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Congress Registration and Call for Papers Now Open!



The Program Committee for the XV International Congress on Molecular Plant-Microbe Interactions welcomes abstract submissions for both oral and poster presentations. Formatting information and session information can be found on the congress website (<http://mpmi2011.umin.jp>). Please note that you must be registered to attend the congress before you can submit your abstract, and you will need your registration number to submit. Visit the meeting website to register, view program information, plan your travel, and more. ■

IS-MPMI Invites You to Kyoto, Japan, for the XV International Congress on Molecular Plant-Microbe Interactions, August 2–6, 2011



The XV International Congress on Molecular Plant-Microbe Interactions (MPMI) is recognized as the most important international meeting for plant-microbe interactions, facilitating networking with colleagues from around the world. This meeting is the global venue for presenting and discussing new research and developments in molecular plant-microbe interactions. Through plenary lectures, concurrent sessions, special workshops, and various events, attendees experience innovative plant-microbe interactions research. The meeting features hundreds of abstracts and provides networking and professional development opportunities.

There are eight plenary and 21 concurrent sessions scheduled for the XV International Congress on MPMI to be held August 2–6, 2011, at the Kyoto International Conference Center in Kyoto, Japan (<http://mpmi2011.umin.jp/program.html>). The list of speakers for the plenary sessions is provided below. These are concurrent sessions to which abstracts can be submitted for poster presentation. For each concurrent session, four abstracts will be selected for oral presentation.



Kyoto International Conference Center

It should be noted that there will be special workshops concerning proteomics and rice defense response on August 2. Four additional workshops will be planned.



Optional Excursion: Kiyomizu-dera

Opening Lecture

Shizuo Akira (Osaka University, Japan)

Confirmed Speakers (Plenary Sessions)

Jeff Dangl (U.S.A.)

Jean Denarie (France)

Maria Harrison (U.S.A.)

Sheng Yang He (U.S.A.)

Jonathan Jones (United Kingdom)

Sophien Kamoun (United Kingdom)

Gregory Martin (U.S.A.)

Paul Schulze-Lefert (Germany)

Brian Staskawicz (U.S.A.)

Jens Stougaard (Denmark)

Jian-min Zhou (China)

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IS-MPMI Reporter

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IS-MPMI REPORTER DEADLINE

The deadline for submitting items for the next issue is April 29, 2011.

Share your news, accomplishments, and upcoming meeting details with your colleagues. Submit articles, announcements, and any ideas you may have for the next issue. You can send an e-mail (ismpmireportereditor@scisoc.org) or submit your item online (www.ismpminet.org/newsletter/submissionform.asp).

Send items to:

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Felice Cervone, President

A Letter from the President

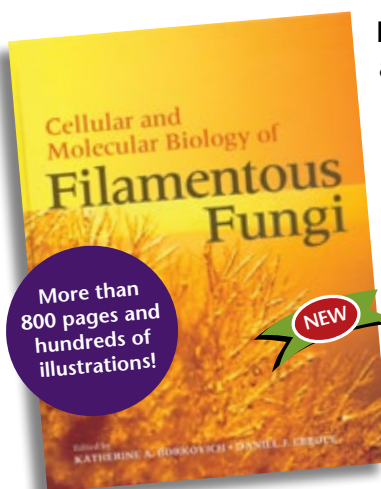
First of all, I wish all of you a happy and fruitful 2011 with plenty of novel results and important discoveries in the field of plant-microbe interactions. I believe that we are facing changes and transformations in almost every sphere of life, and biotechnology is at the center of these changes. I also believe that the scientists of IS-MPMI contribute greatly to this process by discovering the function of plant genes that confer resistance to stresses or regulate the interaction with beneficial microbes. Much has yet to be done; more mechanisms have to be elucidated and applications have to be

implemented. Our scientists are at work. The time is approaching when we will possess tools to be applied for a sustainable agriculture.

