Journal of Shandong Agricultural University
( Natural Science Edition )

# OMGN 2018

## OOMYCETE MOLECULAR GENETICS NETWORK

## April 8 - 12

## Taian, Shandong, China.

The picture was gotten from Wikipedia, the free encyclopedia.

山東農業大學
SHANDONG AGRICULTURAL UNIVERSITY

# Oomycete Molecular Genetics Network Meeting 2018

The Oomycete Molecular Genetics Research Network (OMGN) was initially funded by an NSF Research Coordination Network grant in 2001 and continued to receive funding from the NSF for many years. More recently, the Network has received funding from various USDA AFRI programs. The purpose of our annual meeting is to promote communication and collaboration, and minimize the duplication of effort within the oomycete molecular genetics community. Our community now numbers well in excess of 100 laboratories from around the world, and research on oomycetes attracts considerable attention from outside the community as well as within. The OMGN annual meeting alternates between Asilomar, CA, and another venue, usually outside of the USA. This year, the meeting returns to China, and we are delighted to welcome you to the Ramada Plaza Taian. The 2018 meeting will cover some of the latest research on Oomycete Genomics, Evolution, Population Biology, Host Interactions, and Effector Biology. We look forward to an engaging and dynamic meeting!

# ORGANIZERS

## Scientific Program:

Xiuguo Zhang (Department of Plant Pathology, Shandong Agricultural University, Tai'an, Shandong, China)

Chunyuan Zhu (College of Life Sciences, Shandong Agricultural University, Tai'an, Shandong, China)

## Meeting Logistics:

Joel Shuman (Virginia Tech, Blacksburg, VA, USA)

Shi Wang (Shandong Agricultural University)

Cancan Yang (Shandong Agricultural University)

# INTERNATIONAL SCIENTIFIC ADVISORY COMMITTEE

## Co-chairs:

Francine Govers (Wageningen University, Wageningen, Netherlands)

John McDowell (Virginia Tech, Blacksburg, VA, USA)

## Members:

Brett M. Tyler (Oregon State University, Corvallis, OR, USA)

Sophien Kamoun (The Sainsbury Laboratory, Norwich, UK)

Laura Grenville-Briggs Didymus (Swedish University of Agricultural Sciences, Alnarp, Sweden)

Erica M. Goss (University of Florida, Gainesville, FL, USA)

# ACKNOWLEDGEMENTS

This meeting is supported by Shandong Agricultural University. The project “Earmarked fund” was supported by China Agriculture Research System(CARS-25-03B) with Dr. Du as program leader.

# TABLE OF CONTENTS

# AT-A-GLANCE PROGRAM

*Oral presentations are in Grand Ballroom. Reception/Poster sessions are outside the Grand Ballroom. All meals are in Ramada Plaza Taian.*

| **Monday April 9th** | | |
|---|---|---|
| 8:30 - 9:00 | Registration | |
| 9:00 - 9:05 | Welcome | |
| **9:05 - 10:25** | **Oomycete biology, populations, and evolution I** | **Chair: Audrey Ah-Fong and Kai Tao** |
| 9:05 - 9:20 | Cellular signaling and cytoskeleton dynamics in *Phytophthora* | Francine Govers |
| 9:25 - 9:40 | Biochemical and genetic analyses of the oomycete *Pythium insidiosum* provide new insights into clinical identification, pathogenicity, and evolution of metabolism-related traits | Theerapong Krajaejun |
| 9:45 - 10:00 | Mating type change in *Phytophthora infestans* regulated by histone deacetylases | Li-Yun Guo |
| 10:05 - 10:20 | Ecology and evolution of oomycete communities in response to soybean seed treatments | Zachary Noel |
| 10:25 - 11:00 | Group photo and break | |
| **11:00-12:20** | **Keynote Speech** | **Chair: Francine Govers** |
| 11:00 - 11:35 | Exploring fungal DNA Virus to control crop diseases, challenge and opportunity | Daohong Jiang |
| 11:40 - 12:15 | Omics in Plant Breeding | Sanwen Huang |
| 12:20 - 2:00 | Lunch | |
| **2:00 - 3:40** | **Effectors I** | **Chair: Petra Boevink and Kelsey Wood** |
| 2:00 - 2:15 | Dissecting the transcriptional plasticity of *P. sojae* | Brett Tyler |
| 2:20 - 2:35 | Identification of *Plasmopara viticola* candidate RXLR effector repertoire in *Nicotiana benthamiana* | Jiang Lu |
| 2:40 - 2:55 | How are effectors secreted and delivered to the host? | Shumei Wang |
| 3:00 - 3:15 | Plant recognition of *Phytophthora* GH12 MAMPs | Yan Wang |
| 3:20 - 3:35 | Characteristics of RxLR effector structures in oomycete pathogens | Li Zhao |

| | | |
|---|---|---|
| 3:40 - 4:10 | Break | |
| **4:10 - 6:10** | **Genomics I** | **Chair: Paul Birch and Diya Sen** |
| 4:10 - 4:25 | Towards the identification of effectors and secondary metabolites involved in oomycete-oomycete interactions and mycoparasitism using comparative genomics | Laura Grenville-Briggs |
| 4:30 - 4:45 | Comparative genomics of four species of *Peronosclerospora* | Kyle Fletcher |
| 4:50 - 5:05 | What role does *Phytophthora kernoviae* play in New Zealand forests? | Rebecca McDougal |
| 5:10 - 5:25 | Land-use affects the growth and gene expression of *Phytophthora agathidicida* | Preeti Panda |
| 5:30 - 5:45 | Transcriptional regulation of *Phytophthora sojae* effectors for successful infection | Wenwu Ye |
| 5:50 - 6:05 | Genome sequence of *Plasmopara viticola* reveals effector repertoire and pathogenicity mechanisms | Ling Yin |
| 6:10 - 7:30 | Dinner | |
| 7:30 - 10:00 | Reception/Poster session | |

| **Tuesday April 10th** | |
|---|---|
| **9:00 – 5:00** | **Excursion/free time** |
| 6:00 - 7:30 | Conference Dinner |

| **Wednesday April 11th** | | |
|---|---|---|
| **8:30 - 9:50** | **Host Interactions and Resistance Mechanisms I** | **Chair: Jun Zhao and Veronica Ancona** |
| 8:30 - 8:45 | *Phytophthora palmivora* establishes tissue-specific intracellular infection structures in the earliest divergent land plant lineage | Sebastian Schornack |
| 8:50 - 9:05 | A pathogen suppressor of an NLR immune network | Lida Derevnina |
| 9:10 - 9:25 | A plant pathogen effector utilizes host susceptibility factor NRL1 to degrade the immune regulator SWAP70 | Qin He |
| 9:30 - 9:45 | A potato STRUBBELIG-RECEPTOR FAMILY gene StLRPK1 involves in plant immunity by association | Haixia Wang |

| | | |
|---|---|---|
| | with StSERK3/BAK1 | |
| 9:50 – 10:20 | Break | |
| **10:20-12:00** | **Effectors II** | **Chair: Suomeng Dong and Lida Derevnina** |
| 10:20 - 10:35 | *Phytophthora* infestans RXLR Effector Pi22926 suppresses plant immunity by targeting the host MAP3β to manipulate MAP3K signalling pathway | Zhendong Tian |
| 10:40 - 10:55 | Biochemical basis of an RXLR-WY effector suppression of an NLR network | Jessica Upson |
| 11:00 - 11:15 | Characterization of host-recognized WY domain effectors from *Bremia lactucae* that lack the canonical RxLR motif | Kelsey Wood |
| 11:20 - 11:35 | Functional analysis of an effector PcRxLR101 in *Phytophthora capsici* | Jing Li |
| 11:40 - 11:55 | A potato chloroplast kinase is required for Rpi-vnt1 mediated immunity against late blight pathogen | Chuyun Gao |
| 12:00 - 2:00 | Lunch | |
| **2:00-3:20** | **Oomycete biology, populations, and evolution II** | **Chair: Daolong Dou and Theerapong Krajaejun** |
| 2:00 - 2:15 | Molecular genetics of the oomycete pathogen of lettuce, *Bremia lactucae*. | Lin Zhang |
| 2:20 - 2:35 | Structure determination of three enzymes from *Phytophthora capsici* | Weiwei Song |
| 2:40 - 2:55 | Profiling of the *Phytophthora e*pigenomes | Han Chen |
| 3:00 - 3:15 | Immunity triggered by the small cysteine rich effector PC2 from *Phytophthora infestans* requires proteolytic cleavage by the host protease P69B | Shuaishuai Wang |
| 3:20 - 3:50 | Break | |

| **3:50-5:30** | **Genomics II** | **Chair: Sucheta Tripathy and Wenwu Ye** |
|---|---|---|
| 3:50 - 4:05 | New insights into pathogenicity of the emerging tropical pathogen: *Phytophthora colocasiae* on taro | Diya Sen |
| 4:10 - 4:25 | Mutations in oxysterol binding protein-related protein conferring oxathiapiprolin resistance confirmed using CRISPR/Cas9 in *Phytophthora capsici* and *P. sojae* | Jianqiang Miao |
| 4:30 - 4:45 | Detection of multiple oomycetes in metagenomic data by using E-probe Detection of Nucleic Analysis (EDNA) | Maria Fernanda Proano |
| 4:50 - 5:05 | EumicrobeDBLiteV11: a lightweight genomic resource and analytic platform for draft oomycete genomes | Arijit Panda |
| 5:10 - 5:25 | Diploid genome assembly works better when Pacbio long reads are supplemented with Illumina reads: *P. ramorum* assembly a case study | Mathu Malar C |
| 5:30 - 6:20 | Committee meeting | |
| 6:30 - 7:30 | Dinner | |
| 7:30 - 10:00 | Reception/Poster session | |

| **Thursday April 12th** | | |
|---|---|---|
| **8:30 - 9:50** | **Host Interactions and Resistance Mechanisms II** | **Chair: Rebecca McDougal and Qin He** |
| 8:30 - 8:45 | Synergistic anti-oomycete effect of melatonin with a biofungicide against oomycetic blank shank disease | Maozhi Ren |
| 8:50 - 9:05 | *Phytophthora* utilizes an RxLR effector to hijack the host ER stress as part of their infection strategy | Maofeng Jing |
| 9:10 - 9:25 | A conserved host target of oomycete Avr3a family effectors positively regulates plant resistance to *Phytophthora* | Ruirui Feng |
| 9:30 - 9:45 | NbMORF8 encoding protein localized in mitochondria and chloroplasts negatively regulates plant immunity to *Phytophthora* pathogens | Yang Yang |
| 9:50 – 10:20 | Break | |

| **10:20 - 11:40** | **Effectors III** | **Chair: Jiang Lu and Biao Gu** |
|---|---|---|
| 10:20 - 10:35 | The evolutionarily conserved Phytophthora effector RxLR24 interferes with the secretion of antimicrobial compounds | Iga Tomczynska |
| 10:40 - 10:55 | Structure and function analysis of effector PcRxLR145 of Phytophthora capsici | Liying Wang |
| 11:00 - 11:15 | The Phytophthora sojae essential effector Avh238 targets soybean GmACSs to suppress ethylene biosynthesis and promote infection | Bo Yang |
| 11:20 - 11:35 | Host mRNA methylation pathway, a new target for Phytophthora effector proteins? | Jie Huang |
| 11:40 -12:00 | Closing remarks | |
| 12:00 -2:00 | Lunch/Adjourn | |

# ABSTRACTS OF ORAL PRESENTATIONS

(in order of appearance)

| **Monday**<br><br>**April 9th** | | |
|---|---|---|
| **9:05 - 10:25** | **Oomycete biology, populations, and evolution I** | **Chair:**<br><br>**Audrey Ah-Fong**<br><br>**and Kai Tao** |
| 9:05 - 9:20 | Cellular signaling and cytoskeleton dynamics in *Phytophthora* | Francine Govers |
| 9:25 - 9:40 | Biochemical and Genetic Analyses of the Oomycete *Pythium insidiosum* Provide New Insights into Clinical Identification, Pathogenicity, and Evolution of Metabolism-Related Traits | Theerapong Krajaejun |
| 9:45 - 10:00 | Mating type change in *Phytophthora infestans* regulated by histone deacetylases | Li-Yun Guo |
| 10:05-10:20 | Ecology and evolution of oomycete communities in response to soybean seed treatments | Zachary Noel |

## Cellular signaling and cytoskeleton dynamics in *Phytophthora*

Francine Govers[1], Johan van den Hoogen[1], Kiki Kots[1,2], Tijs Ketelaar[2]
([1]Laboratory of Phytopathology and [2]Laboratory of Cell Biology, Wageningen University, Wageningen, The Netherlands)

Sensing external signals and transducing these into intracellular responses requires a balanced cellular organization that is largely governed by signal transduction pathways and cytoskeleton dynamics. We aim to gain more insight in these processes in *Phytophthora* species, oomycete plant pathogens that cause devastating diseases worldwide. Our studies have shown that *Phytophthora* spp. and other oomycetes possess unique classes of GPCR-bigrams, proteins in which a G-protein coupled receptor (GPCR) domain is linked to an accessory domain. Examples are GPCR-PIPKs that have a phosphatidylinositol kinase (PIPK) domain as accessory domain. This points to a direct connection between the two most important eukaryotic signaling pathways, G-protein signaling and phospholipid signaling. In yeast $PIP_2$ generated by PIPK influences actin dynamics but in *Phytophthora* the relation between $PIP_2$, PIPK and actin is unknown. Actin is a major component of the cytoskeleton, a well-organized, dynamic system of intracellular filaments primary composed of actin and tubulin. Previously we identified actin plaques as highly immobile, long-lived structures that are unique for oomycetes. In addition, we found two other unique actin configurations; one associated with plug deposition in germ tubes and the other with appressoria, infection structures formed prior to host cell penetration. Here we will present an update on GPCR-bigrams, the functional analyses of the Gγ subunit of the heterotrimeric G-protein, and new insights in nuclear division based on life cell imaging of the microtubule cytoskeleton. Our long-term goal is to uncover oomycete or *Phytophthora* specific features that might be instrumental for drug design.

## Biochemical and genetic analyses of the oomycete *Pythium insidiosum* provide new insights into clinical identification, pathogenicity, and

## evolution of metabolism-related traits

Thidarat Rujirawat[1,2], Teerat Kanpanleuk[1], Pitak Santanirand[1], Tassanee Lohnoo[2], Wanta Yingyong[2], Yothin Kumsang[2], Pattarana Sae-Chew[2], Preecha Patumcharoenpol[3], Weerayuth Kittichotirat[3], Theerapong Krajaejun[1]

([1]Department of Pathology, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand; [2]Research Center, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand; [3]Systems Biology and Bioinformatics Research Group, Pilot Plant Development and Training Institute, King Mongkut's University of Technology Thonburi, Bangkok, Thailand)

*Pythium insidiosum* causes the life-threatening infectious condition, pythiosis, in humans and animals. Affected individuals typically endure surgical removal of the infected organ(s). Diagnosis of pythiosis is difficult and delayed. Biochemical assays have been used to characterize *P. insidiosum*, some of which could aid in clinical identification of this organism. Although hydrolysis of maltose and sucrose has been proposed as the key biochemical feature useful in discriminating *P. insidiosum* from other oomycetes and fungi, this technique requires a more rigorous evaluation involving a wider selection of *P. insidiosum* strains. We evaluated 10 routinely-available biochemical assays for characterization of 26 *P. insidiosum* strains, isolated from different hosts and geographic origins. Initial assessment revealed diverse biochemical characteristics across the *P. insidiosum* strains. Failure to hydrolyze maltose and sucrose is observed, especially in slow-growing strains. Thus, use of the biochemical assays for identification of *P. insidiosum* should be cautioned. The ability of *P. insidiosum* to hydrolyze urea is our focus, because this metabolic process relies on the enzyme urease, an important virulence factor of other pathogens. The ability to hydrolyze urea varied among *P. insidiosum* strains, and was not associated with growth rates. Genome analyses demonstrated that urease-encoding genes are present in both urea-hydrolyzing and non-urea-hydrolyzing strains of *P. insidiosum*. Urease genes are phylogenetically-conserved in *P. insidiosum* and related oomycetes, while the presence of urease accessory protein-encoding genes is markedly-diverse in these organisms. This finding suggests that the metabolism of urea might have evolved divergently to suit the ecological niches occupied by different strains.

## Mating type change in *Phytophthora infestans* regulated by histone deacetylases

Xiao-wen Wang, Jiao-yan Tang, Tu-hong Wang, Jia-lu Lv, Yu-mei Wu, Bao-zhu Dong, Li-Yun Guo

(Department of Plant Pathology, China Agricultural University, Beijing, 100193, China)

Late blight caused by *Phytophthora infestans* is a destructive disease on potato. *P. infestans* is a heterothallic oomycete, which commonly requires the presence of both A1 and A2 mating type isolates to complete sexual reproduction. However, large amount of self-fertile isolates has found in the population of *P. infestans* in China. The occurrence of self-fertile isolates increases the frequency of forming new pathogenic type and thick walled oospores through sexual reproduction, which bring difficulty for disease control. Recently, we found that Histone deacetylases (HDACs) inhibitor, Trichostatin A, could induce oospore production in A1 self-fertile isolates. Further investigation through gene silencing and over-expressing showed that *HDACs* could regulated the expression of mating types of *P. infestans*. Here, we will report our recent progress on the mechanism involved in the mating type change of *Phytophthora.*

## Ecology and evolution of oomycete communities in response to soybean seed treatments

Zachary A. Noel, Hyunkyu Sang, Martin I. Chilvers (Department of Plant, Soil and Microbial Sciences, Michigan State University, MI 48824)

Increased crop residue and earlier planting dates exposes soybean seeds and seedlings to adverse conditions for extended periods of time, which can exacerbate oomycete infection. Seeds are coated with fungicides to protect against oomycete infection. To gain a better understanding of soybean seed treatments as community filters, culture-based techniques, evolutionary analysis and genetics were utilized. A high-throughput assay was developed and applied to evaluate the fungicide sensitivity of 81 oomycete species across ten clades to the fungicides ethaboxam and mefenoxam. Species within three separate *Pythium* clades all had reduced sensitivity to ethaboxam, suggesting that reduced sensitivity to ethaboxam is inherent and possibly related phylogenetically. Therefore, the evolutionary relationship of ethaboxam sensitivity was investigated. Bayesian phylogenetics and ancestral sequence reconstruction of *Pythium* β-tubulin gene trees indicated that species with reduced sensitivity to ethaboxam followed a convergent evolutionary pattern, and had evolved three separate times under two transversion mutations. Heterologous expression of *Pythium* tubulin genes in yeast is being used to determine the molecular mechanism of ethaboxam resistance. Based on preliminary investigations on isolation and amplicon sequencing data from roots and rhizosphere soils, filtering based on seed treatment chemistry may be subtle and location specific. Therefore, the ecological dynamics of seed treatments on oomycete communities remain difficult to explain. Currently, other ecological dynamics in combination with seed treatments are being investigated.

