cicera* interactions. From the 4558 putative fungal transcripts, 891 encoded potential secreted proteins, as predicted by SignalP and TargetP algorithms.

From these, a selection of effector proteins will be used as probes to identify the target host proteins as an initial step towards the development of effector-driven legume breeding approaches, maximizing the potential of durability of resistance against such a quickly evolving pathogen.

P3 Giuseppe Andolfo

Tomato genome-wide transcriptional responses to Fusarium wilt and Tomato mosaic virus

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Multiple omics approaches offer new insights of plant-pathogen interactions, owing to the availability of high throughput biological data and computational resources. In order to identify a set of genes of interest in tomato plants infected with *F. oxysporum f. sp. lycopersici* (*Fol*) and Tomato Mosaic Virus (*ToMV*) a transcriptional analysis was performed. A large overlap in differentially expressed genes throughout the two incompatible interactions was found. However, gene ontology enrichment analysis evidenced specific functional categories enrichment in both interactions. Genomic mapping of differentially expressed genes suggested that definite genomic regions are involved in pathogen resistance response. In two specific interactions the chromosome distribution of expressed genes showed wide overlapping regions, except for the region holding genes *I2* and *Tm2*. Indeed, genome regions can be enriched in genes with the specific function for fine-tuning gene expression in a compensatory way. A high level of NB-LRR genes activation and of other gene classes potentially involved in pathogen recognition was also found, suggesting a host-coordinated reaction of defense machinery to monitor integrity of cellular proteins. The NB-LRR system minimizes the cost of defense for the plant, as multiple NB-LRR proteins can be maintained at a low level in the absence of a pathogen. Albeit still fragmented, our depiction provides a global view of the *Fol* and *ToMV* mediated resistance process, in which the gene expression network may be considered the starting point to construct a genomic model for *R* mediated response in the two pathosystems investigated.

P4 Balazs Barna

Heat stress induces changes in possible effector targets and susceptibility of barley to powdery mildew

Balazs Barna

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Heat pre-treatment (49 oC for 30 sec.) significantly increased the number of *Blumeria graminis* f. sp. *hordei* (*Bgh*) colonies on susceptible, the number of lesions on Mla resistant barley lines, and some sporulation could be observed on Mlg and even on mlo resistant plants!

Microscopic investigations showed that heat pretreatment improved the development of secondary germ tube, haustorium formation and suppressed papilla formation, and epidermal hypersensitive response (HR), in addition to epidermal H2O2 formation as indicated by DAB staining.

As a possible effector target integrity of plant membranes was measured by conductivity. Heat stress caused faster, while powdery mildew infection later leakage of ions from barley leaf segments. As another possible effector targets soluble proteins from treated and/or infected leaves were analysed by 2D PAGE. In addition, we found by RT-PCR that expression of Hsp70 was induced both by heat stress and powdery mildew infection.

It was reported recently, that a superfamily of barley powdery mildew effector candidates shows structural affinities to ribonucleases (RNases). PAGE and spectrophotometric assays showed significant increase of RNase activities after heat stress and *Bgh* infection especially in the susceptible line.

The possible involvement of the above changes in elevated susceptibility of barley will be discussed.

P5 Przemyslaw Bidzinski

Drought impact on the expression of the plant defence arsenal and fungal aggressiveness in the interaction between *Magnaporthe oryzae* and rice

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It is well-documented that plants with enhanced tolerance to drought often display increased susceptibility to pathogens, suggesting a massive negative cross-talk between the two biological processes (Mittler, 2006). However, little is known about plant defence against pathogens when they are under drought stress (Prasch and Sonnewald 2013). Furthermore, there is no report on the molecular strategies pathogens adopt when infecting already-stressed plants. Given the available knowledge on genes involved in resistance (receptors, signalling, defence arsenal...) and aggressiveness (developmental regulators, nutrition, effectors...), we chose the model interaction between rice and *Magnaporthe* to characterize how drought is impacting on plant/pathogen interaction.

We have developed a drought protocol that does not significantly affect plant growth but strongly increases susceptibility (2-fold increase in fungal biomass). Surprisingly, preliminary cytological analysis showed slower fungal progression on the leaf surfaces of droughtstressed plants within first day after inoculation. However, two days after inoculation, the development of the fungus was similar and at three and four days after penetration into the plant cells, the fungal growth was higher in stressed-plants than in un-stressed plants. This increase in fungal growth could be attributed to either reduced resistance of the stressedplants or increased aggressiveness of the fungus, or both. Preliminary data suggest that the observed increased susceptibility of the plants may rather be a consequence of an increase of aggressiveness of the fungus. RNAseq data are being produced to further explore this hypothesis.

P6 May Bente Brurberg

Transcriptome analysis of potato tubers during infection with *Phytophthora infestans*

Merete Wiken Dees[1], Mads Sønderkær[2], Monica Skogen[1], Even Sannes Riiser[1], Vinh Hong Le[1], Kåre Lehman Nielsen[2], May Bente Brurberg[1]

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Spoilage of potatoes in storage, by microbes, is a world-wide problem that causes big losses in agriculture, industry and trade. The problem with many of the disease causing organisms is the fact that they do not cause symptoms when the products are harvested, but later during the storage the quality of the products is deteriorating and the symptoms appear. This is common especially when there is free water on the products. In a joint Nordic project we study the changes taking place in potato pathogens and potato tubers during storage in different humidity conditions to understand how the physiology of the pathogens and the host is affected by disease-promoting storage conditions, using Illumina Hiseq seqencing technology for gene expression profiling. Here we report results from a storage experiment using the susceptible cultivar Bintje infected with *Phytophthora infestans*, where samples were harvested 24 and 48 h after infection. In total 40 libraries were sequenced, which generated more than 387 million reads. The majority of the reads were mapped to the potato genome (v4.01; 80% identity over 80% of read length). Hierarchical clustering and principal component analysis were used to find potentially biological relevant gene expression patterns. A large number of genes associated with anoxic conditions as well as defence responses were identified.