In 2010, our society celebrated its 20th anniversary, and we now begin our 21st year looking forward to one of the most notable events, the XV International Congress on Molecular Plant-Microbe Interactions in Kyoto, a charming city full of life and tradition and Japan's former capital. Information about the program and abstract submissions are online! Chair **Ko Shimamoto** already has an impressive list of confirmed speakers, including the most prestigious scientists in the field of plant-microbe interactions. Other speakers will be chosen from the authors of submitted abstracts. Do not miss this unique opportunity to present your results, meet with colleagues, and access the most recent results of researchers from all over the world. I expect that many of you, especially the young members of the society, will attend the Kyoto meeting to continue the long-standing tradition of successful IS-MPMI meetings. I look at the young participants as the future architects of the society and expect from their participation a novel impulse to conduct research and help in shaping the perspectives of our science.

Please, tell your colleagues about the XV International Congress on MPMI in Kyoto and do not forget to renew your membership to receive benefits and a reduction in the congress registration fee. Participate and enjoy the cutting-edge science in plant-microbe interactions at the XV International Congress on Molecular Plant-Microbe Interactions in Kyoto, August 2–6, 2011. ■

An ideal starting point for any research studies on filamentous fungi.



Edited by Katherine Borkovich and Daniel J. Ebbole

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Concurrent Sessions for Abstract Submissions

1. Recognition and signaling I/II
2. Symbiosis I/II
3. Plant-oomycete/fungal interactions
4. Plant-nematode interactions
5. Pathogenic fungi
6. Pathogenic bacteria/phytoplasma
7. Genomics and evolution of virulence in pathogenic fungi/oomycetes
8. Endophytes and parasitic plants
9. Effector proteins
10. Plant-virus/viroid interactions
11. Cell wall modification and resistance
12. Plant response
13. Evolution of susceptibility and resistance
14. Biocontrol interactions
15. Systems biology
16. Plant hormones integrating defense response
17. Biotechnology
18. Structural biology
19. Crop protection

Twitter

The congress is on Twitter at <http://twitter.com/MPMIKyoto>. Be sure to follow us for the latest congress information and be a part of the conversation.

Registration

A reduced congress registration fee is offered for IS-MPMI members. To receive this benefit, you must be a member of IS-MPMI *before* registration. As a member of IS-MPMI, you will receive a ¥10,000 (\$120 USD) discount on your

congress registration. Note that preregistration is only available online at <http://mpmi2011.umin.jp/regist.html>.

Have colleagues interested in attending the congress? Tell them to join IS-MPMI to save on their registration!

Excursion

An optional excursion is planned to Kiyomizu-dera and Gion Hanamikoji dori. For information on this world heritage tour, visit <https://apollon.nta.co.jp/mpmi2011/files/mpmiexcursion.pdf>.

Accommodations

Nippon Travel Agency has booked rooms at the hotels in Kyoto for the duration of the congress. Visit <http://mpmi2011.umin.jp/regist.html> for more information.

Sponsorships

Sponsorship opportunities are available. If your organization is interested in becoming a sponsor for the XV International Congress on MPMI, please contact mpmikyoto2011@bs.naist.jp. Information on sponsorships is available online at <http://mpmi2011.umin.jp/sponsor.html#sponsorship>. ■

Important Dates

Early Registration

December 15, 2010 – May 2, 2011

Abstract Submission

January 12, 2011 – April 6, 2011

	Early Registration (Before May 2, 2011)	Late Registration (After May 3, 2011)	On-Site Registration (After July 1, 2011)
IS-MPMI Member	¥68,000/\$810	¥73,000/\$870	¥78,000/\$930
Nonmember	¥78,000/\$930	¥83,000/\$990	¥88,000/\$1,049
Student		¥34,000/\$405	
Accompanying Person		¥20,000/\$238	
Excursion		¥4,000/\$49	–
Congress Dinner		¥10,000/\$122	–

All fees are listed in Japanese yen (JPY) (¥) and U.S. dollars (\$).