**Monday**

| **April 9th** | | |
|---|---|---|
| **2:00 - 3:40** | **Effectors I** | **Chair: Petra Boevink and Kelsey Wood** |
| 2:00 - 2:15 | Dissecting the transcriptional plasticity of *P. sojae* | Brett Tyler |
| 2:20 - 2:35 | Identification of Plasmopara viticola candidate RXLR effector repertoire in Nicotiana benthamiana | Jiang Lu |
| 2:40 - 2:55 | How are effectors secreted and delivered to the host ? | Shumei Wang |
| 3:00 - 3:15 | Plant recognition of *Phytophthora* GH12 MAMPs | Yan Wang |
| 3:20 - 3:35 | Characteristics of RxLR effector structures in oomycete pathogens | Li Zhao |

# Dissecting the transcriptional plasticity of *P. sojae*

Brett M. Tyler[1,2*]

([1]Interdisciplinary Ph.D. Program in Genetics, Bioinformatics & Computational Biology, Virginia Tech, VA 24061; [2]Center for Genome Research & Biocomputing and Department of Botany & Plant Pathology,

Oregon State University, Corvallis, OR, 97331)

Many oomycete plant pathogens are highly adaptable, readily overcoming chemical and genetic control measures, and jumping to new host species. Genomic studies have implicated hundreds to thousands of genes as potentially contributing to their virulence. In the soybean pathogen, *Phytophthora sojae*, the genome contains nearly 400 genes that encode RxLR effectors that can enter plant cells to promote infection. *P. sojae* strains differ in their repertoires of effector genes, in the DNA sequences of their effector genes, and to a surprisingly large extent, by which effector genes are transcribed. Using gene silencing, and more recently CRISPR/Cas9-mediated gene knockouts, we have shown that the pathogen relies on a relatively small subset of “elite” effectors that seem essential for full virulence. We have successfully targeted these genes for find new soybean resistance genes. Genetic selection for strains that come overcome the loss of essential effector genes has however revealed that the pathogen is extraordinarily effective at recovering from losses of essential effectors through epigenetic changes that affect the expression of other genes in the genome.

## Identification of *Plasmopara viticola* candidate RXLR effector repertoire in *Nicotiana benthamiana*

Yunxiao Liu[1,2],Jiang Xiang[1,2], Xinlong Li[1,2], Xia Lan[1,2], Ling Yin[3], Ian B. Dry[4], Junjie Qu[3], Jiang Xiang[2],

Jiang Lu[2,3*]
([1]College of Food Science and Nutritional Engineering, China Agricultural University, Beijing, China 100083; [2]Center for Viticulture and Enology, School of Agriculture and Biology, Shanghai Jiao Tong University, Shanghai, China 200024; [3]Guangxi Crop Genetic Improvement and Biotechnology Laboratory, Guangxi Academy of Agricultural Sciences, Nanning, China 530007; [4]Wine Innovation West Building, CSIRO Agriculture, Hartley Grove, Urrbrae, Australia, SA 5064)

Downy mildew is one of the most destructive diseases of grapevine, causing tremendous economic loss in the grape and wine industry. The disease agent *Plasmopara viticola* is an obligate biotrophic oomycete. Transcriptome and genome sequencing of this pathogen identified about 160 candidate RXLR effectors. Here, we reported that over half of the candidate effectors could suppress cell death induced by elicitin on *Nicotiana benthamiana* leaves. Live-cell imaging showed that most effectors localized in the nucleus, and some of which showed irregular sub-nuclear localizations. Interestingly, four effectors were targeted to chloroplasts, and one of which was dually targeted to chloroplasts and mitochondria. This study indicated that oomycete effectors could target organelles to modulate plant innate immunity.

## How are effectors secreted and delivered to the host ?

Shumei Wang, Petra C Boevink, Steve C Whisson, Paul R J Birch
(Division of Plant Sciences, University of Dundee, James Hutton Institute, Invergowrie, Dundee DD2 5DA, UK)

Potato blight, a ravaging disease caused by the oomycete *Phytophthora infestans*, is a major threat to global food security. *P. infestans* secretes effector proteins that are delivered inside or outside plant cells to neutralise host immunity. However, knowledge about the mechanism of the effector delivery is very limited. Recently, there is an increasing interest in the study of exosomes, which are extracellular vesicles (EVs). These EVs are secreted to facilitate intercellular and extracellular communication, to promote infection and evade host immune responses. These functions have been exploited by diverse organisms. Therefore, we raised the hypothesis that exosomes may play a key role in the dissemination of pathogen, also host-derived molecules during infection. The first EV was observed in 1975 by Carmela Shimony and John Friend, but there is little information about exosomes in *P. infestans* so far. Here, Mass Spectrometry has been performed to analyse the contents of EVs, showing that secreted EVs mediate cytoplasmic effector and some cell wall degrading enzymes translocation in *P. infestans*. This is a major breakthrough in the plant pathology community. Providing more details for helping to understand weapons of pathogen, to develop a specific chemical to disrupt these processes and inhibit pathogen infection.

## Plant recognition of *Phytophthora* GH12 MAMPs

Yan Wang[1], Yuanpeng Xu[1], Yujing Sun[1], Jiaming Qi[1], Huibin Wang[1], Bowen Wan[1], Fan Liu[1], Justin Waletich[1], Wenwu Ye[1], Suomeng Dong[1], Brett M. Tyler[2], Yuanchao Wang[1]
([1]Department of Plant Pathology, Nanjing Agricultural University, Nanjing, 210095, China; [2]Center for

Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR 97331, USA)

*Phytophthora* species are notorious plant pathogens that cause great damage on crops. During infection, *Phytophthora* pathogens secret a type of highly conserved glycoside hydrolase family 12 (GH12) proteins that can be recognized by plants as a pathogen-associated molecular pattern (PAMP). By a genome-wide assay of membrane-localized receptors, we identified the recognition receptor (RXEG1) that is responsible for response to multiple GH12 proteins. In addition, we dissected the defense-signal transduction pathways in plants upon recognition of the GH12 proteins. We profiled the receptor-like kinases downstream of RXEG1 and analyzed the receptor complexes that participate in XEG1 defense signal. Together, this study provides novel insights on plant innate immunity against *Phytophthora* pathogens and will contribute to the development of durable disease resistance.

## Characteristics of RxLR effector structures in oomycete pathogens

Li Zhao[1,3], Haizheng Fang[1,3], Rui Feng[1,3], Xuefa Zhang[1,3], Chunyuan Zhu[2,3] and Xiuguo Zhang[1,3]
([1]College of Plant Protection; [2]College of Life Sciences and [3]Shandong Provincial Key Laboratory for

Biology of Vegetable Diseases and insect, Shandong Agricultural University, Tai'an, China.)

The RxLR effector is a prominent effector class in oomycetes, comprising major virulence determinants. The RxLR class has little homology in sequence but the C-terminus forms repeat conserved core α-helical fold in 3D structure, which is defined as WY-domain. Solving the protein 3D structure is a very intuitive molecular strategy to understand the functions of effectors. Here we present five structures of RxLR effectors from *Phytophthora capsici*. PcRxLR12 and PcRxLR145 are made up of five WY-domains, PcRxLR23 consists of four WY-domains, PcRxLR81 and PcRxLR121 only have one WY-domain. There are extra "linker" helix between WY-domains of PcRxLR12, PcRxLR145 and PcRxLR23. All these structures have no clear electron density in the RxLR motif, which further prove this region is disordered. The conformational difference of these five structures shows that the polymorphism regions of WY-domain are mainly exist in K-motif and Loop-3. The surface representation of these five structures reveals that almost all K-motif and Loop-3 are full of positively charged surface. Our results extend understanding of RxLR effectors in protein structure and further function study.

| **April 9th** | | |
|---|---|---|
| **4:10 - 6:10** | **Genomics I** | **Chair: Paul Birch and Diya Sen** |
| 4:10 - 4:25 | Towards the identification of effectors and secondary metabolites involved in oomycete-oomycete interactions and mycoparasitism using comparative genomics | Laura Grenville-Briggs |
| 4:30 - 4:45 | Comparative genomics of four species of *Peronosclerospora* | Kyle Fletcher |
| 4:50 - 5:05 | What role does *Phytophthora kernoviae* play in New Zealand forests? | Rebecca McDougal |
| 5:10 - 5:25 | Land-use affects the growth and gene expression of *Phytophthora agathidicida* | Preeti Panda |
| 5:30 - 5:45 | Transcriptional regulation of *Phytophthora sojae* effectors for successful infection | Wenwu Ye |
| 5:50 - 6:05 | Genome sequence of *Plasmopara viticola* reveals effector repertoire and pathogenicity mechanisms | Ling Yin |

## Comparative genomics of four species of *Peronosclerospora*

Kyle Fletcher[1], Frank Martin[2], Fe Dela Cueva[3], Doug Luster[4], Yazmin Rivera[5], Clint Magill[6], Richard

Michelmore[1]
([1]The Genome Center, University of California, Davis, CA, USA; [2]USDA, ARS, Crop Improvement and Protection Research Unit, Salinas, CA, USA; [3]University of The Philippines Los Banos, Philppines; [4]USDA, ARS, Foreign Disease Weed Science Research Unit, Ft. Detrick, MD, USA; [5]USDA, APHIS PPQ S&T, Center for Plant Health Science and Technology; [6]Department of Plant Pathology and Microbiology, Texas A&M University, College Station, TX, USA)

*Peronosclerospora* is an under-studied oomycete genus with species that cause downy mildew diseases of important crops, particularly in tropical regions. These include *P. sorghi* on sorghum, *P. sacchari* and *P. philippinensis* on sugarcane and *P. maydis* on maize. Genomic investigations of these species were performed by sequencing 10x Genomics libraries of two modern *P. sorghi* isolates from Texas and historical isolates of *P. sacchari* (2 isolates), *P. philippinensis* (1 isolate) and *P. maydis* (4 isolates) originating in Asia and stored frozen at Ft. Detrick. Resulting assemblies are large and fragmented and highly contaminated; however, BUSCO scores indicate that the assemblies are nearly complete. The best assembly belongs to a contemporary, largely homozygous *P. sorghi* isolate (pathotype 6). The scaffold $N_{50}$ was over 100 Kb with a total assembly size of 269 Mb. Applying Dovetail Hi-C and HiRise scaffolding to this assembly improved the contiguity to a scaffold $N_{50}$ of over 10 Mb. This assembly shares large regions of synteny with *Phytophthora sojae* (v3.1). Further assembly optimization, to obtain better assemblies of *Peronosclerospora* species using 10x Genomics technology, followed by annotation are under-way. Illumina paired-end libraries of additional isolates from Ft. Detrick and contemporary isolates of *P. philippinensis* have been sequenced. Mitochondrial and k-mer analysis indicates that *P. philippinensis* is conspecific with *P. sacchari*, while at least two species may have been collected from corn characterized as *P. maydis*. Genomic characterization of these pathogens will allow development of novel diagnostic and control strategies disease management/prevention.

## What role does Phytophthora kernoviae play in New Zealand

## forests?

Rebecca McDougal[1], Preeti Panda[1], David Studholme[2], Eugenio Sanfuentes[3], and Nari Williams[1]
([1]Scion (New Zealand Forest Research Institute, Ltd.), Rotorua, New Zealand; [2]Biosciences, University of Exeter, Stocker Road, Exeter, EX4 4QD, United Kingdom; [3]Laboratorio de Patología Forestal, Facultad Ciencias Forestales y Centro de Biotecnología, Universidad de Concepción, Concepción, Chile)

*Phytophthora kernoviae* is an invasive plant pathogen first described in the UK, and later found in New Zealand and Chile. Comparative genomic analysis with genome sequences of *P. kernoviae* isolates from Britain, New Zealand and Chile was performed. This revealed that the New Zealand isolates were genetically more variable that those from Britain or Chile, supporting the current hypothesis that *P. kernoviae* is endemic to New Zealand. *P. kernoviae* is known to have been present in New Zealand since at least the 1950s, but it's impact on *Pinus radiata* plantations was not recognised until another *Phytophthora* species, *P. pluvialis*, was discovered on radiata pine in the early 2000s. *P. kernoviae* is now considered to be the causal agent of a disease that was first recognised in the early 1980s causing disease on *P. radiata*. Initially this disease, known as physiological needle blight (PNB), was considered abiotic as a causal agent could not be identified. Detection of *P. kernoviae* in herbarium data suggests that this pathogen may have been associated with PNB since the 1980s. *In planta* infection studies with detached needles using *P. kernoviae*, and also *P. pluvialis*, have been performed and transcriptomic analysis completed. Higher infection rates were observed with *P. pluvialis* than *P. kernoviae* as evidenced by lesion number, pathogen recovery by isolation and also transcriptomics read counts. Elicitin, RxLR and CRN gene predictions were performed and their gene expression analysed. The results of these studies will be discussed in relation to the potential role of *P. kernoviae* in New Zealand forests.

## Land-use affects the growth and gene expression of *Phytophthora*

## *agathidicida*

Preeti Panda[1], Shane Sturrock[1], Rebecca McDougal[1], Peter Scott[1], Kai Lewis[2], Amanda Black[2], Leo Condron[2], Maureen O'Callaghan[2] & Nari Williams[1]
([1]Scion (New Zealand Forest Research Institute, Ltd.), 49 Sala Street, Rotorua, New Zealand; [2]Bio-Protection Research Centre, PO Box 85084, Lincoln University, Lincoln 7647, New Zealand)

Kauri (*Agathis australis*) is a New Zealand native and iconic tree species of cultural significance, an important ecosystem engineer and valuable timber resource. *Phytophthora agathidicida*, an aggressive soilborne pathogen causes dieback in kauri trees of all ages. This pathogen infects structural roots and damages tissues that distribute nutrients and water within the tree. Surveillance between 2008 and 2017 has shown widespread distribution of the pathogen in many of the regions where kauri is established illustrating the threat to the long- term survival of the species. To investigate the impact of land-use on the growth and spread of this pathogen, the growth response and gene expression profile of *P. agathidicida* grown within soils from three different land-uses (indigenous kauri forest, pasture land and commercial pine forest) were assessed to determine whether these soils were conducive, suppressive, or benign to pathogen growth over time. *Phytophthora agathidicida* was grown in each soil by exposing mycelial mats to soil extracts from each land-use to determine sporulation and growth responses over a 4-day period. Gene expression was also analysed in parallel to better understand the direct response of the pathogen within each soil. Results suggest that there is a difference in sporangia production with time and between soils in association with observed variation in gene expression. Expression of the most abundant gene classes, their associated molecular functions and biological processes, with respect to the life stage development and soil conditions will be presented and the implications for pathogen survival discussed.

## **Transcriptional regulation of *Phytophthora sojae* effectors for**

## successful infection

Wenwu Ye[1], Yang Wang[1], Long Lin[1], Jiawei Wu[1], Yan Wang[1], Suomeng Dong[1], Xiaobo Zheng[1], Brett M Tyler[2], Yuanchao Wang[1*]

([1]Department of Plant Pathology, Nanjing Agricultural University, Nanjing, Jiangsu 210095, China; [2]Center for Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR 97331, USA)

*Phytophthora* belongs to oomycetes which form a distinct phylogenetic lineage of fungus-like eukaryotic microorganisms, but actually share an ancestor with the non-pathogenic organisms, such as brown algae and diatoms. After the long term evolutionary process, successful *Phytophthora* pathogens have obtained an arsenal of virulence factors and evolved regulatory mechanisms required to deploy the virulence factors. For example, in the genome of the soybean root rot pathogen *Phytophthora sojae*, there is a superfamily of effectors, namely RxLR family, which has over 380 members. We have found that the transcription of RxLR effectors could be differentially regulated following infection process, and different classes of effectors target different functional branches of the plant defense response. Our recent results further suggest that transcriptional regulation of pathogenicity-related genes (including many effector genes) underlies the organ-specific infection by *P. sojae*, and that a novel bZIP transcription factor plays a key role. In addition to the regulation by transcription factors, we also found a potential association between long non-coding RNAs (lncRNAs) and effector genes.

## Genome sequence of *Plasmopara viticola* reveals effector repertoire

## and pathogenicity mechanisms

Ling Yin[1,2,*], Yunhe An[1,3,*], Junjie Qu[2,*], Xinlong Li[1], Yali Zhang[1], Ian Dry[4], Huijuan Wu[3✉], Jiang Lu[1,5✉] ([1]College of Food Science and Nutritional Engineering, China Agricultural University, Beijing 100083, China; [2]Guangxi Crop Genetic Improvement and Biotechnology Laboratory, Guangxi Academy of Agricultural Sciences, Nanning 530007, China; [3]Beijing Center for Physical and Chemical Analysis, Beijing 100089, China; [4]CSIRO Agriculture& Food, Wine Innovation West Building, Hartley Grove, Urrbrae, SA 5064, Australia; [5]Center for Viticulture and Enology, School of Agriculture and Biology, Shanghai Jiao Tong University, Shanghai 200024, China; *These authors contributed equally to this work. Correspondence and requests for materials should be addressed to sunnywhj@126.com or jiang.lu@sjtu.edu.cn)

*Plasmopara viticola* causes downy mildew disease of grapevine which is one of the most devastating diseases of viticulture worldwide. Here we report a 101.3 Mb whole genome sequence of *P. viticola* isolate ‘JL-7-2’ obtained by a combination of Illumina and PacBio sequencing technologies. The P. viticola genome contains 17,014 putative protein-coding genes and has ~26% repetitive sequences. A total of 1,301 putative secreted proteins, including 100 putative RXLR effectors and 90 CRN effectors were identified in this genome. In the secretome, 261 potential pathogenicity genes and 95 carbohydrate-active enzymes were predicted. Transcriptional analysis revealed that most of the RXLR effectors, pathogenicity genes and carbohydrate-active enzymes were significantly up-regulated during infection. Comparative genomic analysis revealed that *P. viticola* evolved independently from the Arabidopsis downy mildew pathogen *Hyaloperonospora arabidopsidis*. The availability of the *P. viticola* genome provides a valuable resource not only for comparative genomic analysis and evolutionary studies among oomycetes, but also enhance our knowledge on the mechanism of interactions between this biotrophic pathogen and its host.

| **Wednesdy**<br><br>**April 11th** | | |
|---|---|---|
| 8:30 - 9:50 | **Host Interactions and Resistance Mechanisms I** | **Chair:**<br><br>**Jun Zhao**<br><br>**and Veronica Ancona** |
| 8:30 - 8:45 | *Phytophthora palmivora* establishes tissue-specific intracellular infection structures in the earliest divergent land plant lineage | Sebastian Schornack |
| 8:50 - 9:05 | A pathogen suppressor of an NLR immune network | Lida Derevnina |
| 9:10 - 9:25 | A plant pathogen effector utilizes host susceptibility factor NRL1 to degrade the immune regulator SWAP70 | Qin He |
| 9:30 - 9:45 | A potato STRUBBELIG-RECEPTOR FAMILY gene StLRPK1 involves in plant immunity by association with StSERK3/BAK1 | Haixia Wang |

## *Phytophthora palmivora* establishes tissue-specific intracellular

## infection structures in the earliest divergent land plant lineage

Philip Carella[1], Anna Gogleva[1], Marta Tomaselli[1,2], Carolin Alfs[1], David Hoey[1], Sebastian Schornack[1,2] ([1]Sainsbury Laboratory (SLCU), University of Cambridge, Cambridge, CB2 1LR, UK; [2]Dept. of Plant Sciences, University of Cambridge, Cambridge, CB2 3EA, UK)

The expansion of plants onto land was a formative event that brought forth profound changes to the Earth's geochemistry and biota. Filamentous eukaryotic microbes developed the ability to colonize plant tissues early during the evolution of land plants, as demonstrated by intimate symbiosis-like associations in >400 million-year-old fossils. However, the degree to which filamentous microbes establish pathogenic interactions with early divergent land plants is unclear. We demonstrate that the broad host-range oomycete pathogen *Phytophthora palmivora* colonizes liverworts, the earliest divergent land plant lineage. We show that *P. palmivora* establishes a complex tissue-specific interaction with *Marchantia polymorpha*, where it completes a full infection cycle within air chambers of the dorsal photosynthetic layer. *P. palmivora* invaginates *M. polymorpha* cells with haustoria-like structures that accumulate host cellular trafficking machinery and the membrane-syntaxin MpSYP13B but not the related MpSYP13A. Our results indicate that the intracellular accommodation of filamentous microbes is an ancient plant trait that is successfully exploited by pathogens like *P. palmivora*.