P7 Irene Camboni

Screening the phenome of a transposon tagged gene library in *Fusarium culmorum*, causal agent of Fusarium Head Blight and Fusarium Foot Rot on wheat

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High-throughput methods are needed for functional genomics analysis of *Fusarium culmorum*, causal agent crown and foot rot and Fusarium Head Blight on wheat and a type B trichothecene producer. Here we present a double-component system based on the ability of the *impala* transposase to heterologously activate the miniature inverted-repeat transposable element *mimp1* of *Fusarium oxysporum* to generate a library of transposon-tagged genes in *F. culmorum*. A phenomic analysis that includes time dimension (evolution of phenotypes with time) for library screening has been implemented. Pathogenicity, fungicide resistance and fitness characters are determined. Addition of time dimension measures for screening the mutant's library phenotype may help generating better understanding of each gene role in *F. culmorum*. As an example, we show that large scale phenomic measures with spectrophotometric approaches do guarantee a finer and more sensible identification of gene inactivation effects. We are currently focusing on the identification of genes with unknown function and key genes involved in the plant – pathogen – environment interactions.

P8 Emilie Chanclud

Are cytokinins new effectors from fungi?

Emilie Chanclud, Véronique Chalvon, Thomas Kroj, Jean-Benoit Morel

Cytokinins (CKs) are hormones known to be involved, in plants, in developmental processes and in regulation of important metabolism pathways (mainly carbon and nitrogen). Recently their roles in plant and microorganism interactions emerged.

Some bacterial and fungal plant pathogens have been described to be able to secrete CKs. Taking into account their roles in plant physiology, it has been proposed that producing CK could be an advantage for pathogens to manipulate in their host this hormonal pathway.

Jiang et *al* (2012) showed that the rice blast fungus, *Magnaporthe oryzae*, produces and secretes some CKs but the role of these fungal-derived hormones is not established. It has been hypothesized that during the earlier stages of infection, CKs could serve as effectors to trigger an uptake of nutrient in infected cells that would favor fungal growth. Unexpectedly, our previous results showed that some CKs can increase rice resistance against this pathogen. Thus the role of CKs as effectors of pathogenicity in the interaction between rice and *M.oryzae* is still unclear.

We identified a putative CK biosynthesis gene in *M.oryzae*. We created knock-out mutants for this gene. Since a secondary biosynthesic pathway could exist in fungi, we also generated mutants that overexpress a gene that encodes for an enzyme which de-activates most CKs by oxidation, *OsCKX2*. Preliminary results showed that *in vitro*, *M.oryzae* CK mutants displayed a delay in germination. This phenotype could be complemented by exogenous CK application. The pathogenicity of *M.oryzae* mutants affected in CK metabolism will be presented.

P9 Anna Coll

Functional characterisation of an Ethylene Response Factor involved in Potato- PVY Interaction

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Potato *(Solanum tuberosum L.)* is the world's most widely grown tuber crop and potato virus Y (PVY) is one of the major potato pathogen causing severe crop loss in different areas worldwide. To better understand the potato defence response against PVY we studied the role of *ethylene response factor* (*ERF*) genes from group IX since they have been related to plant defence response and defined as important elements on hormone crosstalk.

Potato *ERF-IX* genes were identified and classified in this study. Among them, *StERF* was selected for further analyses based on previous transcriptomics experiments performed in our group. Expression patterns of the gene in hypersensitive resistance (HR) potato cultivar infected with PVY pointed to its importance as a signalling component in potato defence response. Using virus-induced gene silencing (VIGS) we demonstrated that PVY systemic spread is delayed in *StERF* silenced plants. We further examined the potential hormonal signalling involved in the expression of *StERF* and demonstrated that our gene integrates several signalling pathways. Getting more insights into the regulation of the gene, localisation studies showed that StERF strongly accumulated in cell nucleus after PVY infection.

Taken together our results suggested the importance of *StERF* in potato-PVY interaction. Therefore the data contributes to better understand the complex network of plant defence signalling pathways.

P10 Merete Wiken Dees

Transcriptome analysis of early stages of potato tuber formation during infection with *streptomyces turgidiscabies*

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Common scab is an important disease that has a pronounced effect on potato crops worldwide because it degrades the appearance of the tubers and thereby decreases market value. The disease is caused by soil borne, Gram positive bacteria of the genus *Streptomyces.* Although common scab has been recognized for more than a century, knowledge is still limited regarding the factors that contribute to the occurrence and severity of this disease. Furthermore, there is currently no completely successful method of controlling common scab. Disease resistant potato cultivars would be the best and most desirable control strategy, but most of the cultivars that are commercially available today are very susceptible, and none of them have proven to be completely resistant. One of the goals of our project is to obtain detailed knowledge about potato genes involved in the infection process to better understand the mechanism associated with resistance to potato common scab. A pot experiment was performed in the greenhouse with the aim to study differentially regulated genes in the potato plant during infection with *Streptomyces turgidiscabies*. One tolerant (Beate) and one susceptible (Saturna) potato variety were used in the experiment. Altogether, 32 pots were inoculated with a mix of three different *S. turgidiscabies* isolates originating from different places in Norway. Uninoculated plants of the two varieties were used as control. Samples were taken from the potato plants at different stages of tuber development early hookstage, hookstage, early stage of tuber formation and middle stage of tuber formation. Genes that are expressed during infection of the potato plant with *S. turgidiscabies* will be studied using Illumina Hiseq seqencing technology for gene expression profiling.

P11 Amalia Diaz Granados

Elucidating the mechanism of immune suppression by SPRYSEC effectors from potato cyst nematodes

Diaz Granados, A. , Overmars, H., Postma, W., Slootweg , E. , Bakker, J. , Goverse, A., Smant G.

Persistent nematode infections are a major threat to important food crops. These round worms manipulate plant cell morphology and physiology to establish sophisticated feeding structures. Remarkably, they are able to escape detection by the plant immune system, despite their size and the damage they cause to the host upon infection. Modifications to plant cells are largely attributed to the activity of nematode secreted effectors. It has been recently shown that members of the SPRYSEC effector family from potato cyst nematodes selectively supress host cell death and disease resistance mediated by plant immune receptors. We aim to unravel the, as of yet, unknown molecular mechanism behind immune suppression by these effectors. Evidence supports a model where SPRYSECs function as specificity modifiers of host ubiquitin complexes for nematode-induced proteasomal degradation of host immune receptors. Experiments with protein-protein interaction methods, directed mutagenesis and protein ubiquitination assays among other techniques, will enable us to substantiate our model. If indeed the diverse SPRYSEC effector family functions as versatile suppressors of immunity, the expansion of this family may reflect adaptations to diversification of plant immune receptors. Therefore our results may provide insight into the basis for virulence of nematodes in plants.