Visit <http://mpmi2011.umin.jp>
for the latest congress information



MPMI Scientists Offer Insights into Their Recently Published Research

IS-MPMI Editor-in-Chief **Jean-Pierre Metraux** interviewed **Pietro Spanu**, IS-MPMI member and first author of the recent article “Genome expansion and gene loss in powdery mildew fungi reveal tradeoffs in extreme parasitism” (Spanu et al., 2010, *Science* 330:1543-1546); **Laura Baxter** and **Sucheta Tripathy**, the first coauthors of the recent article “Signatures of adaptation to obligate biotrophy in the *Hyaloperonospora arabidopsidis* genome” (Baxter, Tripathy, et al., 2010, *Science* 330:1549-1551); **Bart Thomma**, one of the authors of the recent article “Conserved fungal LysM effector Ecp6 prevents chitin-triggered immunity in plants” (de Jonge et al., 2010, *Science* 329:953-955); and **Nuria S. Coll**, first author of the recent article “*Arabidopsis* type I metacaspases control cell death” (Coll et al., 2010, *Science* 330:1393-1397).

An Interview with Pietro Spanu

Q: When did the first sparks for this study start?

A: We had been seeking funds to sequence the *Blumeria* genome for ages. The first (unsuccessful) bids were made back in the 1990s. From very early on, we hoped to establish a data platform to mine information about genes and genome structure in an obligate biotrophic pathogen of high impact. With hindsight, our lack of success then was a good thing as we would have been unable to achieve anything close to our original targets because of the unexpectedly large genome sizes in mildews and their massive levels of idiosyncratic repetitive DNA derived from proliferation of the retrotransposons. It was fortuitous that we were funded generously by the BBSRC toward the end of the days of “Sanger Genomes,” just at the advent of the second-generation high-throughput sequencing (HTS) platforms. The long, paired-end reads from conventional Sanger sequence helped to assemble complex, repetitive DNA at a time when the relatively short HTS and the early bioinformatic tools for data handling would not have coped well.



Q: What is the composition of the group involved?

A: The working groups from U.K. labs at Imperial (London), Exeter, Oxford, Norwich, and Paris applied for funds from BBSRC and INRA. When successful, we established the core BluGen consortium, which started on *B. graminis* f. sp. *bordei*. After a year or so, we were joined by colleagues from the Max-Planck Institute in Cologne who had initiated independently work on the dicot mildews *Golovinomyces orontii* and *Erysiphe pisi*. This was driven by the early realization that synergies across labs and comparative genomics was going to add a hugely significant

value to all of our ventures. We were also very fortunate to associate ourselves with groups from Reading (U.K.) and Copenhagen who supplied significant proteomics and additional unpublished EST data vital to the annotation. Once the bulk of sequencing and assembly was underway, we had another very significant influx of colleagues from Zürich, Cornell, and Madrid, who joined the effort to carry out the intense human-curated annotation process and, vitally, the interpretation of the sequence data in the various biological contexts. In the end, a large group of over 60 collaborators from all over worked together effectively and efficiently to achieve our goal. We ranged from senior “dinosaurs” who weighed in at the early fundraising stages and at the final drafting of the manuscript(s), bringing their priceless expertise and experience, to mid-career scientists each with their own specialty and understanding in bioinformatics and biological interpretation of the data, to postgraduate students who contributed vitally to the painstaking manual annotation and curation of the genomic information.

Q: What was the atmosphere?