## A pathogen suppressor of an NLR immune network

Lida Derevnina, Jessica Upson, Chih-hang Wu and Sophien Kamoun
(The Sainsbury Laboratory, Norwich, United Kingdom, NR4 7UH)

Genetic networks are known to boost evolvability and robustness of biological processes. We recently described a plant immune signalling network in which several sensor NLR immune receptors (nucleotide-binding domain and leucine-rich repeat proteins) converged to signal through three partially redundant helper NLRs (NRC2, NRC3, NRC4). This NLR network confers resistance against diverse pathogens, including oomycetes, bacteria, viruses, nematodes, and insects. We reasoned that NRC redundancy in this network confers advantages for the plant to evade immune suppression by pathogens. To test this hypothesis, we screened *Phytophthora infestans* effectors for differential suppression of NRC2, NRC3 and NRC4-mediated immune responses. We identified an effector candidate, namely AVRcap1b, which suppresses NRC2 and NRC3 but not NRC4-dependent cell death. To understand the molecular basis of immune suppression specificty by AVRcap1b, we generated a set of chimeric NRC variants by swapping domains between NRC3 and NRC4. We found that swapping out the N-terminal coiled-coil nucleotide binding site (CC-NB) domains of NRC3 with NRC4 resulted in evasion of AVRcap1b suppression. Co-immunoprecipitation studies suggest that AVRcap1b does not associate with NRC proteins. Currently, we are using immunoprecipitation with mass spectrometry and yeast-two-hybrid analysis to identify host targets of AVRcap1b. Our results support the view that helper NLR redundancy increases robustness of plant immunity in the arms race between plants and pathogens. Understanding the mechanisms of pathogen suppression of NLR networks can offer new opportunities to generate disease resistant crops.

## A plant pathogen effector utilizes host susceptibility factor

## NRL1 to degrade the immune regulator SWAP70

Qin He[1§], Shaista Naqvi[1§], Hazel McLellan[1], Petra Boevink[2], Nicolas Champouret[3], Ingo Hein[1,2], Paul R J Birch[1,2*]

§These authors contributed equally to this work

([1]Division of Plant Science, University of Dundee, at James Hutton Institute, Invergowrie, Dundee DD2 5DA, UK; [2]Cell and Molecular Sciences, James Hutton Institute, Invergowrie, Dundee DD2 5DA, UK; [3]J.R. Simplot Company, Simplot Plant Sciences, Boise, Idaho, United States of America)

Plant pathogens, such as the infamous potato blight agent *Phytophthora infestans*, cause disease by deploying virulence effectors that manipulate various host targets. Some host targets, called susceptibility (S) factors, may be activated or used by effectors to promote disease. S factors are often negative regulators of immunity. A major unresolved issue in the current effector-target interactions, however, is the mechanism of how pathogens use S factors to suppress immunity and how plants overcome the pathogen. We discovered that *P. infestans* effector Pi02860 uses the potato ubiquitin E3 ligase StNRL1 as an S factor by promoting its ability to target a positive regulator of immunity, a GEF protein StSWAP70, for proteasome-mediated degradation. The degradation of SWAP70 may interfere with the proper recycling of the receptor through RAC1, thereby dampening the immune response. Our results reveal that protecting StSWAP70 from degradation either by reducing the activity or availability of NRL1 could enhance plant immunity. We further found Pi02860 and NRL1 both interact with a 14-3-3 protein, which positively regulates plant immunity, potentially by preventing NRL1 from mediating the degradation of SWAP70. This illustrates an example of the complex molecular battle between plant and pathogen in which an effector/S factor complex targets a component of plant immunity for destabilization. Our results also highlight attractive opportunities to undermine infection processes effectively toward improving disease resistance in crops.

## A potato STRUBBELIG-RECEPTOR FAMILY gene *StLRPK1*

## involves in plant immunity by association with StSERK3/BAK1

Haixia Wang[1,2], Yanlin Chen[1], Xingtong Wu[1,2], Zongshang Long[1], Chunlian Sun[1], Zhejuan Tian[1], Hairong Wang[1], Zhendong Tian[1,2,§]

([1]Key Laboratory of Potato Biology and Biotechnology, Ministry of Agriculture, Huazhong Agricultural University, Wuhan 430070, China; [2]Key Laboratory of Horticultural Plant Biology (HZAU), Ministry of Education, Huazhong Agricultural University, Wuhan 430070, China)

Plant *STRUBBELIG(SUB)-RECEPTOR FAMILY (SRF)* gene encodes putative leucine-rich repeat transmembrane receptor-like kinase (LRR-RLK). *SRFs* have been reported dominantly playing an essential role in tissue morphogenesis of many plant organs. Here, we show that a potato *SRF* gene *StLRPK1* involves in plant immunity. *StLRPK1* encodes a typical LRR-RLK which is located at the cell plasma membrane and it was strongly induced by late blight pathogen *Phytophthora infestans* culture filtrate (CF). Overexpression of *StLRPK1* in stable transgenic potato or ectopic expression in *Nicotiana benthamiana* plants strongly enhance *P. infestans* resistance. Whereas RNA interference of *StLRPK1* in potato decreases *P. infestans* resistance. Moreover we found that StLRPK1 interacts with a pivotal co-receptor SERK3/BAK1 which plays a central role in plant immunity. Interaction in planta was confirmed by co-immunoprecipitation and bimolecular fluorescence complementation assays and demonstrated to occur at the plant plasma membrane. Virus-induced gene silencing (VIGS) of *SERK3/BAK1* in *N. benthamiana*, which ectopically and stably expressing *StLRPK1*, attenuates *P. infestans* resistance, indicating that SERK3/BAK1 is essential for *StLRPK1* mediated resistance. Finally, we show that StLRPK1-trigerred late blight resistance depends on mitogen-activated protein kinase kinase (MAP2K) MEK2 and mitogen-activated protein kinase (MAPK) WIPK. We propose a model that the StLRPK1 associates with SERK3/BAK1 to positively regulate plant innate immunity against *P. infestans* through MAPKs cascades. These data provide new insights into our understanding of SRF function in plant immunity.

| **Wednesday April 11th** | | |
|---|---|---|
| 10:20 - 12:00 | **Effectors II** | **Chair: Suomeng Dong and Lida Derevnina** |
| 10:20 - 10:35 | *Phytophthora infestans* RXLR Effector Pi22926 suppresses plant immunity by targeting the host MAP3KÎ² to malipulate MAP3K signalling pathway | Zhendong Tian |
| 10:40 - 10:55 | Biochemical basis of an RXLR-WY effector suppression of an NLR network | Jessica Upson |
| 11:00 - 11:15 | Characterization of host-recognized WY domain effectors from *Bremia lactucae* that lack the canonical RxLR motif | Kelsey Wood |
| 11:20 - 11:35 | Functional analysis of an effector PcRxLR101 in *Phytophthora capsici* | Jing Li |
| 11:40 - 11:55 | A potato chloroplast kinase is required for Rpi-vnt1 mediated immunity against late blight pathogen | Chuyun Gao |

## *Phytophthora infestans* RXLR Effector Pi22926 suppresses plant

## immunity by targeting the host MAP3Kβ to malipulate MAP3K signalling pathway

Yajuan Ren[1,2], Jing Zhou[1,2], Zhendong Tian[1,2*]
([1]Potato Biology and Biotechnology Key Laboratory of Ministry of Agriculture, China; [2]Key Laboratory of Potato Biology and Biotechnology, Ministry of Agriculture, China, Huazhong Agricultural University, Wuhan, 430070, China); *Tianzhd@mail.hzau.edu.cn

*Phytophthora infestans* secretes number of RxLR effector proteins into host cells to subvert plant immune responses and promote colonization. *P. infestans* RxLR effector PITG_22926 was up-regulated during early infection of potato. Transient expression of PITG_22926 promotes *P. infestans* colonization on *N. benthamiana* leaves. PITG_22926 locates in the host nucleus and interacts with a potato Mitogen Activated Protein Kinase MAP3Kβ protein. VIGS *MAP3Kβ* reduces *P. infestans* colonization, indicating that this host protein acts as a positive regulator of immunity. Moreover, PITG_22926 specifically suppresses Avr4/Cf4 and Avrpto/Pto triggered HR. PITG_22926 also suppresses MAP3Kβ triggered cell death. We proved evidence that *P. infestans* secrets RxLR effector PITG_22926 into host cell to suppress immunity by targeting kinase of the MAP3Kβ. Here, we will report our recent progress on the mechanistic investigation of how PITG_22926 manipulates Mitogen Activated Protein Kinase signalling pathway to promote *P. infestans* colonization.

## Biochemical basis of an RXLR-WY effector suppression of an

## NLR network

Jessica Upson, Lida Derevnina, Chih-Hang Wu, Abbas Maqbool, Sophien Kamoun
(The Sainsbury Laboratory, Norwich, UK)

Pathogens secrete effectors to modulate host processes and to promote infection. Many of these effectors function by suppressing plant immune responses to facilitate disease. We recently showed that the *Phytophthora infestans* effector AVRcap1b, a large RXLR protein predicted to have seven WY domains, can perturb immunity by suppressing cell death mediated by the helper NLRs (nucleotide-binding domain and leucine-rich repeat proteins) within a large NLR network. However, the mechanisms by which AVRcap1b suppresses cell death is not clear. To understand the structural basis of AVRcap1b activity, we generated truncation mutants from the predicted WY domains. Our preliminary results suggest that the cell death suppression ability of AVRcap1b resides in a WY domain located at the C-terminus. We are currently employing biophysical and structural approaches to study the molecular architecture of AVRcap1b. Our aim is to understand how tandem WY domains organise in 3D and highlight regions important for interactions within host proteins. Additionally, AVRcap1b orthologs are present in other, closely related, *Phytophthora* species. We plan to exploit the polymorphisms of these AVRcap1b orthologs and test these for suppression of cell death. Understanding the structural basis behind AVRcap1b function and polymorphisms will help to elucidate how *P. infestans* manipulates its host to cause disease.

## Characterization of host-recognized WY domain effectors from

## *Bremia lactucae* that lack the canonical RxLR motif

Kelsey Wood[1], Kyle Fletcher[1], Ayumi Gothberg[1], Juliana Gil[1], Sebastian Reyes Chin Wo[1], Yi Zhai[2], Wenbo Ma[2] and Richard Michelmore[1]
([1]University of California, Davis, CA; [2]University of California, Riverside, CA)

Identification of effectors in oomycete genomes is foundational for understanding the mechanisms of pathogenesis, monitoring field pathogen populations, and for breeding resistant plants. Using comparative genomics and bioinformatics, we identified candidate effectors from the economically important downy mildew of lettuce, *Bremia lactucae*, by searching for WY domains, conserved structural elements implicated in the immune-supressing function of *Phytophthora* effectors. Searching for the WY-domain revealed additional effector candidates that were missed by searching for the RxLR domain alone. These candidate effectors have several characteristics of pathogen effectors, including an N-terminal secretion signal, lineage specificity, and evidence of gene duplication and gene family expansion. Unexpectedly, only a minority of WY effectors contained the canonical N-terminal RxLR motif, which is reported to be a conserved feature in *Phytophthora* effectors. Functional characterization of 21 of the WY domain effectors revealed seven effectors that elicited an immune response on lettuce lines containing introgressions of resistance genes from wild *Lactuca* species. None of these immunity eliciting effectors contained a canonical RxLR motif. One of the recognized WY effectors was found to have RNA silencing suppression activity. Our results indicate that there has been evolutionary divergence in sequence motifs between genera that has important implications for effector prediction in oomycetes.

## Functional analysis of an effector PcRxLR101 of *Phytophthora*

## *capsica*

Li Zhang[1,2], Jing Li[1,2], Haiqing Xu[1,2], Peng Ding[1,2], Tao Liang[1,2], Ruxue Chen[1,2], Liying Wang[1,2], Xiuguo Zhang[1,2]
([1]College of Plant Protection, [2]Shandong Provincial Key Laboratory for Biology of Vegetable Diseases and insect, Shandong Agricultural University, Tai'an, China)

*Phytophthora capsici* is a worldwide soil-borne disease that can have a huge impact on the production of vegetables each year. *P. capsici* pathogen secretes a large array of effectors during infection of host plants. The *P. capsici* genome contains nearly 400 RxLR effectors. RxLR effector0s play an important role in infecting the host and interaction between *P.capsici* and plants. A RxLR effector PcRxLR101 of *P. capsici*, is a homologous gene PpAvh121 of *P. parasitica*, was cloned from *P. capsici* SD33 strain. Our study showed that PcRxLR101 located at the cytoplasmic and nucleus. PcRxLR101 enhanced expression at the early stages of infection and it could suppress BAX-induced cell death. PcRxLR101 was truncated into five domains. Notably, one of these five domains PcRxLR101-Y (240aa-263aa) is still suppressed BAX-induced cell death. PcRxLR101-Y could also suppress the infection of *P.capsici* in *Nicotiana. benthamiana*. Whereas, PpAvh121 induced an HR-like cell death response in *N. benthamiana*. In order to further study the function of PcRxLR101, mCherry was used to replace PcRxLR101 by CRISPR/Cas9. PcRxLR101 homology-directed repair (HDR) reduced the zoospore production, which decreased their virulence on pepper, but not effected the growth of *P. capsici* transformants. Moreover, we obtained two homozygous PcRxLR101 transgenic *Arabidopsis thaliana* by floral-dip transformation. Preliminary results showed that PcRxLR101 probably improved roots growth, but suppressed leaf and stems growth in transgenic plants.

## **A potato chloroplast kinase is required for *Rpi-vnt1* mediated**

## immunity against late blight pathogen

Chuyun Gao[1],¶, Huawei Xu[1],¶, Jie Huang[1], Tingxiu Yan[1], Biying Sun[1], Jie Xu[1], Chih-hang Wu[2], Joe Win[2], Yuanchao Wang[1], Vivianne Vleeshouwers[3], Sophien Kamoun[2], Suomeng Dong[1*]
([1]College of Plant Protection, Nanjing Agricultural University, Nanjing, 210095, China; [2]The Sainsbury Laboratory, Norwich Research Park, Norwich NR4 7UH, United Kingdom; [3]Wageningen UR Plant Breeding, Wageningen University and Research Centre, Droevendaalsesteeg 1, Wageningen 6708 PB, Netherlands)

Pathogen effector genes with an avirulence (AVR) activity interplay with host resistant (R) genes determines the outcome of plant disease. Studying the underlying mechanism is important not only to understand the co-evolution between plant and pathogen but also to deploy effective crop resistance. *Phytophthora infestans* is the casual agent of the Irish potato famine in mid-nineteenth and remains a destructive pathogen that threatens global food security. Although many R genes against *P. infestans* have been identified, little is known about the recognition mechanisms of these R genes. It is known previously that *P. infestans* effector Avrvnt1 can be recognized by a cognate nucleotide binding leucine-rich repeat (NLR) receptor Rpi-vnt1 from *Solanum venturii* and consequently develops resistance in potato. Here, we performed a yeast two-hybrid screening, and identified the chloroplast kinase GLYK from *Solanum tuberosum* as a putative Avrvnt1 binding protein. Transient silencing of *NbGLYK* in *N. benthamiana* specifically compromised Avrvnt1 triggered cell death. Furthermore, this defective cell death can be complimented by overexpression of a synthetic full length *GLYK* constructs but not by the binding-deficient *GLYK* mutant construct. Meanwhile, *GLYK* kinase dead mutant also restore cell death phenotype, suggesting kinase activity may not be required for Rpi-vnt1 mediated cell death. Our cell biology and biochemical data demonstrated that the chloroplast translocation of GLYK can be blocked in the presence of Avrvnt1 *in planta*. All these data suggest that plant GLYK participate in Rpi-vnt1 perception to Avrvnt1. The roles of GLYK in Rpi-vnt1 mediated resistance require further investigations.

| **Wednesdy**<br><br>**April 11th** | | |
|---|---|---|
| **2:00 - 3:20** | **Oomycete biology, populations, and evolution II** | **Chair:**<br><br>**Daolong Dou**<br><br>**and Theerapong**<br><br>**Krajaejun** |
| 2:00 - 2:15 | Molecular genetics of the oomycete pathogen of lettuce, *Bremia lactucae*. | Lin Zhang |
| 2:20 - 2:35 | Structure determination of three enzymes from *Phytophthora capsici* | Weiwei Song |
| 2:40 - 2:55 | Profiling of the *Phytophthora* epigenomes | Han Chen |
| 3:00 - 3:15 | Immunity triggered by the small cysteine rich effector PC2 from *Phytophthora infestans* requires proteolytic cleavage by the host protease P69B | Shuaishuai Wang |

## Molecular genetics of the oomycete pathogen of lettuce, *Bremia*

## *lactucae*

Lin Zhang[1,2], Kyle Fletcher[1], Juliana Gil[1], Kelsey Wood[1], Aubrey Kenefick[1], Lien Bertier[1], Keri Cavanaugh[1], Cayla Tsuchida[1], Richard Michelmore[1]
([1]The Genome Center, University of California, Davis, California 95616, USA; [2]Current Address: Department of Horticulture, College of Agriculture and Biotechnology, Zhejiang University, Hangzhou, China 310058)

Lettuce downy mildew is the major disease affecting lettuce worldwide. However, deployments of resistance genes and fungicide have suffered from ephemeral efficacy due to rapid (a)sexual variation in *B. lactucae*. Both top-down and bottom-up approaches are being used to understand the molecular basis of pathogenicity traits and identify candidate genes to develop more durable control strategies. A high-quality draft genome has been assembled (14 chromosome-scale scaffolds spanning 136 Mb, N50 13.35 Mb). Six F1 or backcross populations were generated with Californian, European, historical, and modern parents including one used for the reference genome and two subsequently inferred as heterokaryotic. Genetic segregation patterns of 248 individuals were determined for 24 avirulence phenotypes, Mefenoxam sensitivity, and mating type, as well as genetics of heterokaryosis. Ultra-dense SNP-based genetic maps are being constructed from WGS data using a pseudo-testcross phase-aware strategy. Linkage analysis has identified regions segregating with virulence against several downy mildew resistance genes, which are undergoing transient assays *in planta*. Candidate effector genes encoding proteins with RxLR motifs and/or WY domains mined *in silico* are being tested similarly. Variants in these genes and mapping candidates are of special focus in linkage and downstream analyses. Draft genomes (Illumina & PacBio) of another five parental isolates together with data on isolates of diverse geographical origins and history are facilitating haplotype assembly, genotyping, and downstream analysis. This study is revealing candidate genes for important traits and will be the foundation for understanding of the molecular genetics of this economically important downy mildew.