P12 Armin Djamei

***Ustilago bromivora* – Brachypodium, developing a suitable grass-pathogen model system for functional effector studies**

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Two-sided functional effector-studies with grass infecting pathogenic filamentous fungi are often hampered by the difficulties of reverse genetic approaches on the host side. In a recent report (1), the smut *Ustilago bromivora* was identified to infect *Brachypodium distachyon.* This model grass is closely related to barley, wheat and other important crop plants. Brachypodium has a short generation times of 6-8 weeks, and stable transformations take only a few months. The smut fungus *Ustilago bromivora* is therefore currently evaluated by us for its suitability as a new monocot infecting model system. I will report about our current progress in establishing culture conditions, transformation of the fungal as well as of the plant side and the development of genetic tools to speed up functional effector research in the future. Furthermore the first analysis of the genome of *Ustilago bromivora* will be presented.

1.) Barbieri, M. *et al.* Host Status of False Brome Grass to the Leaf Rust Fungus Puccinia brachypodi and the Stripe Rust Fungus P. striiformis. Plant Disease 95, 1339-1345, doi:10.1094/pdis-11-10-0825 (2011).

P13 Abdelnaser Elashry

RNAseq analysis of the *H. schachtii* transcriptome reveals new putative effectors

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The beet cyst nematode (BCN) *Heterodera schachtii* depends on a set of effectors for the induction and maintenance of the syncytium inside the root. However, little is known about the type and role of such effectors in the process. The identification of new putative secretory proteins (PSPs) can lead to the discovery of new BCN effectors. We used RNAseq analysis to identify putative secretory proteins (PSPs) that have their transcripts upregulated during the post infective stages, since such proteins are expected to play a role in process of parasitism. By comparative analysis with other available plant-parasitic nematode sequences, we further selected for plant-parasitic specific nematode specific PSPs (PP-PSPs). The annotation of the identified PP-PSPs showed enrichment in particular gene ontologies, such as metabolic and catalytic protein activities in addition to those regulating of cell growth and development. In order to validate PP-PSPs, we are using *in situ* hybridization to verify PP-PSP gene expression within the esophageal glands. Gene knock-down approaches by RNAi are being used to determine the role on infectivity of *H. schachtii*. Our results can lead to the identification of new types of effectors important for the parasitism strategies of BCN in particular and cyst nematodes in general.

P 14 Steven Engelen

Integrated Disease Management Approaches – Developing Durable Disease Resistance in Oilseed Rape and Soybean

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Bayer CropScience provides integrated solutions to the farmer, combining seeds and traits with chemicals and biologicals. The Trait Research department delivers gene based solutions to the breeders in order to obtain varieties with broad-spectrum and durable disease resistance. The poster presentation will illustrate by means of 2 projects the approaches that are taken to tackle main diseases in Oilseed Rape and Soybean. In Oilseed Rape identification and characterization of native R-genes against Blackleg and Clubroot are key to the development of durable stacking approaches. For Soybean, the Microbial Diversity Acquisition (MiDAS) platform is mined to identify antifungal proteins with new modes of action against Asian Soybean Rust. The tools and resources available at Bayer CropScience to identify R-genes and proteins with new modes of action will be presented.

P15 Sebastian Eves-van den Akker

Identification and Characterisation of a Hyper-Variable Apoplastic Effector Gene Family of the Potato Cyst Nematodes

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Sedentary endoparasitic nematodes are obligate biotrophs that modify host root tissues, using a suite of effector proteins to create and maintain a feeding site that is their sole source of nutrition. Using assumptions about the characteristics of genes involved in plant-nematode biotrophic interactions to inform the identification strategy, we provide a description and characterisation of a novel group of hyper-variable extracellular effectors termed HYP, from the potato cyst nematode *Globodera pallida*. HYP effectors comprise a large gene family, with a modular structure, and have unparalleled diversity between individuals of the same population: no two nematodes tested had the same genetic complement of HYP effectors. Individuals vary in the number, size, and type of effector subfamilies. HYP effectors are expressed throughout the biotrophic stages in large secretory cells associated with the amphids of parasitic stage nematodes as confirmed by *in situ* hybridisation. The encoded proteins are secreted into the host roots where they are detectable by immunochemistry in the apoplasm, between the anterior end of the nematode and the feeding site. We have identified HYP effectors in three genera of plant parasitic nematodes capable of infecting a broad range of mono- and dicotyledon crop species. *In planta* RNAi targeted to all members of the effector family causes a reduction in successful parasitism.

P16 Barbara Franco

Identification and characterisation of *Rhynchosporium commune* pathogenicity factors

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R. commune is the causal agent of scald, one of the most destructive diseases of barley. Grain quality can also be affected leading to significant yield losses. *R. commune* populations are highly diverse and can change rapidly overcoming barley resistance and fungicides control and thus leading to infection. Little is known about *R. commune* pathogenicity factors. At the moment is recognized the action of secreted effector proteins released to suppress host immune system. A small family of necrosis inducing peptides (NIPs) have been identified; their infiltration into barley and some dicotyledonous leaves (Rohe *et al*., 1995) caused necrosis resembling the disease symptoms (Wevelsiep *et al.,* 1991). The principal objective of the project is the understanding of molecules released by pathogen during the interaction. Analysis of transcripts produced by *R. commune* during infection of barley plants is a valuable resource for identification of pathogenicity factors. At the same time gene knockout can be used to elucidate gene function.

P17 Barbara Geric Stare

In planta activity and 3D model structure of effector expansin-like proteins (EXPB2) from plant parasitic nematode *Globodera rostochiensis*

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Infective larvae (J2) of potato cyst nematodes secrete effector proteins which facilitate successful infection of the host plants. Expansins are one group of the effectors, which help in degradation of plant cell wall by loosening non-covalent interactions between components of the plant cell wall. Molecular variability of *exp*B2 gene was shown in diverse populations of the *Globodera rostochiensis.* In this study we have shown the *in planta* expression and activity of GrEXPB2 protein. Systemic expression of GrEXPB2 type protein (CAC84564.1) induced chlorosis and dwarfing in *Nicotiana benthamiana* while variant EXPB2 proteins although expressed had no effect. Further GrEXPB2 type protein induced cell death in tomato and potato, but no symptoms in *N. tabacum.* Variant proteins induced no visible symptoms in tomato. Model of 3D structure for GrEXPB2 type protein was determined based on previously determined crystal structure of EXPB1 from maize (PDB: 2hczX). All three AA changed in the variant proteins are positioned on the outer surface of the protein model.