## **Structure determination of three enzymes from *Phytophthora***

## *capsici*

Weiwei Song[1], Xiuqi Li[1], Cancan Yang[1], Zhenling Huang[1], Xiaolei Gao[1], Chunyuan Zhu[2], Xiuguo Zhang[1]
(1College of Plant Protection, Shandong Agricultural University, Taian, China, 271018; 2College of Life sciences, Shandong Agricultural University, Taian, China, 271018)

*Phytophthora capsici* is a notorious phytopathogenic oomycete that causes devastating diseases on many economically important vegetables and has a huge impact on agriculture. Understanding the basic biology of *Phytophthora* and other oomycete pathogens play an important role in the development of novel strategies for controlling the diseases. Enzyme catalysis involves in almost all metabolic processes in cell, which is necessary for sustain life. To study enzymes that take part in the processes of growth, development and pathogenesis of Phytophthora might provide new targets for chemical fungicides screening. We have identified three different enzymes from P.capsici and solved their crystal structures: i) a putative 2-oxoglutarate and Fe2+-dependent prolyl hydroxylase, PcPHD, which is strikingly similar to the hypoxia-inducible transcription factor prolyl hydroxylases in quaternary structure, while it has different substrate specificity. ii) a novel Thiamine diphosphate (ThDP)-dependent enzyme, PcTDD, plays a key role in the pyruvate metabolism. iii) PcUP, a uridine phosphorylase, that is a central enzyme of pyrimidine salvage pathways. Compared with 2'-deoxyuridine, PcUP has a higher catalytic efficiency for uridine. Future efforts will focus on identifying specific substrates for these enzymes. The deeper understanding of these enzymes is expected to help to characterize new protein targets for controlling oomycete-related diseases.

## Profiling of the *Phytophthora* epigenomes

Han Chen[1], Haidong Shu[1], Liyuan Wang[1], Fan Zhang[1], Xi Li[1], Hairong Xie[2], Sylvans Ochieng Ochola[1],

Fei Mao[2], Hongyu Ma[1], Wenwu Ye[1], Tingting Gu[3], Lubing Jiang[4], Yufeng Wu[2], Yuanchao Wang[1], Sophien Kamoun[5], Suomeng Dong[1]

([1]College of Plant Protection, Nanjing Agricultural University, Nanjing, Nanjing 210095, China; [2]National Key Laboratory for Crop Genetics and Germplasm Enhancement, Nanjing Agricultural University, Nanjing 210095, China; [3]College of Horticulture, Nanjing Agricultural University, Nanjing 210095, China; [4]Institute Pasteur of Shanghai, Chinese Academy of Sciences, Shanghai 200031, China; [5]The Sainsbury Laboratory, Norwich Research Park, Norwich NR4 7UH, United Kingdom)

Oomycetes, a distinct group of filamentous eukaryotes that are related to brown algae and diatoms, include many notorious animal and plant pathogens such as *Phytophthora infestans*, the casual agent of Irish potato famine in mid-nineteenth century. Genome sequencing of oomycetes uncovers complex genome architecture with gene sparse, repeat-rich compartments serving as a cradle for adaptive evolution. Increasing evidences suggest epigenetic regulation play important roles in rapid adaptability of the pathogen to host plants. However, the epigenomes and their functions in this group of organisms remain largely unknown. Here, we show that the oomycete plant pathogens *Phytophthora infestans* and *Phytophthora sojae* possess functional adenine N6-methylation (6mA) methyltransferases that modulate patterns of 6mA marks across the genome. Methylated DNA Immunoprecipitation Sequencing (MeDIP-seq) of each species revealed that 6mA is depleted around the transcriptional starting sites (TSS) and is associated with low expressed genes, particularly transposable elements. Remarkably, genes occupying the gene-sparse regions have higher levels of 6mA compared to the remainder of both genomes, possibly implicating the methylome in adaptive evolution of *Phytophthora*. Beside DNA methylation, we will also present our recent study on profiling histone H3 lysine 27 tri-methylation (H3K27me3) and histone H3 tri-methylation (H3K36me3) modifications across the genomes by Chromatin Immunoprecipitation Sequencing (ChIP-seq). These epigenomic data will provide new insights into oomycete genome structures and organizations, and facilitate our understanding of pathogen adaptation on host adaptive evolution.

## Immunity triggered by the small cysteine rich effector PC2 from *Phytophthora infestans* requires proteolytic cleavage by the host

## protease P69B

Shuaishuai Wang[a], Rongkang Xing[a], Yan Wang[a], Diane Saunders[b], Liliana M. Cano[b], Mark Youles[b], Yuanchao Wang[a], Xiaobo Zheng[a], Vivianne Vleeshouwers[c], Renier van der Hoorn[d], Sophien Kamoun[b], Suomeng Dong[a*]

([a]College of Plant Protection, Nanjing Agricultural University, Nanjing, China; [b]The Sainsbury Laboratory, Norwich Research Park, Norwich NR4 7UH, United Kingdom; [c]Wageningen UR Plant Breeding, Wageningen University and Research Centre, Droevendaalsesteeg 1, Wageningen 6708 PB, The Netherlands; [d]Department of Plant Sciences, University of Oxford, South Parks Road, Oxford OX1 3RB, United Kingdom)

Oomycetes, a group of filamentous plant pathogens, are responsible for many notorious crop diseases such as potato late blight that threaten global food security. Interactions between plants and oomycetes involve several apoplastic players. One example includes small cysteine rich (SCR) effector genes, which tend to be highly induced during oomycete infection. However, the molecular mode of actions of the majority of SCR effectors remain largely unknown. A preliminary bioinformatics analysis combined with high-throughput *in planta* screening assay resulted in the identification of a *Phytophthora infestans* cysteine rich effector PC2, which could trigger significant reactive oxygen burst, defence related gene induction and hypersensitive response in *Nicotiana benthamiana* and potato. Unlike many other *SCR* genes, *PC2* orthologs are conserved in genomes of a variety of oomycetes including species from *Phytophthora*, *Hyaloperonospora* and *Pythium*. Furthermore, chemical inhibition assay suggesting that serine proteases are involved in PC2 perception by plants. Additionally, PC2 effector could be proteolytic cleaved by tomato serine protease P69B, and this action can be compromised by adding P69B protease inhibitors such as EPI1 from *P. infestans*. Consistently, transient overexpression EPI inhibitors or silencing P69B homologous in *N. benthamiana* significantly impaired PC2 triggered immune response, suggesting plant P69B protease activity is involved in PC2 triggered immunity. In summary, we identified a novel SCR effector with immune induction activity, and studying the underlying mechanism uncovers that proteolytic cleavage of effectors maybe an important layer of plant-oomycete interaction.

**Wednesdy**

| **April 11th** | | |
|---|---|---|
| **3:50 - 5:30** | **Genomics II** | **Chair: Sucheta Tripathy and Wenwu Ye** |
| 3:50 - 4:05 | New insights into pathogenicity of the emerging tropical pathogen: *Phytophthora colocasiae* on taro | Diya Sen |
| 4:10 - 4:25 | Mutations in oxysterol binding protein-related protein conferring oxathiapiprolin resistance confirmed using CRISPR/Cas9 in *Phytophthora capsici* and P. sojae. | Jianqiang Miao |
| 4:30 - 4:45 | Detection of multiple oomycetes in metagenomic data by Using E-probe Detection of Nucleic Analysis (EDNA) | Maria Fernanda Proano |
| 4:50 - 5:05 | EumicrobeDBLiteV11: a lightweight genomic resource and analytic platform for draft oomycete genomes | Arijit Panda |
| 5:10 - 5:25 | Diploid genome assembly works better when Pacbio long reads are supplemented with Illumina reads: *P. ramorum* assembly a case study | Mathu Malar C |

## New insights into pathogenicity of the emerging tropical pathogen: *Phytophthora colocasiae* on taro

Diya Sen[1*], Ramesh R. Vetukuri[1], Sandeep K. Kushwaha[2], Kurt H. Lamour[3], Sucheta Tripathy[4], Laura Grenville-Briggs[1]

([1]Department of Plant Protection Biology, Swedish University of Agricultural Sciences, Alnarp, Sweden; [2]Department of Plant Breeding, Swedish University of Agricultural Sciences, Alnarp, Sweden; [3]Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, Tennessee; [4]Department of Structural Biology and Bioinformatics, CSIR Indian Institute of Chemical Biology)

*Phytophthora colocasiae* is an oomycete that causes leaf blight and corm rot of taro (*Colocasia esculenta*). Taro is a staple crop in many tropical countries and the emergence of *P. colocasiae* as a devastating pathogen is a matter of serious concern for food security in the region. *P. colocasiae* strain 7290 was isolated from a diseased taro plant in Vietnam in 2010 and is primarily triploid. We sequenced paired-end and mate-pair libraries of *P. colocasiae* 7290 by Illumina Hiseq and assembled the genome by ALLPATHS-LG. The draft genome of *P. colocasiae* 7290 contains 738 scaffolds with a length of 57.6 Mb, N50 of 171,568, where the longest scaffold is 738,496 bp in length. A total of 19,984 genes were predicted from the genome. Annotation of the genome and differential gene expression analysis of taro-*P. colocasiae* interactions has identified several putative carbohydrate-active enzymes, peptidases, secretory proteins (1,782) that include cytoplasmic effectors such as RxLR (337) and CRN members (203), as well as novel effectors all of which may be candidate genes for pathogenicity. The genome and transcriptome analysis of *P. colocasiae* is a first step towards determining pathogenicity factors so that effective pest management strategies can be adopted to control the disease. A detailed comparison of 7290 with isolates of *P. colocasiae* collected from other hosts will allow us understand the evolution of pathogenicity in this unique tropical species.

## Mutations in oxysterol binding protein-related protein conferring oxathiapiprolin resistance confirmed using CRISPR/Cas9 in

## *Phytophthora capsici* and *P. sojae*

Jianqiang Miao[1], Weizhen Wang[1], Yuan Fang[1], Qin Peng[1], Brett M. Tyler[2], Xili Liu[1]
([1]Department of Plant Pathology, College of Plant Protection, China Agricultural University, Beijing, China 100193; [2]Center for Genome Research and Biocomputing, Oregon State University, Corvallis, OR, USA 97331)

Oxathiapiprolin is a novel fungicide that was recently registered in a number of countries to control plant-pathogenic oomycetes. In our previous study, point mutations G770V and G839W in Oxysterol binding protein-Related Protein-1 (ORP1) were detected in oxathiapiprolin-resistant *P. capsici* mutants. In the current study, the CRISPR/Cas9 system was used to verify the effects of these two point mutations on characteristics of *P. capsici*. Transformants containing heterozygous G770V and G839W mutations in PcORP1 showed high levels of oxathiapiprolin resistance. Otherwise, the G770V transformants showed similar overall phenotypes to wild type isolate BYA5, including sporangia and zoospore production, cyst germination and pathogenicity. However, two independent transformants with heterozygous G839W mutations in PcORP1 could not produce sporangia. Three transformants with an unexpected point mutation in PcORP1, ΔN837, were obtained that showed high oxathiapiprolin resistance, but their virulence was slightly reduced. The same deletion, ΔN837, was confirmed to confer oxathiapiprolin resistance in *P. sojae* by using CRISPR/Cas9. These homozygous *P. sojae* mutants showed strongly reduced virulence. The *PsORP1*-ΔN837 gene with the original promoter was also transferred ectopically into the lab strain, P6497; positive transformants showed oxathiapiprolin resistance and an otherwise similar phenotype, including virulence, compared with wild type P6497. These results will improve our understanding of oxathiapiprolin resistance in *Phytophthora* species, and will be useful for the development of novel OSBPI fungicides. *PsORP1*-ΔN837 will be useful as a new transformation selection marker for *Phytophthora* species.

## Detection of multiple oomycetes in metagenomic data by Using E-probe Detection of Nucleic Analysis (EDNA)

Maria Fernanda Proaño, Andrés Espíndola, Carla Garzon
(Department of Entomology and Plant Pathology, Oklahoma State University, Stillwater, OK 74078)

Species identification of plant pathogenic oomycetes based on morphology is challenging. Hence, time demanding isolation and ITS sequencing for molecular identification are routinely conducted for diagnostic purposes. Metagenomic diagnostic methods allow identification of multiple pathogens from a single sample without isolation of pure cultures or specific gene-targeted sequencing. E-probe Diagnostic Nucleic acid Analysis (EDNA), a novel method that couples next-generation sequencing and bioinformatics, was used to detect a variety of plant pathogenic oomycetes in metagenomic data. Short pathogen-associated sequences (E-probes), with a range of 60-120 nucleotides, were designed and tested on metadata containing the genomes of a known host and pathogenic oomycetes. E-probes were designed for each pathogen by comparing its genome to the genome of the closest relative available and identifying unique species-specific sequences. To avoid false positives, E-probes with similarity to non-target sequences in NCBI's Nucleotide database were removed. Mock sequencing databases (MSDs) were created using MetaSim. Resulting databases contained 10'000,000 reads at different titers of pathogen abundances. A metasample was considered positive for the presence of a pathogen when a significant number of e-probes were found in a MSD. EDNA detected the target pathogens in metadata with high accuracy, thus, it is a powerful tool for detection of oomycetes from metasamples.

## EumicrobeDBLiteV11: a lightweight genomic resource and analytic platform for draft oomycete genomes

Arijit Panda[1], Diya Sen[1], Arup Ghosh[1], Akash Gupta[1], Mathu Malar C[1], Gyan Prakash Mishra[1], Deeksha Singh[1], Wenwu ye[2,3], Brett M. Tyler[2,*], Sucheta Tripathy[1,*]

([1]Computational Genomics Laboratory, Structural Biology and Bioinformatics Division, CSIR–Indian Institute of Chemical Biology, 4, Raja S.C. Mullick Road, Jadavpur, Kolkata 700032, India; [2]Center for Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR 97331-7303, USA; [3]Department of Plant Pathology, Nanjing Agricultural University, Nanjing 210095, China)

EumicrobedbliteV11 is an updated version of V10. We have added 5 new genomes including *Phytophthora plurivora* and *Aphanomyces invadans* and several versions of *P. ramorum* isolates. It analyses the data before uploading, and the analysis includes signal peptide prediction, prediction of transmembrane helices in proteins, InterPro, proprotein convertase prediction, prediction of sub-cellular localisation and so on. The genomic comparison is computed by running all to all comparison using LastZ. The data visualisation part has five main components namely genome browser page, gene details page, synteny page, query page and toolkit page. We have integrated several EMBOSS tools in the gene details page like PLOTORF, remap, revseq, cai and so on for easy on click analysis. We have also integrated Genome Synteny Viewer(GSV) tool to understand the genomic rearrangements in the synteny page. One can also upload their Genome Feature File(GFF) for quick view of genome comparison which comes with the browser page. It presently has 31 publicly oomycetes genomes and ten expressed sequence tag (EST) datasets of oomycete organisms. The database resource is available at www.eumicrobedb.org.

## Diploid genome assembly works better when Pacbio long reads are supplemented with Illumina reads: *P. ramorum* assembly a

## case study

Mathu Malar C[1,5#], Jennifer Yuzon[2,3#], Takao Kasuga[2,3*], Brett M.Tyler[4], Sucheta Tripathy[1,5*]
([1]Computational Genomics Lab, Structural Biology and Bioinformatics Division, CSIR Indian Institute of Chemical Biology, Kolkata, India-700032; [2]Department of Plant Pathology, University of California, Davis, California; [3]USDA-ARS, Davis, California; [4]Center for Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon 97331-7303; [5]Academy of Scientific and Innovative Research (AcSIR), Ghaziabad, India)
[#]: Equal contributors
Email: Sucheta Tripathy: tsucheta@iicb.res.in/tsucheta@gmail.com
[*]To whom correspondence should be addressed: tkasuga@ucdavis.edu or tsucheta@iicb.res.in

Sequencing technologies have been improving fast, but there are challenges in producing better quality assembly for diploid genomes. Several assembly strategies were used for two *P. ramorum* isolates, ND886 from ornamental Camellia and Pr102 from coast live oak. The ND886 genome was sequenced for the first time using Pacbio reads using P6-C4 chemistry. The genome was assembled using the FALCON long read assembler resulting in 302 primary contigs, 9 unplaced contigs, 397 alternate contigs (78.4 Mb genome). Haplotype Phasing was performed on Primary contigs with Illumina and Pacbio reads using WhatsHap. Three Illumina libraries were used to call genotypes and identify consensus genotypes. We found 222,892 phased variants, 20 Crinklers, 485 RXLR's with many new paralogs that were not identified before including 24 new RXLR's in the phased assembly. Our phased haplotypes were able to confirm the "two-speed genome" organization. The genome assembly of isolate Pr102 posed an even greater challenge. Pr102 was sequenced using PacBio P5-C3 chemistry. Several approaches were done to obtain the final assembly for Pr102. Of the several assembly strategies, the best approach was using the MASURCA assembler which combined Pacbio, Illumina and Sanger reads to produce a hybrid assembly. To resolve the base errors in the assembly, we performed polishing with Illumina and Sanger reads from Pr102. Our final assembly contains 1512 scaffolds (70 Mb), N50 value of 186608, and 6752 gaps (12 Mb in V1 assembly). We conclude that Pacbio long reads, when supplemented with Illumina short reads, produce reliable assemblies of diploid genomes.

**Thursday**

| **April 12th** | | |
|---|---|---|
| **8:30 - 9:50** | **Host Interactions and Resistance Mechanisms II** | **Chair:**<br>**Rebecca McDougal**<br>**and**<br>**Qin He** |
| 8:30 - 8:45 | Synergistic anti-oomycete effect of melatonin with a biofungicide against oomycetic blank shank disease | Maozhi Ren |
| 8:50 - 9:05 | *Phytophthora* utilizes an RxLR effector to hijack the host ER stress as part of their infection strategy | Maofeng Jing |
| 9:10 - 9:25 | A conserved host target of oomycete Avr3a family effectors positively regulates plant resistance to *Phytophthora* | Ruirui Feng |
| 9:30 - 9:45 | NbMORF8 encoding protein localized in mitochondria and chloroplasts negatively regulates plant immunity to *Phytophthora* pathogens | Yang Yang |

## Synergistic anti-oomycete effect of melatonin with a biofungicide against oomycetic blank shank disease

Shumin Zhang[1], Sen Liu[2], Jiankui Zhang[2], Li Feng, Maozhi Ren[1]
([1]School of Life Sciences, Chongqing University, Chongqing 401331, China; [2]College of Agronomy and Biotechnology, Southwest University, Chongqing 400700, China)

Human health, food safety and agriculture have been threatened by oomycetic diseases caused by notorious pathogenic oomycetes. Chemical oomyceticides are the main approaches in control of pathogenic oomycetes. However, the overused chemical oomyceticides have resulted in serious environmental pollution and drug resistance. The eco-friendly bio-oomyceticides are required for sustainable development through screening synergistic drug combinations. In this study, *Phytophthora nicotianae,* as one of the most destructive oomycetic diseases in agriculture, was used as a model system to screen the novel bio-oomyceticides based on drug combination. The results showed that treatment of melatonin or ethylicin alone displayed similar phenotypes such as the inhibition of the hyphal growth, reduction of the cell viability and suppression of the virulence of *P. nicotianae*. Importantly, melatonin and ethylicin shared the same targets of interfering with the amino acid metabolism, overexpressing apoptosis-inducing factor and dysregulating the virulence-related genes. Furthermore, strong synergism against *P. nicotianae* was induced by combining melatonin with ethylicin. Under treatment of the combination of melatonin and ethylicin, the expression of genes associated with amino acid, the apoptosis-inducing factor, and the virulence-related genes were much more significantly dysregulated than that of single drug treatment. Thus, the tobacco black shank caused by *P. nicotianae* can be successfully controlled by using the combination of melatonin and ethylicin. These observations suggest that the synergistic effect based on the combination of melatonin and ethylicin is an eco-friendly alternative for the control of the destructive oomycetic diseases.

## ***Phytophthora*** **utilizes an RxLR effector to hijack the host ER stress as part of their infection strategy**

Maofeng Jing[1,2], Yurong Li[3,4], Haonan Wang[1,2], Kun Yang[1,2], Yang Zhou[1,2], Gan Ai[1,2], Bo Yang[1,2], Tian Bai[1,2], Baodian Guo[1,2], Martin B. Dickman[3,4], Yuanchao Wang[1,2], Daolong Dou[1,2]
([1]Department of Plant Pathology, Nanjing Agricultural University, Nanjing 210095, China; [2]Key Laboratory of Integrated Management of Crop Diseases and Pests, Nanjing 210095, China; [3]Institute for Plant Genomics and Biotechnology, Texas A&M University, College Station, TX 77843, USA; [4]Department of Plant Pathology and Microbiology, Texas A&M University, College Station, TX 77843, USA)

In order to promote colonization, *Phytophthora* pathogens secrete an array of specific effector proteins to manipulate host innate immunity. *Phytophthora* uses an essential effector Avh262 to stabilize Binding immunoglobulin Protein (BiP) in the ER, which acts as a negative regulators of plant resistance to *Phytophthora* and ER stress-triggered cell death, resulting in attenuated plant defense responses. However, little is known about how *Phytophthora* hijacks the host BiP to regulate the ER machinery for successful infection. In this work, we show that Avh262 interacts with the binding protein of BiP, BAG7, which is an ER-localized co-chaperone that helps maintain for the maintenance of the unfolded protein response and ER stress. The translocation of BAG7 from the ER to nucleus is required for activating the downstream pathways. Here we show that BAG7 negatively regulates plant resistance to *Phytophthora* in the ER by attenuating the ER stress, but a positive role in the nucleus via upregulating the defense genes. We also found the crosstalk between PTI and ER stress pathway, which PAMPs trigger the translocation and cleavage of BAG7 from the ER to nucleus for upregulating the ER stress-associated defense genes. However, this processing can be prevented by the *Phytophthora* effector Avh262-mediated accumulation of BiP. In conclusion, during infection, *Phytophthora* accumulates host BiP to retain BAG7 in the ER, prevents cleavage and translocation of BAG7, then suppresses ER stress-triggered cell death and defense responses.

## A conserved host target of oomycete Avr3a family effectors positively regulates plant resistance to *Phytophthora*

Ruirui Feng[1], Tingting Li[1], Guangjin Fan[2], Yu Du[2], Yuling Meng[2], Weixing Shan[1]

([1]State Key Laboratory of Crop Stress Biology in Arid Areas and College of Agronomy, Northwest A&F University, Yangling, China 712100; [2]State Key Laboratory of Crop Stress Biology in Arid Areas and College of Plant Protection, Northwest A&F University, Yangling, China 712100)

Oomycete pathogens have a major ecological and economic impact which threaten natural and managed ecosystems. Oomycetes secrete an array of effector proteins to modulate host cell defense and to enable successful plant infection. However, the underlying mechanisms remain largely unknown. We are interested in the conserved host targets of *Phytophthora* effectors. In this study, we showed that multiple Avr3a family effectors interacted with RIP5 (RXLR-effector Interacting Protein 5) as shown by yeast two-hybrid, bimolecular fluorescence complementation and co-immunoprecipitation. In *Arabidopsis*, *RIP5* has been reported in defense response to bacterial pathogen infection. To investigate its role in plant defense to *Phytophthora* infection, we overexpressed *RIP5* in *Arabidopsis* and performed inoculation assays which showed that overexpression of *RIP5* enhanced host plant resistance to *P. capsici*. Consistently, silencing of the *RIP5* homolog in *N. benthamiana* decreased host resistance to *P. capsici* and *P. infestans*. Cell death assays showed that *RIP1* promoted INF1-triggered cell death. In conclusion, our results show that RIP5 is a conserved target of Avr3a family effectors and positively regulates host defense.

## ***NbMORF8*** **encoding protein localized in mitochondria and chloroplasts negatively regulates plant immunity to** ***Phytophthora***

## pathogens

Yang Yang[1], Guangjin Fan[2], Qujiang Wen[2], Ran Zhou[2], Weixing Shan[1]
([1]State Key Laboratory of Crop Stress Biology in Arid Areas and College of Agronomy, Northwest A&F University, Yangling, China 712100; [2]State Key Laboratory of Crop Stress Biology in Arid Areas and College of Plant Protection, Northwest A&F University, Yangling, China 712100)

In the process of infecting plants, *Phytophthora* pathogens secrete a large number of effectors into the host intercellular space or cells. Screening for the candidate effector targets and identifying host factors that have vital roles in plant immunity are useful approaches to dissect molecular mechanisms underlying plant susceptibility to *Phytophthora* infection. In this study, we employed VIGS (Virus Induced Gene Silencing) to examine the function of the candidate proteins targeted by the Avr3a family effector *Phytophthora sojae* effector PsAvr1b, obtained in a previous research by mass spectrometry analysis. This led to the identification of NbMORF8, a MORF (Multiple organellar RNA editing factor) family protein involved in C to U RNA editing in mitochondria and chloroplasts, which was shown to play dual roles in plant immunity, promoting plant infections of *P. parasitica* and *P. infestans* by negative regulation of PR1 expression as a susceptibility gene, and mediating specific HR immune response induced by specific recognition of effector by R protein. In addition, *NbMORF8* was essential for the regulation of plant growth and development. Further analyses showed that the *NbMORF8* encoded protein was located in both mitochondria and chloroplasts, and such subcellular location was crucial for its function.

| **Thursday**<br><br>**April 12th** | | |
|---|---|---|
| 10:20 - 11:40 | **Effectors III** | **Chair:**<br><br>**Jiang Lu**<br><br>**and**<br><br>**Biao Gu** |
| 10:20 - 10:35 | The evolutionarily conserved *Phytophthora* effector RxLR24 interferes with the secretion of antimicrobial compounds | Iga Tomczynska |
| 10:40 - 10:55 | Structure and function analysis of effector PcRxLR145 of *Phytophthora capsici* | Liying Wang |
| 11:00 - 11:15 | The *Phytophthora sojae* essential effector Avh238 targets soybean GmACSs to suppress ethylene biosynthesis and promote infection | Bo Yang |
| 11:20- 11:35 | Host mRNA methylation pathway, a new target for *Phytophthora* effector proteins? | Jie Huang |

# The evolutionarily conserved *Phytophthora* effector RxLR24 interferes with the secretion of antimicrobial compounds

Iga Tomczynska, Michael Stumpe, Felix Mauch
Department of Biology, University of Fribourg, Chemin du Musée 10, 1700 Fribourg, Switzerland

The cell surface and the apoplast are the first sites of the contact between pathogen and plant. Upon attack, host cell secretes antimicrobial compounds to the apoplast in order to inhibit pathogen growth and to restrict the spread of the disease. To compromise the plant immunity reaction, pathogens secrete effectors to change the plant metabolism and sustain its own growth. Although the precise function of most oomycetes effectors remains undiscovered, it is not surprising that some of them have evolved to interfere with the plant secretory pathway.

Here, we report the functional characterization of the evolutionary conserved *Phytophthora* RxLR24 effector. Homologous RxLR24 versions from *Phytophthora brassicae* and *P. infestans* both interact in planta with small GTPases of their hosts, Arabidopsis and potato, respectively. The function of proteins targeted by RxLR24 is the regulation of vesicle trafficking between trans-Golgi network and the plasma membrane. Consistent with the location and the role of the targeted proteins, RxLR24 localizes to the plant plasma membrane and to mobile vesicle-like structures. We demonstrate that the interaction between RxLR24 and its targets undermines transport of the antimicrobial proteins PR-1 and PDF1.2 to the plant apoplastic space. Moreover, RxLR24 expressed directly in Arabidopsis plants reduces the disease resistance against *P. brassicae*.

# Structure and function analysis of effector PcRxLR145 of *Phytophthora capsici*

Liying Wang[1,3], Li Zhao[1,3], Jing Li[1,3], Yujiao Wang[1,3], Li Zhang[1,3], Tao Liang[1,3], Peng Ding[1,3], Tongtong Zhu[1,3], Chunyuan Zhu[2,3], Xiuguo Zhang[1,3]

([1]College of Plant Protection; [2]College of Life Sciences and [3]Shandong Provincial Key Laboratory for Biology of Vegetable Diseases and insect, Shandong Agricultural University, Tai'an, China)

*Phytophthora capsici* L. is an important phytopathogenic oomycete that causes enormous loss to the production of vegetables such as pepper each year. The RxLR effectors secreted by oomycete pathogens can be identified by disease-resistant protein of the host to elicit hypersensitive response and also inhibit defense response of the plant. We cloned a RxLR effector named PcRxLR145 from *P. capsici* SD33 strain and got the crystal structure of PcRxLR145. The structure of its show that the N-terminal RxLR motif has no clear electron density and the C-terminus consists of five WY-domains (W1-W5). Every WY-domain comprising 4 α-helices and linked with each other by one "linker" helices. Then, the PcRxLR145 could induce HR response in *N. benthamiana* and inhibited cell death caused by BAX, INF1, and CRN4. Verifying the function of truncations, the first three domains, W1-3, are key region for inhibiting cell death caused by BAX, INF1, and CRN4. The W1-3 inhibitory effect of *P. capsici* zoospores infection is stronger than that of PcRxLR145. Also, we have proved that PcRxLR145 interacted with acetaldehyde dehydrogenase by yeast two hybrid, co-immunoprecipitation and bimolecular fluorescence complementation. We have also successfully obtained PcRxLR145 transgenic *Arabidopsis thaliana* by floral-dip transformation. This result revealed that PcRxLR145 suppressed the growth of roots and leaf in transgenic plants. However, all these results still need further genetic data.

## The *Phytophthora sojae* essential effector Avh238 targets soybean GmACSs to suppress ethylene biosynthesis and promote infection

Bo Yang, Baodian Guo, Maofeng Jing, Yuyin Wang, Hao Zhou, Yufei Li, Haonan Wang, Yan Wang, Wenwu Ye, Suomeng Dong, Yuanchao Wang*
(Department of Plant Pathology, Nanjing Agricultural University, Nanjing 210095, China)

*Phytophthora* pathogens secrete an arsenal of effectors to manipulate host innate immunity and thus facilitate infection. However, the molecular basis of their functionalities remains largely enigmatic. Here we show that the pathogen *Phytophthora sojae* uses an essential effector Avh238 to target the soybean 1-aminocyclopropane-1-carboxylate synthase (ACS) isoforms. By destabilizing GmACSs, Avh238 suppresses ACS-participated ethylene (ET) biosynthesis and facilitates *Phytophthora* infection. Silencing of GmACSs or inhibition of ET signaling increases susceptibility to *P. sojae* infection, supporting a role for GmACSs and ET in anti-*P. sojae* immunity. Moreover, wild-type *P. sojae* but not the Avh238-disrupted mutants, inhibits ET induction and promotes *P. sojae* multiplication in soybean. This work highlights the ET biosynthesis pathway as an anti-*P. sojae* defense mechanism and a direct effector target.

# Host mRNA methylation pathway, a new target for *Phytophthora* effector proteins?

Jie Huang[1], Xinyu Lu[1], Ling Chen[1], Qian Peng[1], Brett M. Tyler[2], Daolong Dou[1], Guifang Jia[3], Yuanchao Wang[1], Suomeng Dong[1]

([1]Department of Plant Pathology, Nanjing Agriculture University, Nanjing, China 210095; [2]Center for Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon 97331, USA; [3]Synthetic and Functional Biomolecules Center, Beijing National Laboratory for Molecular Sciences, Key Laboratory of Bioorganic Chemistry and Molecular Engineering of Ministry of Education, College of Chemistry and Molecular Engineering, Peking University, Beijing 100871, China)

Increasing evidences suggested that epigenetic modulation is an appealing layer of plant-pathogen interactions. $N^6$-methyladenosine ($m^6A$) is a reversible mRNA nucleotide epitranscriptomic modification and is found to participate versatile biological process. Unlike $m^6A$ in mammal research, $m^6A$ modifications have been poorly reported in plant immunity. Here, we use *Phytophthora infestans* and tomato pathosystem to study the role of $m^6A$ in plant-pathogen interaction. We show that tomato SlFIP37, the orthologs of *Arabidopsis thaliana* $m^6A$ methylation complex protein FIP37, is a positive regulator of plant immunity against *P. infestans*. To dissect how *Phytophthora* manipulate host $m^6A$ modification process, we have screened an effector library to identify potential interactors of SlFIP37 and we identified some *P. infestans* effectors that bind to SlFIP37. Whether these effectors affect host $m^6A$ level and what are the modes of action of effectors on $m^6A$ methylation machinery remain further investigations.

# ABSTRACTS OF POSTER PRESENTATIONS

## The virulence function of Phytophthora infestans RxLR effector Pi04314/RD24 and screens for resistances that recognise it

Xiaodan Wang[1,2], Petra Boevink[2], Hazel McLellan[2], Qin He[2], Shaohui Sun[1], Zhendong Tian[3], Vivianne Vleeshouwers[4], Ingo Hein[2], Paul R.J. Birch[2*]

([1]Virus-free Seedling Research Institute of Heilongjiang Academy of Agricultural Sciences, Harbin Heilongjiang, China,150086; [2]Division of Plant Sciences, College of Life Sciences, University of Dundee at The James Hutton Institute, Invergowrie, Dundee, DD2 5DA, UK; [3]Key Laboratory of Horticultural Plant Biology (HAU), Ministry of Education, Huazhong Agricultural University, Wuhan, Hubei, China, 430070; [4]Wageningen UR Plant Breeding, Wageningen University, Wageningen, The Netherlands, P.O. Box 386, 6700 AJ)

The oomycete pathogen *Phytophthorainfestans* is responsible for causing late blight disease in *Solanaceous* plants. The pathogen secretes essential effectors into plant cells that target critical host cellular mechanisms to promote disease. Recently, we reported that an RXLR effector, Pi04314/RD24, interacts with protein phosphatase 1c (PP1c) isoforms in the host nucleus to enhance leaf colonization by *P. infestans*. Data and research demonstrate that effector Pi04314 targets PP1c isoforms as host susceptibility factors (S factors) to promote virulence and thus cause late blight disease. PP1c isoforms are required for late blight infection and Pi04314 utilises their activities presumably to dephosphorylate and inactivate host immune regulators. In addition, we will report our recent progress on identifying novel resistance proteins that recognise this effector in wild potatoes, and the mechanistic relationship between such recognition and the host target-PP1c. We will investigate whether effector Pi04314 as a tool can be used to explore new host resistance genes to accelerate disease resistance breeding against late blight in future work.

# A host-specific RxLR effector of *Phytophthora palmivora* contributes to virulence on cacao

Zhiying Cai[1,2], Natasha Navet[1], Janice Uchida[1], and Miaoying Tian[1]
([1]Department of Plant and Environmental Protection Sciences, University of Hawaii at Manoa, Honolulu, HI 96822, USA; [2]Department of Plant Protection, Yunnan Institute of Tropical Crops, Yunnan, Jinghong 666100, China)

*Phytophthora palmivora* is an oomycete plant pathogen with a wide range of hosts, including many economically important ones such as papaya and cacao. Host specificity of *P. palmivora* is largely unknown, but exists. For example, the cacao isolate does not infect papaya, and vice versa. Similar to other oomycete pathogens, the *P. palmivora* genome encodes a large set of effector proteins, including the RxLR family, which are believed to play key roles in pathogenicity. From the published genome and transcriptome data, we identified an RxLR effector designated PpalRxLR1 as highly induced during infection of cacao by a cacao isolate, but absent in the papaya isolate. PCR and RT-qPCR confirmed these findings. Three near identical copies were found in the genome assembly, with each encoding a protein with a putative signal peptide and a canonical RxLR-EER motif. A single guide RNA targeting all three was used to generate mutants via CRISPR/Cas9 gene editing. We obtained three single zoospore derived mutants with PpalRxLR1 either completely or partially mutated. The mutants exhibited reduced pathogenicity on cacao leaves compared with the wild-type strain. PpalRxLR1a was predicted to have a nuclear localization signal using Localizer, and its nuclear localization was confirmed by transiently expressing the mature protein of PpalRxLR1a fused with DeRed in *Nicotiana benthamiana*. Altogether, these data suggest that PpalRxLR1 mediates *P. palmivora* pathogenicity likely by manipulating host gene expression. Its presence in the cacao but not papaya isolate suggests that it may play a determinant role in host jump of *P. palmivora*.