Small changes (1 or 2 AA change compared to type protein GrEXPB2) resulted in inactive EXPB2 protein variants. These changes probably affect the interaction with polymers of the cell wall and the proteins function as well as the 3D structure of the protein (one variant possibly affecting a disulphide bond).

P18 Fabian Giska

Phosphorylation of HopQ1, a Type III Effector from *Pseudomonas syringae,* Creates a binding for host 14-3-3 proteins

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HopQ1, a type III secretion effector secreted by *Pseudomonas syringae* pv. *phaseolicola,* promotes the development of halo blight in common bean. However, when this same effector is injected into tobacco cells, it is recognized by the immune system and prevents infection. Although the ability to synthesize HopQ1 determines host specificity, the role it plays inside plant cell remains unexplored. Following transient expression *in planta*, HopQ1 was shown to co-purify with host 14-3-3 proteins. The physical interaction between HopQ1 and 14-3-3a was confirmed *in planta* using FRET-FLIM techniques. Moreover, mass spectrometric analysis detected specific phosphorylation of the canonical 14-3-3 binding site (RSXpSXP, pS donates phosphoserine) located in the N-terminal region of HopQ1. Amino acid substitution within this motif abrogated the association and led to altered subcellular localization of HopQ1. In addition, the mutated HopQ1 protein showed reduced stability *in planta*. Furthermore it was shown that HopQ1 ability to interact with 14-3-3 increases *P. syringae* virulence in plants. These data suggest that the association between host 14-3-3 proteins and HopQ1 is important for modulating the properties of this bacterial effector.

P19 Cynthia Gleason

Characterization of novel root-knot nematode effectors

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Root-knot nematodes are small, soil borne pathogens that infect plant roots. Of the phytonematodes, root-knot nematodes are one of the most important due to their broad host range and their widespread distribution. The crop damage and reduced yield caused by these small pathogens contributes to billions of dollars in economic losses each year. During infection the nematode secretes a variety of molecules to both to de-differentiate plant cells into feedingsites as well as to suppress plant immunity. Using the known root-knot nematode secretome/genome(s), we identified several novel genes from the nematode that may be acting as effectors that can help quash plant defences. Utilizing *Pseudomonas syringae* to then deliver these potential effectors into the plant, we could prioritize the candidates which may be targeting plant immune responses. We are now in the process of further characterizing these candidates. For example, we have evidence that some of these effectors, when ectopically expressed in plants, can enhance nematode susceptibility. At least one of these candidates is localized to the glands of nematode, an indication that it can be secreted via the stylet during infection. Most interestingly, a yeast-two-hybrid analysis has shown that an effector candidate can interact with an Arabidopsis protein that is predicted to be involved in vesicular transport, highlighting the possibility that the nematode may be manipulating intracellular trafficking. Overall, by studying these effector candidates in more detail, we hope to better understand how the nematode can bypass plant defences and establish themselves in the roots.

P20 Rafal Hoser

Plant immunity suppression by natural variants of *Pseudomonas syringae* HopQ1 effector

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HopQ1 is supposed to be a type three effector recently acquired by *Pseudomonas syringae* and it is present only in a limited number of strains. The exact role of HopQ1 in promoting *P. syringae* virulence remains largely elusive. However, it has been recently shown that HopQ1 and XopQ, its homolog form *Xanthomonas euvesicatoria*, inhibit MAPKs activities and thereby probably prevent immune response in infected plant cells (Hann et al., 2013;Teper et al., 2013). Interestingly, our data suggest, that strain-specific variants of HopQ1, that is PstHopQ1-1 from *P. syringae* pv. *tomato* and PspHopQ1 from *P. syringae* pv. *phaseolicola*, significantly differ in their abilities to suppress MAPKs activities in Arabidopsis protoplasts. Consistently, *NHL10* promoter induction, which is a marker of MAPK activity in Arabidopsis cells, is suppressed only by PspHopQ1 and not by PstHopQ1-1. Furthermore, we found, that PstHopQ1-1, in contrast to PspHopQ1 seems to be specifically cleaved at its N-terminus by an unknown plant protease. Surprisingly, PstHopQ1-1 and PspHopQ1 differ in only 6 amino acid positions, that are localized at their N-termini. To determine which of these amino acids affect HopQ1 stability and its ability to inhibit kinase activity in Arabidopsis cells, we tested mutated variants of PstHopQ1-1 and PspHopQ1, that possess natural amino acids combinations present in all known HopQ1 effectors in various *P. syringae* strains. In Arabidopsis protoplasts expressing PstHopQ1-1 S87L-L91R double mutant, in contrast to its wild-type form, we observed suppression of *NHL10* promoter activity. Moreover, PstHopQ1-1 S87L-L91R, was observed only as the full-length protein suggesting it was insensitive to proteolytic cleavage. So, probably, these two amino acid positions (or one of them) are critical for both MAPK activity inhibition and proteolysis of HopQ1 in Arabidopsis cells. It is proposed that the sequence differences between HopQ1 homologs, which influence their stability and function, are a consequence of an adaptation of the recently acquired effector to specific hosts of bacterial strains. We are currently testing this hypothesis.

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P 21 Ece Bortecine Kasapoglu

Comparative transcriptome analysis of three cyst nematode species

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Cyst nematodes cause a high economical losses worldwide on a wide range of crops. Some cyst nematode species have their host range limited to monocotyledons such as *Heterodera avenae* as one of the cereal cyst nematodes (CCN). Other cyst nematode species such as *H. schachtii* (beet cyst nematodes) and *Globodera pallida* (potato cyst nematodes) parasitize exclusively on dicotyledons. The success of cyst nematode parasitism depends on effective manipulation of the host plants. We expect that different cyst nematodes species have a common set of effectors facilitating the establishment and maintenance of syncytia. Our study aims to identify common and species specific-putative secretory proteins (PSP) among the cyst nematode species *H. schachtii*, *H. avenae*, and *G. pallida*, by comparing their transcriptomes and secretomes. All datasets were compared for sequences and common functional domains. We identified 15 overlapping PSPs while 9 of them were common PSPs. Interestingly, one of the highly conserved common PSP is annotated as bactericidal permeability-increasing protein (Hs_BPI). BPI proteins are known to have potent killing activity against gram-negative bacteria. Quantitative PCR and *in-situ* hybridization of Hs_BPI has shown that it is upregulated within post-infective stages and localized within the esophageal gland, respectively. We conclude that BPI protein may have dual functions in protecting the nematodes and its feeding site from microbial infection. We expect that our results can help identifying additional putative effectors that function as a part of common parasitism processes across cyst nematode species. These may give a basis for developing novel successful management strategies against several cyst nematode species.