# Changes in histone H3 Lys27 tri-methylation at the *Avr1b* locus causes loss of avirulence in *Phytophthora sojae*

Liyuan Wang[1], Han Chen[1], Haidong Shu[1], JiangJiang Li[1], Brett M. Tyler[2], Yuanchao Wang[1], Suomeng Dong[1,*]

([1]Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China; [2]Center for Genome Research and Biocomputing, and Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR 97331, USA)

*Phytophthora* is a group of destructive plant pathogens that threatens world agricultural production. One of the well-known features of *Phytophthora* pathogens is their rapid adaptability on host plants. The interaction between soybean and its root rot pathogen *Phytophthora sojae* exhibits gene-for-gene interactions. Earlier studies demonstrated that genetic mutations and small RNA mediated gene silencing at avirulence gene loci enable *P. sojae* strains to gain virulence on resistant soybeans. However, whether other epigenetic variations are employed by *P. sojae* to evade host immune surveillance remain unknown. It has been observed that the *Avr1b* locus is silenced in two virulent strains without any sequence change compared to unsilenced strains, suggesting this locus may be subject to epigenetic regulation. We found significant accumulation of histone H3 Lys27 tri-methylation (H3K27me3) at the *Avr1b* loci of the silencing reference strain from our Chromatin Immunoprecipitation-Sequencing (ChIP-seq) assays. To further investigate this interesting observation, we selected one avirulent strain and a virulent strain that possess identical *Avr1b* genotype but exhibit distinct gene transcript levels. We observed hypermethylation of H3K27me3 at the *Avr1b* locus in the virulent strain but hypomethylation in the avirulent strain through ChIP-qPCR. Furthermore, we generated H3K27me3 methyltransferase co-factor knockout mutants by CRISPR/Cas9 to test our hypothesis. Both ChIP-seq and RNA-seq data of the mutants are consistent with our hypothesis that H3K27me3 plays a role in regulating *Avr1b* expression. Here, we will report our recent progress on the mechanistic analysis of H3K27me3 in regulating *Avr1b* gene silencing in *P. sojae*.

# G-protein is essential for sporangium formation by a serine/threonine protein kinase in *Phytophthora sojae*

Min Qiu[1], Yaning Li[1], Baiyu Zhang[1], Xin Zhang[1], Yan Wang[1], Wenwu Ye[1], Brett M. Tyler[2], Yuanchao Wang[1*]

([1]Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China; [2]Center for Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR 97331, USA)

Sporangia reproduction and zoospore motility are essential in the early stages of oomycete pathogen infection. Previously, we identified a G-protein α subunit encoded by *PsGPA1* which regulates the chemotaxis and pathogenicity of *Phytophthora sojae*. In the present study, we identified PsYPK2, a serine/threonine protein kinase, which could interact with PsGPA1 *in vitro*. Analysis of the obtained CRISPR-knockout and complement strains revealed that PsYPK2 contributed to sporangia production and virulence of *P. sojae*. The mutants showed significantly decreased growth rate and lost the ability to produce oospore. Interestingly, the *PsGPA1*-overexpressing strains exhibited decreased sporangia number and increased growth rate of mycelia, which resembled to the phenotypes of *PsYPK2*-knockout strains. These results indicate that PsGPA1 plays opposite function of PsYPK2 during sporangium formation. Based on these data, we propose the following two probable models for G-protein dependent regulation of sporangium formation in *Phytophthora sojae*. During sporangium formation, active Gα protein acts as negative regulator of signaling while free Gα directly inhibit PsYPK2 function by their interaction. However, according to the phenotype of Gβ in *Phytophthora infestans*, the other possible model is that higher expression of Gα protein sequesters the available pool of Gβ in trimeric conformation, resulting in decreased sporangia numbers. To be noted, an independent role of free Gβ proteins in interacting with PsYPK2 to regulate sporangium formation cannot be ruled out at this stage.

# A *Phytophthora* effector inhibits secretion of a plant aspartic protease to promote infection

Baodian Guo[a,b,1], Haonan Wang[a,1], Bo Yang[a], Maofeng Jing[a], Haiyang Li[a], wenjing jiang[a], Qinli Hu[b], Fangfang Wang[b], Yan Wang[a], Wenwu Ye[a], Suomeng Dong[a], Weiman Xing[b*], Yuanchao Wang[a*]
([a]Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China; [b]Shanghai Center for Plant Stress Biology, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences, Shanghai 201602, China)

Plants secrete defense molecules into extracellular space (apoplast) to combat attacking microbes. Until now, how successful pathogens subverting plant apoplast immunity remains poorly known. Here we show that a virulence effector PsAvh240, secreted by soybean pathogen *Phytophthora sojae*, forms a dimer in plant plasma membrane. PsAvh240 significantly promotes *Phytophthora sojae* infection by targeting a soybean aspartic protease GmAP1, an apoplastic protein required for soybean resistance against *Phytophthora sojae*. PsAvh240 inhibits the secretion of GmAP1 in plant plasma membrane and this inhibition depends on the homo-dimerization of PsAvh240. Overall, our work highlights an example on how pathogen effectors interfering with plant apoplastic immunity during pathogen counter-defense.

# A nucleus-localized effector of *Phytophthora sojae* hijacks a host acetyltransferase to enhance plant susceptibility

Haiyang Li, Haonan Wang, Maofeng Jing, Jinyi Zhu, Baodian Guo, Yang Wang, Yachun Lin, Han Chen, Liang Kong, Zhenchuan Ma, Yan Wang, Wenwu Ye, Suomeng Dong, Yuanchao Wang*
(Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China)

Filamentous fungi and oomycete pathogens secrete various intracellular effector proteins to manipulate host immunity during infection. Identifying the plant targets of these effectors expands our understanding of plant immunity. We previously showed that PsAvh52, an early-induced RxLR effector secreted from the soybean root rot pathogen *Phytophthora sojae*, can suppress pathogen-associated molecular patterns (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI) in plants. In the present study, we found that PsAvh52 is essential for full virulence of *P. sojae* on soybean and that PsAvh52 interacts with a soybean acetyltransferase, namely GmTAP1. PsAvh52 recruits GmTAP1 to the nucleus where it can acetylate histones H2A and H3, can promote expression of plant disease susceptibility genes, and can promote susceptibility to *P. sojae* infection. In the absence of PsAvh52, GmTAP1 remains confined to the cytoplasm and does not modify plant susceptibility. These results demonstrate that GmTAP1 is a susceptibility factor that is hijacked by PsAvh52 in order to manipulate epigenetic modifications that enhance *P. sojae* colonization in soybean.

# A *Phytophthora sojae* effector promotes infection by suppressing secretion of an apoplast effector inhibitor GmGIP1

Haonan Wang, Baodian Guo, Haiyang Li, Bo Yang, Yan Wang, Wenwu Ye, Suomeng Dong, Yuanchao Wang*

(Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China)

The *Phytophthora sojae* Glycoside Hydrolase family 12 protein PsXEG1 can be recognized as a pathogen-associated molecular pattern (PAMP) by soybean and many solanaceous plants. PsXEG1 also acts as an important virulence factor for *Phytophthora sojae*, depending on its xyloglucanase and β-glucanase activity. However, soybean produces Glucanase Inhibitor Proteins like GmGIP1 to the apoplast to inhibit the β-glucanase activity of PsXEG1 in order to decrease the virulence function of PsXEG1. In addition, overexpression of GmGIP1 restricts *P. sojae* infection, indicating that GmGIP1 is an important resistance component against *Phytophthora*. As counter-defense, *P. sojae* can secrete a PsXEG1-like apoplast effector PsXLP1 as a decoy to protect PsXEG1 against the inhibition of GmGIP1. In addition, we recently identified two cytoplasmic effectors that interact with GmGIP1 and suppress the secretion of GmGIP1. This finding revealed a new strategy employed by *Phytophthora* pathogens to counter host defense.

# *Phytophthora sojae* avirulence effector Avr1d interacts with *Glycine max* U-box Ubiquitin E3 ligases to promote infection

Yachun Lin, Weixiao Yin, Yuanyuan Shao, Yan Wang, Wenwu Ye, Suomeng Dong, Yuanchao Wang*
(College of Plant Protection, Nanjing Agriculture University, Nanjing 210095, China)

*Phytophthora* pathogens secrete a repertoire of effectors to host cell to manipulate host immunity and benefit the pathogen infection. Until now, the functional mechanisms of many effectors remain uncovered. Avr1d is a *Phytophthora sojae*-secreted RxLR effector, which can promote *P. sojae* infection when expressed in soybean hairy-roots. Our study showed that Avr1d directly interacted with an ubiquitin E3 ligase Gm860 which has a U-box domain in the middle and ARM-repeat at the C-terminus. Avr1d could block the E3 ligase activity of Gm860 and stabilized Gm860 to a higher protein level. Silencing of Gm860 in soybean hairy-roots decreased the infection of *P. sojae* indicating that Gm860 negatively regulated plant immunity to *P. sojae*. Together, our data show that Avr1d promote *P. sojae* infection in *Glycine max* by stabilizing Gm860, a negative regulator of plant immunity.

# Glycosylation is essential for PsXEG1-triggered plant immunity and *Phytophthora* virulence

Yeqiang Xia, Zhenchuan Ma, Lin Zhu, Baodian Guo, Qi Zhang, Haibin Jiang, Liang Sun, Yan Wang, Wenwu Ye, Suomeng Dong, Yuanchao Wang*
(Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China)

*Phytophthora* secretes a range of effectors into the extracellular and intracellular space of host. Previously, we found that PsXEG1, an apoplastic effector, can trigger plant immunity and contribute to *P. sojae* virulence. Here, we found PsXEG1 has two forms that are different in glycosylation and both could be secreted into the plant extracellular space. Interestingly, the glycosylation is essential for PsXEG1-triggered plant immunity and *Phytophthora* virulence. In line with this, we found that the glycosylation is also implicated in the binding of PsXEG1 to both the recognition receptor RXEG1 and the host inhibitor GmGIP1. We are currently analyzing how this modification regulates the function of PsXEG1 in these two processes.

# Identification of novel apoplast effectors from *Phytophthora sojae*

Yuanpeng Xu, Yan Wang, Zhou Yang, Baodian Guo, Yuanchao Wang*
(Department of Plant Pathology, Nanjing Agriculture University, Nanjing 210095, China)

The apoplast space is the first and vital battlefield for plant-pathogen interaction. Last decade, the RxLR effectors, which function as powerful weapons to manipulate host immunity, attracted most of attentions from the world-wide *Phytophthora* groups. Recently, the *Phytophthora sojae* apoplast effector XLP1 was identified as decoy to protect the virulence effector XEG1 against host inhibition. It represents a complicated and astonishing defense and counter-defense event happened in the apoplast space during pathogen-host interaction. To further understand the apoplast interactions, we profiled the apoplastic effectors secreted by *P. sojae* and identified 32 unknown proteins with a predict signal peptide. Transient expression assays in *Nicotiana benthamiana* leaves revealed six of these proteins could induce plant cell death. We currently functionally analyze these six candidates to figure out their roles in *Phytophthora* virulence and how these effectors being recognized by their hosts.

# An RXLR effector promotes *Phytophthora* infection by manipulating host Histone methylation

Xufang Qiu, Yan Wang, Wenwu Ye, Yuanchao Wang*
(Department of Plant Pathology, Nanjing Agriculture University, Nanjing, China 210095)

Histone methylation plays critical roles in gene regulation in animal and plant immunity. Till now, how eukaryotic pathogens manipulate host modification process remains poorly understood. For human pathogen *Legionella pneumophila,* activation of host rDNA transcription is a general bacterial virulence strategy through the secretion of type IV secretion system (TFSS) effector. Here, we found a cytoplasmic effector secreted by *Phytophthora sojae* can suppress H3K4 methylation in soybean by interfering with the formation of transcriptional activation complex. This is tightly linked to the virulence function of the effector. We are currently assaying the mechanism of the H3K4 methylation suppression activities and the virulence function of the RxLR effector.

## Metabolic adaptation of *Phytophthora* and *Pythium* to their pathogenic lifestyles.

Audrey Ah-Fong, Meenakshi Kagda, Melania Abrahamian, Howard Judelson
(Department of Microbiology and Plant Pathology, University of California, Riverside, USA)

Fundamental differences exist between plant pathogens that exhibit distinct lifestyles. That the effector complements of biotrophs and necrotrophs vary is well-documented. Differences also exist in how pathogens colonize the host. Biotrophs feed from living cells, while necrotrophs derive nutrients by macerating host tissues. Nutrient environment thus varies with pathogenic lifestyle, which we hypothesize is associated with distinct strategies for nutrient acquisition and metabolism. This premise is supported by a comparison of two oomycetes, the hemibiotroph *Phytophthora infestans* and the necrotroph *Pythium ultimum*, during potato tuber colonization. During early infection, differences in many metabolic pathways were seen between the species. For instance, we observed a significant induction of genes devoted to lipid and starch metabolism in *Py. ultimum* while amino acid and sucrose metabolism were higher in *Ph. infestans*. Variation in the use of nitrogen sources were also observed through the use of isotopic labeling. Rye-sucrose media amended with $^{15}NO_3$ showed that it is taken up in late growth stages of *Ph. infestans* while being incorporated in both early and late stages by *Py. ultimum*. The assimilation of different nutrients is likely the result of each pathogen modulating its metabolism to its microenvironment, which in the case of *Ph. infestans* is mostly limited to the apoplast. The metabolic differences could also be due to the rewiring of regulatory networks during the evolution of the two species.

## Dramatic expansion of potential virulence factors in the genome of the oomycete mosquito pathogen *Pythium guiyangense*

Danyu Shen, Chaoyang Tang, Yun Wei, Cong Wang, Jing Wang, Maofeng Jing, Ai Xia, Daolong Dou*
(Department of Plant Pathology, Nanjing Agricultural University, Nanjing, China 210095)

*Pythium guiyangense*, an oomycete pathogen, exhibits a high ability for killing mosquito vectors, and is considered as an ideal biocontrol agent of mosquito populations. However, little is known about biology and pathogenesis of *Py. guiyangense* at the molecular level. Here, we present a high-quality genome assembly of *Py. guiyangense* using a hybrid sequencing strategy. The *Py. guiyangense* genome is 110 Mb and encodes 30,973 genes. Comparison of *Py. guiyangense* with plant pathogenic oomycetes suggests that during evolution the host cellular environment has driven distinct patterns of gene expansion and loss in the genomes of plant and insect pathogens. Plant cell wall degrading enzymes are largely degenerated in *Py. guiyangense*. In contrast, *Py. guiyangense* has a full set of proteases including many expanded subtilisins, trypsins, papain that can digest mosquito cuticles and the host body. *Py. guiyangense* also secreted a large number of kazal-type protease inhibitors which specially inhibit serine proteases that play crucial roles in various biological and physiological processes of invertebrates. To regulate the complex signal transduction pathways, *Py. guiyangense* has a very large kinome, 16% of which is induced upon infection. The CRN effectors are specifically evolved in the *Py. guiyangense* genome, and several CRN effectors are verified to be virulent to mosquitoes by experiments. Moreover, Elicitin and NLP effectors are expanded in *Py. guiyangense* genome, and many of these genes are upregulated in the infection process. These findings add to our understanding of evolution and pathogenesis of insect pathogenic oomycetes.

# Know thy enemy: plant pathogen genomics advancing disease management

Rebecca McDougal[1], Preeti Panda[1], Simren Brar[2], Lukas Hunziker[2], Pierre-Yves Dupont[2], Melissa Guo[2], Rosie Bradshaw[2] and Nari Williams[1]
([1]Scion (New Zealand Forest Research Institute, Ltd.), Rotorua, New Zealand; [2]Massey University)

Genomics studies are contributing enormously to research and disease management applications in plant pathology. In addition to rapidly increasing our understanding of the molecular mechanisms underpinning pathogenesis and resistance, genomics greatly contributes to the development of novel markers for rapid pathogen detection and diagnosis, and offers insights into the genetics of pathogen populations on a larger scale. The importance of understanding the diversity of pathogen populations is essential to mitigation of biosecurity risks, protecting market access and for resistance screening. Effectoromics is the study of plant pathogen effector proteins using functional genomic tools to find host resistance. It is now being used in applied research, and operationally, as a rapid high-throughput method to screen for disease resistance in many types of crops around the world. While trees present many challenges for applying these types of tools, the rewards have the potential to be hugely beneficial in productive and conservation systems worldwide.

# *GmDAD1*, the conserved Defender against cell death 1 (*DAD1*) from soybean, positively regulates resistance against *Phytophthora* pathogens

Qiang Yan, Jierui Si, Daolong Dou*
(Department of Plant Pathology, Nanjing Agriculture University, Nanjing, China 210095)

The regulation of programmed cell death (PCD) pathway have been demonstrated that relevant to both abiotic and biotic stress responses in plants. Defender against cell death 1 (DAD1), was initially identified from a temperature sensitive mutant hamster tsBN7 cell line as a mammalian apoptotic suppressor and subsequent demonstrate that act is a subunit of the mammalian oligosacryltransferase. The DAD1 proteins are conserved in plants and also function as negative regulator of cell death, but the molecular mechanism and significance for *Phytophthora sojae* resistance in soybean have not been elucidated. Here, we report the characterization of *GmDAD1* gene in soybean. *GmDAD1* encodes an endoplasmic reticulum (ER)-localized protein with typical structural features of DAD1 proteins. Its expression was induced in both compatible and incompatible interactions with *P. sojae* infection. Transgenic studies in soybean hairy roots and *Nicotiana benthamiana* confirmed that *GmDAD1* plays a positive role in regulating plant resistance. The DAD1 protein catalyzes the first step of protein N-linked glycosylation, presumably involved in ER stress response. The transcription characterization of several ER stress marker genes showed that silencing *GmDAD1* in soybean hairy root caused a severe ER stress. Our findings showed that *GmDAD1* plays a critical rule in plant defense, and provide evidence linking ER stress signaling with *P. sojae* resistance.

# *Phytophthora infestans* effector promote infection by targeting inside host nucleus

Yu Du[1,3], Yalu Guo[1,3], Xiaokang Chen[1,3], Xiaojiang Zhang[1,3], Weixing Shan[1,2]
([1]State Key Laboratory of Crop Stress Biology for Arid Areas, Northwest A&F University, Yangling, Shaanxi 712100, China; [2]College of Agronomy, Northwest A&F University, Yangling, Shaanxi 712100, China; [3]College of Horticulture, Northwest A&F University, Yangling, Shaanxi 712100, China)

*Phytophthora infestans* is the causal agent of late blight disease on potato and tomato. It secretes hundreds of effectors to modulate host cellular processes to promote colonization. The well-known are the RXLR effectors that are translocated into host cells to manipulate host cell machinery. The subcellular localization of RXLR effectors are diverse, and some have specific subcellular localizations that are closely related to their virulence activities. For instants, RXLR effector AVRblb2 localizes to the host plasma membrane, targets and suppresses the secretion of C14 to promote colonization. Here, we report that the RXLR effector E47 is highly expressed at early infection stage and contains a nuclear localization signal at its C-terminus. By fusing nuclear localization/export and myristolation signals to E47, we investigated the effect of targeting this effector to different subcellular compartments on its functionalities. We showed that its nuclear localization is required for promoting *P. infestans* colonization. Further investigation of its host targets is undergoing.

# A conserved *Phytophthora parasitica* RxLR effector can promote infection and affect plant growth

Qiang Zhang[1,2], Weixing Shan[1,3]
([1]State Key Laboratory of Crop Stress Biology for Arid Areas, Northwest A & F University, Yangling, China, 712100; [2]College of Plant Protection, Northwest A & F University, Yangling, China, 712100; [3]College of Agronomy, Northwest A & F University, Yangling, China, 712100)

*Phytophthora* pathogens employ an arsenal of effectors such as the RxLR proteins to suppress plant immunity. Based on the *P. parasitica* genomes and RNA-seq data, we identified a conserved RxLR effector-*Pp18*, which was highly upregulated at early stages of infection and peaked at 3hpi. *Pp18* promoted *P. parasitica* infection when being transiently expressed in *Nicotiana benthamiana* leaves or stably expressed in *Arabidopsis thaliana*. *P. parasitica* transformants overexpressing Pp18-flag showed enhanced virulence on *N. benthamiana* leaves compared to the wild type. Pp18 was localized in vesicle-like structures. *Pp18* suppresses the HR triggered by PsojNIP, but not INF1 nor BAX. When stable expressing GFP-Pp18 in *A. thaliana*, the growth of transformants were significantly delayed compared with transformant expressing free-GFP.

# *AtRTP5* is a negative regulator of immunity in *Arabidopsis thaliana* to *Phytophthora parasitica*

Weiwei Li[1,2], Weixing Shan[1,3]
([1]State Key Laboratory of Crop Stress for Arid Areas, Northwest A & F University, Yangling, China 712100; [2]College of Plant Protection, Northwest A & F University, Yangling, China 712100; [3] College of Agronomy, Northwest A&F University, Yangling, China 712100)

Oomycetes form a distinct phylogenetic lineage of fungus-like eukaryotic microorganisms, but have a closer relationship with the Chromista in evolution. *Phytophthora* genus belongs to oomycete and causes disastrous diseases of many ornamental and food crops. *Phytophthora* pathogens encode diverse virulence factors such as RXLR effectors to enable successful infection and to establish compatible interaction. However, little is known on the mechanisms of compatible/susceptible plant interaction with *Phytophthora* pathogens. We describe the identification of the host negative regulator *AtRTP5*, which mediates susceptibility of *A. thaliana* to *P. parasitica*. The T-DNA insertion mutant *rtp5-1* showed enhanced resistance to *P. parasitica*, while *AtRTP5* over-expressing transformants were more susceptible compared to the wild type. *AtRTP5* was slightly induced by *P. parasitica* and RTP5-GFP fusion protein was accumulated in nucleus and plasma membrane. *rtp5-1* was shown to be down-regulated expression of jasmonate biosynthesis -associated genes, and up-regulated expression of salicylic acid biosynthesis -associated genes. The results showed that *AtRTP5* plays an important role in the compatible interaction between *P. parasitica-Arabidopsis*, possibly by interfering with JA and SA signal pathways.

# A New AP2/ERF Transcription Factor from *Nicotiana benthamiana* Confers Resistance to *Phytophthora parasitica*

Jing Yu[1], Chunyue Chai[2], Gan Ai[1], Yuling Jia[1], Danyu Shen[1], Tian Bai[1], Xiong Zhang[1], Daolong Dou[1*]
([1]Department of Plant Pathology, Nanjing Agriculture University, Nanjing, China 210095; [2]College of Life Science and Technology, Nanyang Normal University, Nanyang China, 473061 )

When RNA-Seq data of *Nicotiana benthamiana* upon *Phytophthora parasitica* infection was analyzed, we found that ERFs are one of the members that were strongly *Phytophthora*-induced. Ethylene responsive factors (ERFs) play important roles in plant growth, development and resistance. Among them, one member (NbERF173) containing a conserved 52-residue AP2/ERF DNA binding domain was a most highly up-regulated gene. Knock down of the gene led to significant reduction of the resistance to both *P. parasitica* and *B. cinerea* while its overexpression exhibited the enhanced resistance, suggesting that it positively regulates disease resistance. RNA-Seq analysis of NbERF173-slienced leaves under *P. parasitica* infection showed many defense-related genes were regulated, among of which, two proteinase inhibitors were highly suppressed. Furthermore, transient overexpression of these two proteinase inhibitors can promote resistance. Therefore, our study identified a novel ERF family gene that contributes to disease resistance by regulating expression of the defense-related genes.

## Screening of *Peronophythora litchii* effectors which can induce or suppress plant immunity

Guanghui Kong, Lang Wan, Wensheng Yang, Wen Li, Pinggen Xi, Zide Jiang*
(Department of Plant Pathology/Guangdong Province Key Laboratory of Microbial Signals and Disease Control, South China Agricultural University, Guangzhou 510642, China)

*Peronophythora litchii* is one of the most destructive pathogens on lychee (*Litchi chinensis*). There is no reports focus on the interaction between this pathogen and lychee. We screened the secreted proteins of *P. litchii* to identify effectors which can induce or suppress plant immunity. So far, we found 6 effectors could induce cell death and one protein containing pectin acetylesterase (PAE) motif promoted the infection of *P. capsici* on *Nicotiana benthamiana* suggesting PAE might be virulence effector of *P. litchii*. The deletion of PAE motif abolished the function of this protein on *N. benthamiana*. We also knockouted this gene using CRISPR/Cas9 system and found that deletion of this gene impaired the virulence of *P. litchii* but not growth, suggesting pectin acetylesterase is virulence effector of *P. litchii*. This study firstly reported that PAE protein is involved in the infection of plant pathogenic oomycete.

# PlMAPK10, a mitogen-activated protein kinase (MAPK) in *Peronophythora litchii*, is required for mycelial growth, sporulation, laccase activity and plant infection

Liqun Jiang, Junjian Situ, Yi Zhen Deng, Lang Wan, Dandan Xu, Yubin Chen, Pinggen Xi*, Zide Jiang*
(Guangdong Province Key Laboratory of Microbial Signals and Disease Control, South China Agricultural University, Guangzhou 510642, China)

Mitogen-activated protein kinase (MAPK) pathways are ubiquitous and evolutionarily conserved signal transduction modules directing cellular respond to a diverse array of stimuli, in the eukaryotic organisms. In this study, *PlMAPK10* was identified to encode a MAPK in *Peronophythora litchii*, the oomycete pathogen causing litchi downy blight disease. PlMAPK10, containing a specific and highly conserved dual phosphorylation lip sequence SEY (Serine-Glutamic-Tyrosine), represents a novel group of MAPKs as previously reported. Transcriptional profiling showed that PlMAPK10 expression was up-regulated in zoospore and cyst stages. To elucidate its function, the *PlMAPK10* gene was silenced by stable transformation. *PlMAPK10* silence did not impair oospore production, sporangium germination, zoospore encyst, or cyst germination but hindered hyphal growth, sporulation, pathogenicity, likely due to altering laccase activity. Over all, our results indicated that a MAPK encoded by *PlMAPK10* gene in *P. litchii* is important for pathogenic development.

## Molecular and morphological characterization of *Phytophthora* isolates from citrus roots in Texas

Veronica Ancona, Shima Chaudhary
(Citrus Center, Texas A&M University-Kingsville, Weslaco, TX 78599)

Citrus in highly susceptible to foot rot, gummosis and fruit brown rot diseases caused by *Phytophthora nicotianae* (Syn. *P. parasitica*), *P. citrophthora* and *P. palmivora*. Recently, we reported that in south Texas 97% of citrus orchards have foot rot and gummosis infections, many of them with more than 50% of trees affected. However, the *Phytophthora* species associated with these infections remain uncharacterized. Our goal was to identify and characterize the *Phytophthora* species present in the citrus rhizosphere in Texas. Eighty-nine *Phytophthora* isolates were extracted from soil and roots collected from thirty two commercial citrus orchards across south Texas. Sequence analysis of the ITS region generated using primers ITS4 and ITS6 revealed that all isolates were *P. nicotianae* with 98-100% sequence identity. Morphological differences within *P. nicotianae* isolates were found in colony growth patterns, sporangium and oogonium shape and size. Ovoid, obpyriform and spherical sporangia, with single and double papilla were observed. Both *P. nicotianae* A2 and A1 mating types were identified from isolates coming from different and same locations. *P. nicotianae* isolates also exhibited differences in sensitivity to mefenoxam (EC50 ranged from .01 to 144µg/ml), pathogenicity and virulence to several citrus and non-citrus hosts. Our results demonstrate intraspecies diversity amongst the *P. nicotianae* isolates, which may have important implications in the development of management strategies for *Phytophthora* diseases of citrus in the region.

# Modulation of host selective autophagy during *Phytophthora infestans* infection

Pooja Pandey[1], Alex leary[1], Yasin tumtas[1], Nattapong sanguankiattichai[1], Mauricio Contreras[1,2], Lyu Chen[1], Radoslaw Kowalczyk[1], Maria e. Segretin[1,2], Tolga o. Bozkurt[1]
([1]Imperial College London, Department of Life Sciences, London, UK; [2]INGEBI-CONICET, Obligado 2490, C.A.B.A., C1428ADN, Argentina)

A selective form of autophagy organized by autophagy cargo receptors contributes to immunity in plants. Autophagy cargos are enclosed in autophagosomes, double-membrane vesicles that are coated by conserved ubiquitin-like ATG8 protein either to be degraded or relocated. In plants ATG8 is expanded into multiple isoforms, possibly to mediate selective autophagy. However, we know little about how selective autophagy is regulated and contributes to immunity. Recently, we discovered that the *P. infestans* effector PexRD54 subverts host defences mediated by plant autophagy cargo receptor Joka2. PexRD54 outcompetes Joka2 for binding the solanaceous ATG8 isoform ATG8CL. To better understand the molecular mechanisms of selective autophagy in plants, we exploited PexRD54, which stimulates autophagosome formation through ATG8CL binding. We discovered that PexRD54 stimulates autophagosome formation by coupling host vesicle transport regulators to ATG8CL-coated autophagosomes. Furthermore, effector-targeted autophagosomes are delivered toward the pathogen interface, possibly to allocate cellular resources. Strikingly, we also identified two diverse plant RabGAPs with validated ATG8 binding motifs that compete with the effector and Joka2 for ATG8CL binding. Finally, our proteomics screen for Joka2 interactors in pathogen infected tissue revealed defense proteins and related signaling components. Our results implicate effector-mediated employment of host components in autophagosome biogenesis and show that effectors can serve as tools to study molecular mechanisms of selective autophagy.

# Functional Analysis of Transporter Of Putrescine And Spermidine (TOPAS1) a member of a major class of conserved transporters in oomycetes

Nilanjana Chakrabarti, and Paul F. Morris
Department of Biological Sciences, Bowling Green State University, Bowling Green OH 43403

Membrane transporters play an essential role in the acquisition, export, and sequestration of metabolites in cells. The membrane transporter PPTG_16698, Transporter of Putrescine and Spermidine (TOPAS1) came to our attention because it has the 5th highest expression of all membrane transporters expressed in the zoosporic stage of the broad host pathogen Phytophthora parasitica. TOPAS1 is one of 13 highly conserved membrane transporters in P. parasitica, and orthologs to these proteins are found in all oomycetes, although there are no homologues to these proteins in other eukaryotes. In prior work (Chibucos and Morris 2006) we showed that swimming zoospores actively acquire polyamines from the aqueous environment. Zoospores accumulate mM levels of polyamines and we hypothesize that the acquisition of high levels of polyamines from the soil environment may contribute to the virulence of encysted zoospores. We hypothesized that members of this family were involved in either the direct uptake or sequestration of polyamines in hyphae and zoospores. Heterologous expression of this gene in yeast cells did not result in direct uptake of radiolabeled putrescine and spermidine from the media. However, expression of this gene is associated with decreased toxicity to toxic levels of exogenous polyamines. In yeast, this decreased toxicity is consistent with the sequestration of polyamines from the cytosol and export via the polyamine specific transporter TPO5. In ongoing work, we are using fluorescent tagging of this, and other polyamine transporters to localize proteins involve in polyamine transport in oomycetes.

# Functional analysis of an effector PcRxLR23 in *Phytophthora capsici*

Jing Li[1,2], Haiqing Xu[1,2], Peng Ding[1,2], Ruxue Chen[1,2], Li Zhang[1,2], Tao Liang[1,2], Liying Wang[1,2], Xiuguo Zhang[1,2]

([1]College of Plant Protection; [2]Shandong Provincial Key Laboratory for Biology of Vegetable Diseases and insect, Shandong Agricultural University, Tai'an, China)

*Phytophthora capsici* is a soil-borne plant pathogen with a wide range of hosts. *P. capsici* encodes a large number of host cytoplasmic effectors, including 357 putative RxLR and 84 CRN effectors. However, RxLR effectors remain largely unknown on the roles of these effectors in virulence especially in *P.capsici*. In this study, *P.capsici* SD33, a highly pathogenic strain, collected and preserved in our laboratory was used as experimental material. PcRxLR23 was found to cause cell death when transiently expressed in *Nicotiana. benthamiana* and pepper. The assay of subcelluar location showed that PcRxLR23 localized in the cytoplasm and nucleus. PcRxLR23 could also promote zoospores of *P. capsici* infection. In order to further study the function of PcRxLR23 of *P. capsici*, homology-directed repair (HDR)-mediated replacement of the PcRxLR23 was carried out with mRFP4 by CRISPR/Cas9. PcRxLR23 homology-directed repair (HDR) decreased their virulence on pepper, but had no effect on colony growth and sporangium morphology of *P. capsica* transformants. Homozygous PcRxLR23 transgenic *Arabidopsis thaliana* were obtained using floral-dip transformation. Preliminary results showed that PcRxLR23 probably suppressed the growth of leaves and stems, but had no effect on roots growth in transgenic plants. However, the molecular targets of PcRxLR23 remains to be identified and its molecular mechanism is required to further study.

# The Structural and Functional analysis of an effector PcRxLR81 in *Phytophthora capsici*

Tao Liang[1,3], Li Zhang[1,3], Peng Ding[1,3], Liying Wang[1,3], Jing Li[1,3], Haiqing Xu[1,3] , Chunyuan Zhu[2,3]and Xiuguo Zhang[1,3.]

[1] College of Plant Protection, [2] College of Life Sciences and [3]Shandong Provincial Key Laboratory for Biology of Vegetable Diseases and insect, Shandong Agricultural University, Tai'an, China.

Pepper blight of caused by *P. capsici* is a worldwide disease in vegetable production, and its serious occurrence causes great economic losses. *P. capsici* secretes a number of effectors that can promote the pathogen strains infection and colonization on the host plant. RxLR effectors play an important role in infecting the host and interaction between *P. capsici* and plants. In this research, Strain SD33 of *P. capsici* was used as a model to clone an effector named PcRxLR81. At first, we got the crystal structure of PcRxLR81. Its structure only has one WY-domain comprising 4 α-helices. The structure of PcRxLR81 shows mainly difference from AVR3a11 that located in the first α-helix and loop-4(the loop between third α-helices and fourth α-helices). The function study showed that PcRxLR81 significantly suppressed cell death induced by Bax, but could not induce hypersensitive response (HR) in *Nicotiana benthamiana*. Subcellular localization assay showed PcRxLR81 distributed in both plant cytoplasm and nucleus. The expression of PcRxLR81 was upregulated in the late stage of *P. capsici* infection and significantly suppressed zoospores of *P. capsici* infection. However, PcRxLR81 impaired the suppression of cell death induced by Bax and *P. capsici* infection after mutating the first α-helice domain and loop-4 based on the described structure, suggesting this α-helice domain might play an important role for interacting with other proteins. However, the molecular target of PcRxLR81 remains to be identified and its molecular mechanism is required to further study.

# Fungicidal activity of *Trichoderma* strain HMQAU140012 on blueberry Phythophthora root rot pathogen

Yanwei Gong, Chen Liang*, Honghai Zhao, Baodu Li
*Chen Liang. Email: syliangchen@163.com

*Phytophthora cinnamomi* is one of important pathogen of blueberry root rot in China, fungicides and varieties could not solve blueberry *Phytophthora* root rot disease, and biocontrol method could be considered as a suitable method for blueberry *Phythophthora* root rot. Plate confrontation experiment result showed that *Trichoderma* strain HMQAU140012 has the best potential for *P. cinnamomi*. The result of indoor bioassay indicated that the inhibition rate of strain HMQAU140012 on mycelium growth of *P. cinnamomi* was 78.8% after 6d plate confrontation. After fermentation medium and fermentation condition of *Trichoderma* strain HMQAU140012 were optimized with single factor tests and orthogonal tests, the effects of fermentation filtrate of *Trichoderma* strain HMQAU140012 on *P. cinnamomi* was determined by mix medium method and growth rate method, the experimental results indicated that the inhibition rate of mycelium growth was 65.7% when the fermentation filtrate diluted 640 times. The inhibitory rates of the fermentation filtrate of the strain on sporangium, zoospore release and germination of the pathogen were 100%. The inhibitory rates of 100 times dilution of fermentation filtrate to each stage were 91.3%, 89.1%, 92.1%, respectively; When the fermentation filtrate was diluted by 400 times, the inhibition rates of each stage were still above 35%. By morphological and molecular biological analysis, the strain HMQAU140012 was identified as *Trichoderma virens*. This is the first report in China that the blueberry *P. cinnamomi* was inhibited by *T. virens*.

# The MADS-box Transcription Factor *PsMAD1* Is Required for Zoosporogenesis and Virulence in *Phytophthora sojae*

Long Lin, Jiawei Wu, Yufei Li, Mingrun Xuan, Hui Qian, Wenwu Ye, Suomeng Dong, Yuanchao Wang*
(Department of Plant Pathology, Nanjing Agricultural University, Nanjing 210095, China)

Transcriptional regulation is critical for development and virulence of *Phytophthora sojae*. MADS-box transcription factors belong to a highly conserved transcriptional regulators family involved in various important biological processes. In *P. sojae*, only one predicted MADS-box gene *PsMAD1* was identified and *PsMAD1* is highly expressed during sporangia and infection stages. To investigate the function of *PsMAD1*, we generated *PsMAD1* mutants by CRISPR/Cas9 system. Compared with the wide-type strain, *PsMAD1* mutants did not show any changes in growth rate and oospore production. Although the sporangia production is normal, no zoospore release can be detected in *PsMAD1* mutants. The microscopy analyses revealed that *PsMAD1* mutants failed in cleavage of the cytoplasm into uninucleate zoospores. Furthermore, the mutants showed reduced virulence to soybean. By RNA-seq analysis, we identified 280 and 44 differentially expressed genes in the mutants at sporangia and infection stages, respectively. In summary, our results indicated that *PsMAD1* is involved in *P. sojae* zoospore development and virulence.