Key Words: Cyst Nematodes, effectors, nematode parasitism

P22 Ralf Koebnik

Xanthomonas translucens – role of type III effectors in pathogenicity?

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Bacterial leaf streak (BLS), a bacterial disease of small grain cereals and grasses caused by *Xanthomonas translucens* (*Xt*), can cause significant yield losses under unfavorable conditions. BLS of barley and wheat is a seed-borne disease and thus a constraint for international germplasm exchange. Several countries list *Xt* as a quarantine organism. To cause disease, most xanthomonads depend on a highly conserved type III secretion system (T3SS) which translocates type III effectors (T3Es) into target host cells, among them members of the family of Transcription Activator-Like (TAL) effectors which modulate plant gene expression upon specific binding to target boxes in the promoter regions of plant genes.

Virtually nothing is known about pathogenicity mechanisms of *Xt*. Here, we report on three *Xt* draft genome sequences which allowed us to predict new candidate T3Es. Using *avrBs1* reporter assays, candidate T3Es were confirmed. Site-specific mutagenesis allowed us to establish an essential role of the T3SS for pathogenicity on barley. Systematic knock-out mutageneses of *tal* genes were performed and mutants were characterized by pathogenicity assays, suggesting an important contribution of TAL effectors to pathogenicity. Several *tal* genes were cloned and sequenced in order to identify new susceptibility genes in barley. Progress on the identification of effector targets will be presented.

P23 Philippe Lecomte

Using SILAC strategy to identify protein effectors in the wheat-Fusarium graminearum pathosystem

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Fusarium head blight (FHB) is a widespread and destructive disease of wheat. Causal agents of FHB mainly target spikes and substantially alter grain yield and quality through the production of harmful mycotoxin. In spite of a number of agronomic strategies, our ability to control FHB spreading is still limited and requires increasing knowledge at the molecular scale. Deciphering the molecular crosstalk that controls plant – *Fusarium spp* interaction is a gate to develop new strategies to sustain plant resistance. To ensure their own development, pathogens use a wide range of effectors able to interfere with the host immune system and the host metabolism. Although protein effectors can be detected in the secretome of the pathogen, their identity and their roles remain poorly documented. To define the repertoire of fungal effectors dynamically produced by *F. graminearum* during infection, we customized the SILAC (Stable Isotope Labeling by Amino acids in Cell culture) approach. We developed specific culture media for the production of labeled mycelium and obtained a Lys 15N13C incorporation rate in the mycelium of up to 90%. The analysis of proteins extracted from infected ears with spores produced by this mycelium will allow labeled fungus proteins to be distinguished from the host proteins and the identification of fungal proteins which were produced during infection from non labeled host substrates. We will present the developments of this approach and the first results obtained with 'omics' tools to decipher the molecular dialogue that occurs between wheat and *Fusarium graminearum*.

Keywords: FHB disease, *Fusarium graminearum*, fungal effectors, plant susceptibility, SILAC

P24 Jana Libantova

Antifungal potential of crude protein extract from carnivorous plant *Drosera rotundifolia*

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Sundew (*Drosera rotundifolia L.*) is a carnivorous plant that represents a source of wide range of compounds with fungitoxic activity applicable in biotechnology programs. So far only plumbagin, secondary metabolite, has been shown to inhibit fungal growth in conditions *in vitro*. Here we focused on the evaluation of crude protein extracts isolated from *D. rotundifolia* in terms of their ability to inhibit the growth of selected fungal pathogens. Overall there was a considerable variation in the degree of sensitivity exhibited by fungi tested. The most sensitive fungus *Fusarium oxysporum* f. sp. *lini* and the least sensitive *Fusarium sambucinum* exhibited retardation of their growth at concentration of sundew crude protein extracts 30 μg and 80 μg, respectively. When fungicidal effect of sundew leaf protein extract was compared with extracts isolated from *Solanum tuberosum* and *Linum usitatissimum* the same amount of leaf extract from sundew exerted higher inhibitory effect on the growth of *Fusarium oxysporum* f. sp. *lini* than both extracts from potato and flax. In addition, sundew leaf protein extract was able to suppress the germination of spores of tested fungi as well.

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P25 Xiao Lin

Isolation and characterization of apoplastic immune receptors in potato; towards a novel type of durable resistance against late blight

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The potato (*Solanum tuberosum* L.) is one of the three most consumed crops world-wide. The most devastating disease of potato is late blight, which is caused by the oomycete *Phytophthora infestans* that is renowned for triggering the Irish potato famine in the 1840s. The most sustainable strategy to manage late blight is to breed broad-spectrum disease resistance into potato. However, traditional disease resistance breeding that exploits cytoplasmic resistance genes (*R* genes) has been of limited success, as *P. infestans* has a remarkable capacity to rapidly adapt to resistant plants. Another, yet unexploited layer of immunity occurs at the surface of plant cells. This apoplastic immunity has generally a broader spectrum and is based on recognition of conserved proteins of pathogens. To obtain novel potato pattern recognition receptors (PRRs) that can recognise oomycete apoplastic effectors, we are deploying an effectoromics approach. A variety of predicted oomycete apoplastic effectors are subjected to functional screens on almost 100 wild potato species. One of the selected effectors is Scr74, a 74 amino-acid *s*ecreted *c*ysteine *r*ich protein that is highly polymorphic in *P. infestans*. Various *Solanum* genotypes were identified that showed specific responses to Scr74. A map-based cloning approach was initiated to identify the gene that mediates response to Scr74. F1 populations were generated, and phenotyping and genotyping are in progress. The ultimate goal of these studies is to understand and achieve a more effective resistance against oomycete pathogens by combining cytoplasmic and apoplastic immunity.

P26 Carsten Pedersen

Functional analyses of barley powdery mildew effector candidates

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Previous studies have identified around 500 *Bgh* effector candidates (CSEPs) from the barley powdery mildew fungus (*Blumeria graminis* f. sp. *hordei*) and they are believed to manipulate host defence response and facilitate susceptibility (Spanu et al. 2010, Pedersen et al. 2012), but functional studies are hampered by the fact that *Bgh* is an obligate biotroph and recalcitrant to genetic transformation. To study the function of effector candidates we therefor employ particle bombardment as a transient technique for silencing effector candidate genes in the pathogen by host-induced gene silencing (HIGS).