# Investigation of the genetic diversity of *Phytophthora capsici* in China using a universal fluorescent labeling method

Xiao-Ren Chen[1*], Ye Zhang[1], Shen-Xin Huang[1], Ting-Ting Liu[1], Guang-Hang Qiao[2*]
([1]College of Horticulture and Plant Protection, Yangzhou University, 48 Wenhui Eastern Road, Yangzhou 225009, Jiangsu Province, China; [2]Institute of Plant and Environment Protection, Beijing Academy of Agriculture and Forestry Sciences, 9 Shuguanghuayuan Middle Road, Haidian District, Beijing 100097, China)

*Phytophthora capsici* is an important oomycete pathogen threatening the vegetable production in China, but very little is known about the population structure. The objective of the present study was to evaluate the genetic diversity of 54 *P. capsici* isolates obtained from 2007–2015 at 12 provincial locations in China. Isolates were assessed for mating type, metalaxyl resistance, and simple sequence repeat (SSR) genotype. Mating-type analyses of 54 isolates showed that both mating types were present in most (9) of the sampled production regions (12), and the mating-type frequency in the total Chinese population did not deviate significantly from a 1 : 1 ratio. Responses of isolates to the fungicide metalaxyl indicated the absence of metalaxyl-resistance among the field populations. A universal fluorescent labeling method was adapted in this study to improve the efficiency of SSR genotyping. Microsatellite genotyping using 7 SSR markers was performed on 54 isolates. Genetic analyses using the program STRUCTURE indicated the existence of 2 genetic clusters within Chinese *P. capsici* collection. The same result was obtained by using the program Power Marker. Further studies with expanded scale sampling at single locations over multiple years will be valuable to define the genetic diversity of *P. capsici* in China.

# PARTICIPANTS

**A**

**Arijit Panda**
arijpanda@gmail.com
CSIR-Indian Institute of Chemical Biology, Structural Biology and Bioinformatics Kolkata, West Bengal, India

**Audrey Ah-Fong**
audreya@ucr.edu
University of California, Microbiology & Plant Pathology Riverside, California, United States

**B**

**Baodian Guo**
2016202012@njau.edu.cn
Nanjing Agricultural University, Department of Plant Pathology

**Brett M. Tyler**
brett.tyler@oregonstate.edu
Oregon State University, Botany and Plant Pathology Corvallis, OR, USA

**Biao Gu**
bgu@nwafu.edu.cn
Northwest A&F University, Yangling, Shaanxi, China

**Bo Yang**
2014202004@njau.edu.cn
Nanjing Agricultural University, Department of Plant Pathology

**Baihua Sun**
Shandong Agriculture University Department of Plant Pathology

**C**

**Chunyun Gao**
Nanjing Agricultural University

**Chen Han**
2014202014@njau.edu.cn
Nanjing Agricultural University, Plant Protection Nanjing, jiangsu, China

**Chuyun Gao**
2017202005@njau.edu.cn
Nanjing Agricultural University

**Chao huo**
YNhuochao@hotmail.com
Industrail Crops Institite of Yunan Academy of Agricultural Sciences, Yunan Academy of Agricultural Sciences Kunming, Yunnan, China

**Chunyuan Zhu**
zhuchunyuan@sdau.edu.cn
Shandong Agriculture University Department of Plant Patholog

**Congcong Ai**
Shandong Agriculture University Department of Plant Pathology

**Cancan Yang**

Shandong Agriculture University
Department of Plant Pathology

**Chaoyang Tang**
2015202006@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Chen Liang**
Qingdao Agricultural University,
Department of Plant Pathology
Qingdao, Shandong, China

**D**

**Diya Sen**
diya.sen@slu.se
Swedish Universit of Agricultural Sciences, Department of Plant Protection Biology Alnarp, Sweden

**Daolong Dou**
ddou@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Daohong Jiang**
daohongjiang@mail.hzau.edu.cn
Huazhong Agricultural University,
Wuhan, Hubei, China

**F**

**Francine Govers**
francine.govers@wur.nl
Wageningen University,
Laboratory of Phytopathology
Wageningen,Netherlands

**G**

**Guanghui Kong**
gkong@scau.edu.cn
South China Agricultural University

**H**

**Haixia Wang**
13212798446@163.com
Huazhong Agricultural University

**Haiyang Li**
2014202015@njau.edu.cn
Nanjing Agricultural University,
Department of Plant Patholog

**Han Chen**
2014202014@njau.edu.cn
Nanjing Agricultural University
Weigang No.1, Xuanwu District
Nanjing, jiangsu, China

**Haonan Wang**
2015202021@njau.edu.cn
Nanjing Agricultural University,
Department of Plant Pathology

**Haiqing Xu**
Shandong Agriculture University
Department of Plant Pathology

**Haohua Li**
Shandong Agriculture University
Department of Plant Pathology

**Han Han**
Shandong Agriculture University
Department of Plant Pathology

**Haiyang Li**
Shandong Agriculture University
Department of Plant Pathology

**Hui Sheng**
Shandong Agriculture University
Department of Plant Pathology

**I**

**Iga Tomczynska**
iga.tomczynska@gmail.com
University of Fribourg,
Biology Fribourg, Switzerland

**J**

**Jessica Upson**
Jess.upson@sainsbury-laboratory.ac.uk
The Sainsbury Laboratory,
Norwich Research Park
Norwich,Norfolk,United Kingdom

**John McDowell**
johnmcd@vt.edu
Virginia Tech, Plant Pathology,
Physiology & Weed Science
Blacksburg, VA, USA

**Jang lu**
lyxnesta@163.com
Shanghai Jiao Tong University
School of Agriculture and Biology
Shangha

**Jie Huang**
2015202004@njau.edu.cn
Nanjing Agriculture University

**Jing Li**
13579981032@163.com
Shandong Agricultural University
Department of Plant Pathology

**Jun zhao**
zhaojun02@hotmail.com
Inner Mongolia Agri. Univ.,

**Junjie Xu**
junjiexu@nwafu.edu.cn
Northwest A & F University

**Junjie Qu**
624315158@qq.com
Guangxi Zhuang Autonomous
Region Academy of Agricultural
Sciences, Guangxi

**Justin**
Nanjing Agricultural University,
Nanjing

**Ji Wang**
wangjibiology@163.com
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Jierui Si**
2015102009@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Jing Yu**
2015202007@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Jianqiang Miao**
mjq2014@cau.edu.cn
China Agricultural University
Beijing, China

**K**

**KaiTao**
TaoKai.tao@oregonstate.edu
Oregon State University
MCB/BPP Corvallis,United States

**Kelsey Wood**

klsywd@ucdavis.edu
University of California - Davis, Genome Center Davis, California, United States

**Kyle Fletcher**
kfletcher@ucdavis.edu
University of California, Genome Center Davis, California, United States

**L**

**Lanlan Li**
476196842@qq.com
Yunnan institute of tropical crops, Jinghong, Yunnan, China

**Laura Grenville-Briggs Didymus**
laura.grenville.briggs@slu.se
Swedish University of Agricultural Sciences, Department of Plant Protection Biology Alnarp, Choose a State/Province, Sweden

**Lida Derevnina**
lida.derevnina@tsl.ac.uk
The Sainsbury Laboratory, Colney Lane Norwich, United Kingdom

**Li Zhao**
zhaolisdau@163.com
Shandong Agricultural University Department of Plant Pathology

**Lin Zhang**
linzhang@ucdavis.edu
University of California, Davis, The Genome Center Davis, California, United States

**Liyuan Wang**
2017202015@njau.edu.cn
Nanjing Agricultural University Plant Pathology Nanjing, jiangsu, China

**Liying Wang**
1479779905@qq.com
Shandong Agricultural University Department of Plant Pathology

**Long Lin**
2015202005@njau.edu.cn
Nanjing Agricultural University, Department of Plant Pathology

**Ling Yin**
779335723@qq.com
Guangxi Zhuang Autonomous Region Academy of Agricultural Sciences, Guangxi

**Li Zhang**
Shandong Agriculture University Department of Plant Pathology

**Lina Yang**
yikeshu1114@126.com
Fujian Agricultuel and Forestry University Fuzhou, Fujian, China

**Liyun Guo**
ppguo@cau.edu.cn
China Agricultural University, Department of Plant Pathology Beijing, China

**M**

**Maofeng Jing**
jingmf@njau.edu.cn
Nanjing Agricultural University,

Plant Pathology Nanjing, jiangsu, China

**Maria Fernanda Proano**
maria.f.proano_cuenca@okstate.edu
Oklahoma State University,
127 Noble Resarch Center
Oklahoma, United States

**Mathu Malar C**
mmadhubioinfo@gmail.com
Indian Institute of Chemical Biology,
4 raja s c mullick road, Jadavpur
Kolkata, west bengal, India

**Min Qiu**
2015202016@njau.edu.cn
Nanjing Agricultural University,
Department of Plant Pathology

**Ming Zhang**
2015102010@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Mehran Khan**
Fujian Academy of Agricultural Sciences Institute of Plant Protection
Fuzhou, fujian, China

**Maozhi Ren**
renmaozhi@cqu.edu.cn
Chongqing University,
School of Life Sciences
Chongqing, China

**O**

**Ola**
Nanjing Agricultural University

**P**

**Paul Birch**
P. Birch@dundee.ac.uk
University of Dundee,
Plant Sciences Dundee, United Kingdom

**Paul Morri**
pmorris@bgsu.edu
Bowling Green State University,
Biological Sciences Bowling Green,
Ohio, United States

**Petra Boevink**
petra.boevink@hutton.ac.uk
The James Hutton Institute,
Cell and Molecular Sciences
Dundee, United Kingdom

**Pinggen Xi**
xpg@scau.edu.cn
South China Agricultural University

**Preeti Panda**
Preeti.Panda@scionresearch.com
Forest Protection, Rotorua, Bay of Plenty, New Zealan

**Peng Ding**
Shandong Agriculture University
Department of Plant Pathology

**Peiqing Liu**
Fujian Academy of Agricultural Sciences Institute of Plant Protection
Fuzhou, fujian, China

**Q**

**Qiang Zhang**
qfzhangqiang@gmail.com
Northwest A&F University,
College of Plant Protection

Yangling, Shaanxi, China

**Qin He**
He.Qin@hutton.ac.uk
University of Dundee,
Division of Plant Sciences At James Hutton Institute Dundee, Scotland, United Kingdom

**Qun Wang**
Shandong Agriculture University
Department of Plant Pathology

**Qi Li**
liqi@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Qiang Yan**
yanqiang@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Qinghe Chen**
Fujian Academy of Agricultural Sciences Institute of Plant Protection
Fuzhou, fujian, China

**Qin Peng**
pengqin1991@126.com
China Agricultural University
Beijing, China

**R**

**Rebecca McDougal**
rebecca.mcdougal@scionresearch.com
Scion (New Zealand Forest Research Institute, Ltd,
Forest Protection
Rotorua, Bay Of Plenty, New Zealand

**Ruofang Zhang**
ruofang_zhang@163.com
Inner Mongolia University

**Ruirui Feng**
fengruiruiw@nwafu.edu.cn
Northwest A & F University

**Rui Feng**
Shandong Agriculture University
Department of Plant Pathology

**Ruxue Chen**
Shandong Agriculture University
Department of Plant Pathology

**Rongbo Wang**
Fujian Academy of Agricultural Sciences Institute of Plant Protection
Fuzhou, fujian, China

**S**

**Sebastian Schornack**
sebastian.schornack@slcu.cam.ac.uk
University of Cambridge,
Sainsbury Laboratory (SLCU)
Cambridge, United Kingdom

**Shuaishuai Wang**
2015202022@njau.edu.cn
Nanjing Agricultural University

**Shumei wang**
smwang@dundee.ac.uk
university of dundee,
plant science Dundee

**Shuyun chen**

178685504@qq.com
Shanghai Jiao Tong University,
School of Agriculture and Biology
Shanghai

**Sucheta Tripathy**
tsucheta@gmail.com
CSIR- Indian Institute of Chemical Biology, 4, Raja, S.C Mullick Road, Jadavpur Select

**Sophien Kamoun**
Sophien.Kamoun@sainsbury-laboratory.ac.uk
The Sainsbury Laboratory
Norwich, UK

**Suomeng Dong**
smdong@njau.edu.cn
Nanjing Agricultural University,
College of Plant Patholo
Nanjing,Jiangsu,China

**Shi Wang**
Shandong Agriculture University
Department of Plant Pathology

**Susu Gao**
Shandong Agriculture University
Department of Plant Pathology

**Shengting Huang**
Shandong Agriculture University
Department of Plant Pathology

**Shujie Wu**
Shandong Agriculture University
Department of Plant Pathology

**Shanshan Chen**
Shandong Agriculture University
Department of Plant Pathology

**Shenxin Huang**
sxhuang11@163.com
Yangzhou University
Yangzhou, Jiangsu, China

**Shuqing Yang**
Heilongjiang Academy Of Agricultural Sciences
Haerbin, heilongjiang, China

**Sanwen Huang**
The Instituteof Vegetables and Flowers Chinese Academy of Agricultural Sciences Beijing, China

**T**

**Tao ma**
470904757@qq.com
Shanghai Jiao Tong University
School of Agriculture and Biology
Shanghai

**Theerapong Krajaejun**
theerapong.kra@mahidol.edu
Mahidol University,
Department of Pathology,
Ramathibodi Hospital
Bangkok, Bangkok, Thailand

**Tao Liang**
Shandong Agriculture University
Department of Plant Pathology

**Tongtong Zhu**
Shandong Agriculture University
Department of Plant Pathology

**U**

**Unnati Sonawala**

unnati@vt.edu
Virginia Tech,
Plant Pathology, Physiology and
Weed Science Blacksburg, Virginia,
United States

**V**

**Veronica Ancona**
veronica.ancona-contreras@tamuk.edu
Texas A&M University-Kingsville,
312 N International Blvd Weslaco

**W**

**Weiwei Li**
weiweili@nwafu.edu.cn
Northwest A & F University

**Weixing Shan**
wxshan@nwafu.edu.cn
Northwest A & F University,
Xianyang City, Shaanxi Province,
China

**Wen Li**
1034229389@qq.com
South China Agricultural University

**Wenjie Wu**
jiezi_1@126.com
Northwest A & F University,
Xianyang City, Shaanxi Province,
China

**Weiwei Song**
weiweisdau@126.com
Shandong Agricultural University,
Department of Plant Pathology

**Wenwu Ye**
yeww@njau.edu.cn
Nanjing Agricultural University,
Department of Plant Pathology

**Weizhen Wang**
wzwangyx163@163.com
China Agricultural University
Beijing, China

**X**

**Xia lan**
359990855@qq.com
Shanghai Jiao Tong University,
School of Agriculture and Biology
Shanghai

**Xufang Qiu**
2016102038@njau.edu.cn
Nanjing Agricultural University,
Department of Plant Pathology

**Xiuguo Zhang**
zhxg@sdau.edu.cn
Shandong Agriculture University
Department of Plant Pathology

**Xiuqi Li**
Shandong Agriculture University
Department of Plant Pathology

**Xiaoman Liu**
Shandong Agriculture University
Department of Plant Pathology

**Xuefa Zhang**
Shandong Agriculture University
Department of Plant Pathology

**Xiaoren Chen**
xrchen@yzu.edu.cn
Yangzhou University
Yangzhou, Jiangsu, China

**Xin Zhang**

ailuo36@163.com
The Institute of Plant Protection,
Hunan Province ChangSha, Hunan,
China

**Xiong Zhang**
2014202002@njau.edu.cn
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Xili Liu**
seedling@cau.edu.cn
China Agricultural University
Beijing, China

**Xiaodan Wang**
wxd_210@126.com
Heilongjiang Academy Of
Agricultural Sciences
Haerbin, heilongjiang, China

**Xinyu Yang**
yangxy@syau.edu.cn
Shenyang Agricultural University
Shenyang, jilin, China

**Y**

**Yachun Lin**
2013202014@njau.edu.cn
Nanjing Agricultural University
Department of Plant Pathology

**Yan Wang**
yan.wang@njau.edu.cn
Department of Plant Pathology,
Faculty, Research Associate,
Professional

**Yang Yang**
YangYang9514@nwafu.edu.cn
Northwest A & F University

**Yeqiang Xia**
2016202017@njau.edu.cn
Nanjing Agricultural University,
Department of Plant Pathology

**Yu Du**
yu.du@nwafu.edu.cn
Northwest A&F University,
Xianyang City,Shaanxi Province,
China

**Yuanchao Wang**
wangyc@njau.edu.cn
Nanjing Agricultural University，
Department of Plant Pathology

**Yuanpeng Xu**
2015202017@njau.edu.cn
Nanjing Agricultural University，
Department of Plant Pathology

**Yunxiao liu**
lyxnesta@163.com
Shanghai Jiao Tong University,
School of Agriculture and Biology
Shanghai

**Yujiao Wang**
Shandong Agriculture University
Department of Plant Pathology

**Yang Zhou**
Nanjing Agricultural University
College of Plant Protection
Nanjing, Jiangsu, China

**Ye Zhang**
424396702@qq.com

Yangzhou University
Yangzhou, Jiangsu, China

**Yue Jiang**
Fujian Academy of Agricultural Sciences Institute of Plant Protection
Fuzhou, fujian, China

**Yuan Fang**
fangyuan7852@163.com
China Agricultural University
Beijing, China

**Z**

**Zachary Noel**
noelzach@msu.edu
Michigan State University ,
578 Wilson Rd. CIPS 104
East Lansing,United States

**Zhijian Zhao**
zhijianzhao@hotmail.com
Industrial Crops Institute, Yunnan Academy of Agricultural Sciences, Beijing Road 2238#, Kunming
Kunming, Yunnan, China

**Zhiying Cai**
391640395@qq.com
Yunnan institute of tropical crops,
Jinghong, Yunnan, China

**Zide Jiang**
zdjiang@scau.edu.cn
South China Agricultural University

**Zhongyang Wang**
Shandong Agriculture University
Department of Plant Pathology

**Zhenling Huang**
Shandong Agriculture University
Department of Plant Pathology

**Zhendong Tian**
Tianzhd@mail.hzau.edu.cn
Huazhong Agricultural University
College of Horticulture & Forestry Sciences Wuhan, Hubei, China

**Zhuo Zhang**
18637994@qq.com
The Institute of Plant Protection, Hunan Province ChangSha, Hunan, China

**Zhaolin Xue**
xuezhaolin1215@163.com
China Agricultural University
Beijing, China

**Note: our apologies if an attending author was not listed in the participants or an abstract is missing. Any error or omission is strictly my fault (Cancan Yang)**