We use the Yeast Two-hybrid system for screening for putative effector targets in the host and confirm interactions *in planta* by BiFC in *N. benthamiana*. To further examine the interaction we use transient gene silencing (TIGS) against putative effector targets in the host to see if silencing these targets increases susceptibility. Likewise, we employ over-expression of CSEPs or host-targets to test whether this increases or reduces susceptibility. We are now trying to implement virus-based techniques for both silencing and over-expression to overcome limitations with the single-cell based particle bombardment technique. Here we will give an overview and update on the functional studies of CSEPs in our lab.

P27 Krzysztof Pawłowski

Bioinformatics prediction of a novel family of pseudokinase effector proteins present in several plant and animal pathogens

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Novel protein kinase-like families continue to be discovered and characterised, e.g. the vertebrate secreted kinases (FAM20) or the *Plasmodium* calcium-dependent kinases involved in infection. Our lab has recently discovered in silico a number of putative kinases (e.g. FAM69 kinases in *Metazoa*, and the SELO kinases in eukaryotes and bacteria). Here, using bioinformatics tools for remote homology detection, we present a robust prediction of protein kinase-like (PKL) structure for a group of uncharacterised bacterial proteins present in several plant pathogens (e.g. some strains of *Pseudomonas syringae*), in a few animal pathogens (e.g. some strains of *Burkholderia multivorans*) and in some free-living organisms.

Strikingly, all the proteins in the novel family lack the catalytic aspartate residue ("Asp166") characteristic of PKL kinases. Thus, they most likely are pseudokinases, i.e. proteins of kinase-like structure, but devoid of catalytic activity, and possibly interfering in phosphorylation signalling pathways by non-productive substrate binding. Proteins from the novel family are poorly studied, but several of them are annotated as type III effectors, e.g. HopBF1 protein from *Pseudomonas syringae* pv. *avellanae.*

Three-dimensional structure modelling presents a typical kinase fold and allows exploration of the putative binding sites. Homologue survey and phylogenetic analysis of the novel family of genes is used to delineate their complex evolutionary history leading to the present sparse presence in several bacterial taxa. Analysis of genomic neighbourhoods of the novel pseudokinase genes provides insights into their participation in regulatory and functional networks. This is supported by exploration of distant homology relationships to other PKL proteins.

The novel protein kinase-like family expands the known kinome and deserves experimental determination of the detailed molecular mechanisms and biological roles within the signaling networks. The presence of different family members in plant and animal pathogens raises interesting questions regarding similarities in infection mechanisms involving effector pseudokinases in different hosts.

P28 Marcin Piechocki

Functional analysis of homologs of bacterial effector HopQ1

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HopQ1, a type III effector secreted by *Pseudomonas syringae* pv. *phaseolicola*, is widely conserved among diverse genera of plant bacteria. It promotes the development of halo blight in common bean. However, when injected into tobacco cells, it is recognized by the immune system and prevents infection. *In silico* analysis of its amino acid sequence revealed that HopQ1 is a putative nucleoside hydrolase. We have shown that HopQ1 binds to plant 14-3-3 proteins in a phosphorylation-dependent manner (Giska et.al., 2013). To address the question of the mechanism of HopQ1 action we searched for evolutionary related proteins. We found that HopQ1 homologs also occur in lower plants such as mosses and algae. The protein PHYPADRAFT_168432 from *P. patens* draws 67% homology to HopQ1 and contains also a canonical mode 1 14-3-3 binding site. In order to characterize its properties we cloned sequence encoding PHYPADRAFT_168432 to express it in plant and bacterial cells. We failed to detect nucleoside hydrolase activity for the recombinant protein produced in *E. coli*. We observed however, that in contrast to HopQ1, PHYPADRAFT_168432 is not recognized by tobacco immune system, because when expressed in tobacco leaves no macroscopic signs of tissue necrotization developed. Our ongoing studies focus on assessing putative virulence properties of PHYPADRAFT_168432. Since as a plant protein, it does not have a signal sequence recognized by type III secretion system of *P. syringae*, we added an appropriate sequence from HopQ1. The fusion protein did not positively affect *P. syringae* growth in bean plants.

Giska F, Lichocka M, Piechocki M, Dadlez M, Schmelzer E, Hennig J, Krzymowska M (2013)

Phosphorylation of HopQ1, a Type III Effector from *Pseudomonas syringae*, Creates a Binding Site for Host 14-3-3 Proteins. Plant Physiol 161: 2049-2061

P29 Crina Popa

AWR5 bacterial effector affects TOR signalling pathway in *Saccharomyces cerevisiae*

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AWRs are a multigenic family of 5 type III effectors from the plant pathogen *Ralstonia solanacearum*, involved in bacterial infection as showed from previous experiments *in planta* (Solé et. al, 2012). Our previous work showed that AWR5 protein causes growth inhibition phenotypes on budding yeast *S. cerevisiae*. Expression of T3SS effectors in *Saccharomyces cerevisiae* oversteps the limitations of their study in plants, as yeast lacks resistance (R) proteins that can trigger ETI responses. In order to characterize AWR5 function, we performed a gene expression profiling using heterologous expression of AWR5 in the yeast *Saccharomyces cerevisiae*. Expression of AWR5 in yeast has showed a gene expression profile reminiscent of TOR complex inhibition: down-regulation of genes involved in ribosome biogenesis or rRNA processing and up-regulation of genes involved in the metabolism of nitrogen. Data will be presented on the molecular mechanism(s) underpinning these AWR5-dependent changes in gene expression in *S. cerevisiae*. We are currently investigating the AWR5 effect on TOR pathway on non-host *Nicotiana benthamiana,* where it causes hypersensitive responselike cell death.

This research project will unravel the mechanism of action of a type III effector which has a dramatic impact on plant physiology.

P30 Elżbieta Różańska

Arabidopsis Sec21 protein is involved in development of syncytium induced by *Heterodera schachtii.*

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Plant parasitic cyst-forming nematodes induce a specific feeding structure called a syncytium in host roots. Syncytial elements differ transcriptionally, metabolically and ultrastructurally from typical differentiated plant cells. One of the characteristic features of syncytium ultrastructure is vacuolar system consisting of numerous small vacuoles and vesicles. The biogenesis of these vesicles and their trafficking pathways remain unknown. Sec21 protein is a component of the COPI vesicle coatomer and it is involved in the anterograde and retrograde cargo transport between endoplasmic reticulum and Golgi apparatus. We analysed spatial and temporal expression patterns of genes encoding Sec21 proteins using semi-quantitative (sq) RT-PCR and immunolocalised their products in Arabidopsis roots containing syncytia induced by *H. schachtii*. Immunolocalisation confirmed the presence of Sec21 protein predominantly in syncytia. sq RT-PCR procedures revealed that genes encoding Sec21 proteins are up-regulated in 3-days-old syncytia. The presence of Sec21 proteins indicates that small vesicles abundantly present in syncytia are involved in COPI-related transport events.

P31 Diego Rubiales

Identifying allelic variants for papilla based powdery mildew adult plant resistance against powdery mildew in oat by association studies

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Cereal crops, including oats, generally show less powdery mildew infection on older plants than on seedlings. This field or 'adult plant' resistance (APR) is expressed by lateformed leaves of oats, barley and wheat although the magnitude differs among genotypes. This kind of resistance is highly interesting from the breeding point of view since it has insufficiently been deployed by breeding and can contribute to durability. Based on a collection of commercial varieties and landraces of oats we identified genotypes with adult plant resistance and further characterized the components of this resistance. Detailed study revealed a large increase of penetration and post-haustorial resistance in the older compared to the younger leaves. The collection was genotyped with 31 simple sequence repeat (SSR) and 15,000 Diversity Arrays Technology (DArT) markers to reveal association with adult plant resistance. 1872 polymorphic markers combining DArT and SSR markers were considered for association analysis. Following population structure and linkage disequilibrium analysis five different general and mixed linear models accounting for population structure and/or kinship corrections and two different statistical tests were carried out. One DArT sequence, oPt-5014, was strongly associated with powdery mildew rust resistance in adult plants. Characterization of the resistance sources and the associated markers will be useful for future breeding programs aiming to improve durable resistance.

P32 Anja Karine Ruud

Necrotrophic effectors and sensitivity genes – gene-for-gene interactions in the wheat- *Stagonospora nodorum* pathosystem

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Leaf blotch diseases in wheat can cause significant yield losses and reduce grain quality. In Norway, *Stagonospora nodorum* is the dominant causal agent. The mechanisms of the pathosystem of this necrotroph have been thought to be mostly quantitative and nonspecific. More recent research suggests that very specific, inverse gene-for-gene actions are involved. Many host-selective necrotrophic effectors (NEs) and corresponding sensitivity (*Snn*) genes in the host have already been identified. The effectors induce cell death in the host, which enables the pathogen to invade the dead tissues. The core of this research has been to "Mendelize" the host-pathogen system by deconstructing the components of a single interaction between the host and the pathogen. Single effector molecules are purified from culture and infiltrated into wheat lines from a segregating mapping population. The chromosomal location of the corresponding sensitivity gene in the host can then be found using linkage mapping and DNA markers. The focus of my PhD is to identify and map NE/*Snn*-interactions in the Norwegian pathogen population and wheat material, and to determine the importance of these interactions under field conditions. Preliminary results from field and controlled environments in Ås, Norway and Fargo, USA, indicate that several novel QTL are found in our material, and that some are detected under both field and seedling experiments. Also, sensitivity and resistance to confirmed NEs, i.e. Tox3, is confirmed. Further work will include fine mapping of the chromosomal regions of interest and development of diagnostic markers for practical breeding.

P33 Remco Stam

Quantitative transcriptomics of the *P. capsici* – host interaction

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Plant-microbe interactions feature dynamic interplay between pathogen encoded virulence factors and host (defence) signalling components. Despite major advances in host-microbe interaction research, little is known about the processes that result in pathogen virulence or host susceptibility. *Phytophthora capsici* is a hemibiotrophic plant pathogen that infects important crop plants such as pepper, tomato and cucumber. The *P. capsici* infection cycle features a dynamic developmental programme, driven by stage specific gene expression in both pathogen and the host. A detailed understanding of the transcriptional changes associated with susceptible interactions thus has the potential to unveil processes associated with virulence and immunity. Previously, we have shown that during infection, transcriptional shifts occur and appear coordinated in tomato and *P. capsici.* These shifts coincide with the early infection phase (after germination) and the switch from biotrophy to necrotrophy and suggest modulation of host immune signalling by both organisms. Here we expand on the transcriptional profiling of *P. capsici*-tomato interactions by means of RNA sequencing of plant and pathogen using Illumina technology. For this purpose we performed a detailed timecourse experiment (0, 4, 8 16 and 24 hours after inoculation) on both infected and mock inoculated tomato leaves. Here we will report on our latest progress on these experiments and the new information arising from downstream analyses.

P34 Doron Teper

Identification of new type III effectors of *Xanthomonas campestris* pv. *Vesicatoria* by using a machine learning approach

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The plant pathogen *Xanthomonas campestris* pv. *vesicatoria* utilizes a type III secretion system to translocate a pool of about 30 effector proteins into host cells. A genome-scale computational machine learning approach was used to indentify new *Xcv* effector proteins. To distinguish between effector and non-effector open reading frames (ORFs) in the genome of the *Xcv* strain 85-10, we computationally defined a set of features covering a wide range of characteristics that include expression profiles, GC content, genome localization, similarity to proteomes of type III phytopathogens, regulatory sequences, and amino acid bias. Machine learning algorithms utilizing these features were then applied to classify all the ORFs within the *Xcv* 85-10 genome and predict candidate effectors. A group of 36 predicted effectors were tested for translocation into pepper cells and seven of them were validated as translocated effectors. Interestingly, four of the newly identified effectors belong to previously undefined effector families. Inactivation of the effector *xopAP* resulted in mutant bacteria that caused reduced chlorophyll loss and cell death when compared to wild-type bacteria. No difference was observed in bacterial growth of the *Xcv xopAP* mutant, thus indicating that the reduction in virulence was not determined by a reduced bacterial titer. Analysis of wound and defense response marker genes by qRT-PCR in infected pepper plants revealed that the expression level of the jasmonic acid marker genes *opr3* and *loxD* was altered in the *Xcv xopAP* mutant, raising the possibility that XopAP is involved in modulation of host hormone signaling.

P35 Gaetan Thilliez

Non host resistance in *Solanaceae.* The role of Phytophthora secreted effector in determining the host range

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Non-host resistance is a form of durable defence which provides protection to all isolates of a given pathogen species. Host resistance is thought to be mediated by two layers of defence induced by recognition of Pathogen Associated Molecular Patterns (Pattern triggered Immunity, PTI) or recognition of pathogen effector activity (Effector Triggered Immunity, ETI). It has been hypothesised that the phylogenetic distance between host and non-hosts dictates the relative contributions of PTI and ETI to non-host resistance. The oomycete pathogens *Phytophthora infestans* and *Phytophthora capsici* share the hosts *Nicotiana benthamiana* and *Solanum lycopersicum*, but are unable to infect pepper (*P. infestans*) and potato (*P. capsici*). With the availability of genome sequences for both pathogens, we can study the contribution of effector activity and recognition to non-host resistance in these related plant species. A Markov Cluster (MCL) analysis of *P. infestans* and *P. capsici* predicted protein sets reveal that those pathogens seem to share most of their predicted protein set. However the analysis revealed that both *P. infestans* and *P. capsici* share only a fraction of their effector repertoires and carry species specific RXLR effector families. Further efforts are now deployed to: A) Test if effectors conserved at the sequence level retain the similar functions in host and non- host plants; B) Understand the role of *P. infestans* or *P. capsici* specific effectors toward virulence and host range.

P36 Fabienne Vailleau

The *Ralstonia Solanacearum Secretome*: type III effectors, type III associated proteins and their role in pathogenicity

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Ralstonia solanacearum, the causal agent of bacterial wilt, exerts its pathogenicity through more than a hundred secreted proteins, many of them depending directly on the functionality of a type III secretion system. To date, only a few type III effectors have been identified as being required for *R. solanacearum* pathogenicity, which probably results from the existence of functional redundancy among a large effector repertoire. In order to identify groups of effectors collectively promoting disease on susceptible hosts, we investigated the role of post-transcriptional regulations in the control of type III secretion in *R. solanacearum* strain GMI1000. We analyzed the secretome of strain GMI1000 as well as mutants for several type III chaperones or type III secretion-associated proteins using mass spectrometry experiments. This analysis revealed specific subsets of effectors differentially secreted in some of the mutant strains. The pathogenicity of these *R. solanacearum* mutants was evaluated on several plants, and interestingly, different host specificities could be identified. Advantages of such a global approach to highlight sets of T3Es potentially required for *R. solanacearum* pathogenicity on hosts belonging to diverse botanical families (solanaceous, legumes…) will be discussed.

P37 Keke Wang

Functionnal analysis of Type III effector RipG7 of *Ralstonia solanacearum*

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The soil-borne pathogen *Ralstonia solanacearum* naturally infects plant roots and causes bacterial wilt disease in over 200 plant species, especially *solanaceous* plants. A main pathogenicity determinant of *Ralstonia solanacearum* is type III secretion system (T3SS). *R. solanacearum* use T3SS to inject a large repertoire of effectors into plant cells. Among these effectors, RipG (formerly named "GALA") is a family of seven effectors in GMI1000 that have homologies with plant F-box proteins. RipG7 is an essential effector among the GALA genes for the virulence on *Medicago truncatula* and also involved in the pathogenicity on tomato and *Arabidopsis*. F-box proteins are known to form E3-ubiquitin ligases *in planta,* we showed that RipG7 is localized both in the cytoplasm and the nucleus of infected cells and that this latter localization is sufficient for its function. To gain insight into the genetic and functional variation of RipG7, we showed that RipG7 genes from four phylotypes spanning the whole diversity of *R. solanacearum*, have different contributions to pathogenicity on *Medicago truncatula.* Furthermore, in our effort to understand the relationship between structure and function of this type III effector, we are currently investigating whether the strong positive selected sites identified in RipG7 can be correlated to the virulence function of specific allelic versions.

P38 Ronja Wonneberger

Can susceptibility to net blotch in barley be explained by sensitivity to necrotrophic effectors?

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Net blotch is a major barley disease in Norway caused by the necrotrophic pathogen *Pyrenophora teres* leading to yield losses up to 40%. Many necrotrophic pathogens such as the wheat pathogen *Stagonospora nodorum* secrete necrotrophic effectors (NEs) which act as virulence factors in order to gain entry into the host. In the host NEs can cause cell death in the presence of a corresponding dominant plant susceptibility factor. So far, NEs have not been discovered in the barley net blotch pathosystem.

The main objective is to explore the potential role of NEs and to determine loci contributing to susceptibility to net blotch in barley. It will be examined whether *P. teres* produces NEs, and whether the presence of corresponding host receptors in barley leads to of susceptibility to this disease under field conditions.

In order to characterize the Norwegian net blotch pathogen population, a representative isolate collection will be established by collecting naturally infected barley samples from different regions. Isolates as well as their culture filtrates will be screened for specific reactions against differential barley lines and segregating mapping populations with available marker data to investigate the role of NEs, to characterize novel NE-host susceptibility interactions and to map the corresponding sensitivity loci. Preliminary inoculations with a small number of isolates revealed a QTL on barley chromosome 3H in the Hector x NBD112 mapping population and two QTLs on 3H and 6H in the CI5791 x Tifang mapping population. Effector protein candidates will be purified and subjected to further analysis to verify their effect on disease development.

P39 Elitsur Yaniv

Fine-mapping of the *Rpt5* net blotch resistance gene region in barley

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The net blotch disease, caused by the *Pyrenophora teres* f. *teres*, is one of the most important fungal diseases of barley (*Hordeum vulgare*) in Finland. During testing of a collection of barley accessions, including landraces, for resistance to net blotch, the Ethiopian landrace CI9819 turned out to be 98% resistant, an optimal level for providing sustainable resistance. The resistance gene in CI9819 was designated *Rpt5.* We are using a variety of mapping strategies, including exploitation colinearity, to fine-map *Rpt5*. So far we have narrowed the segment to 0.03cM. We have also carried out association genetics on a wide set of barley cultivars and made expression analyses of resistant and susceptible mapping parents. These approaches, combined with the genetically and physically mapped barley gene space ("gene-ome") and emerging barley genome sequence will greatly enhance efforts to positionally clone *Rpt5.*

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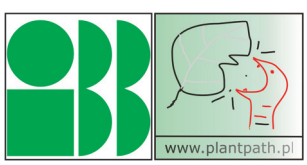
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