A: Well, what do you expect when 60 or more people work together for over five years? We experienced everything, I suppose. Excitement to drive the early applications; disappointment when these initially failed; hope when we tried again; elation when we succeeded in getting the funds; and worry that the process was actually doable. Would we really be able to get DNA, RNA, and protein of quality to sequence, profile transcripts, and identify proteins by mass spectrometry from organisms that cannot be cultured except on a plant in a greenhouse or growth room? Joy and satisfaction when we found that we could actually do this; heart-stopping realization that something was not as planned, when the size of the genomes appeared to be so “off the predicted scale”; anxiety that the clock was ticking and that we needed to complete the task before the end of grant deadlines; courage instilled by the incredible camaraderie shown all over at our first annotation workshop; playful competition during annotation when we would all see what everyone else was doing, how many and how well the annotations were coming along; short-tempered arguments and disagreements over what and how we should draft the main paper; heart-in-hand spine-chilling wait for the journal responses to our submission; a glow of pride when the pack of glossy prints arrived with FedEx this week.... You name it: we experienced it.

Q: What were the big stumbling blocks? How did you manage to get them out of the way?

A: Of course there were stumbling blocks. Some of these are mentioned above. The first one was at the grant application stage: we were not successful the first time, nor the second...but we persevered. When the time was right, we got the funds to do this. Hindsight is a great thing! Then, I suppose, the day the first batch of Sanger sequence came in and the preliminary assemblies were telling us that we had either sequenced more than one organisms (remember, we are dealing with an obligate biotrophic pathogen) or that the genome was much larger and much more repetitive than predicted. Fortunately, we could prove that the second was the case. But the money had nearly run out for Sanger sequencing when we finally realized that we were not going to get anywhere near the necessary coverage to get a half-decent assembly. Cue a great stroke of luck: HTS came on the scene with competing companies and platforms vying for projects to demonstrate their wares. We were extremely fortunate to be able to take advantage of pilot runs at cut-down prices for early runs of Solexa/Illumina, 454 pyrosequence, and SOLiD, without which we would never have achieved what we have in the time frame available. Another difficulty, which is well known but not very often formally talked about, is that data and information often have a sell-by date. Literally. It became clear to most of us that if we wanted to publish this in a high-profile journal, we needed to finish fast, and around 2010. We knew there were “competing” genomes from other interesting biotrophic pathogens, some of them obligate. We overcame this stumbling block by openly discussing with other consortia possible joint submission (as separate papers). I am duty bound to acknowledge the amazing goodwill of many colleagues in potentially competing consortia to “play fair” together. We were also advised very wisely and effectively by the editors at *Science*, who steered us through the process in a way that respected the necessary peer review but coordinated effectively the simultaneous publication of four significant manuscripts in the same issue: no mean feat.

Such a large amount of work needed to be condensed in less than 2000 words—and words that made an impact sufficient to draw the attention of a general science readership. In many ways, this is an impossible feat. Of course, there were disagreements about both content and style. Many of us were disappointed that our “pet” topics did not make it to more than a short sentence in the Supplementary On-Line Material. But the realization that it was either going to be this or nothing, pushed us to inevitable compromises. Also, I have pushed very hard (and still am) for the publication of as much of the data as possible in associated manuscripts that are coming out and are in preparation.

Q: What's next?

A: From a purely research perspective, there are two main products of this work. First, a huge data set of annotated sequence (DNA, RNA, and protein) from powdery mil-

dews available to anyone who wishes to use it. We expect this to be an incredible reference resource for years to come to build on and to inform and support research in pathology and plant-microbe interactions at all levels. Second, an effective, motivated, and energized “community” of mildew researchers who have worked together successfully and plan to continue doing so. We will continue to work as a consortium to share unpublished data and ideas to promote experimental platforms (e.g., tools for functional genomics) and have already planned meetings (Cologne in February 2011 and Kyoto in August 2011). Many of us collaborate in formal, funded projects or in informal cooperation. In many cases, these links were established by the genome project and are an invaluable legacy for all to exploit.

An Interview with Baxter and Tripathy

Q: When and where did the first sparks for this study start? Was it all planned in one block or did the project evolve?

A: Tripathy: *Hpa* is an obligatory biotroph causing downy mildew disease. Post-genome era, many of the mechanisms of a biotrophic to a necrotrophic life cycle switch were analyzed and understood in *Phytophthora* spp. The effector entry into the host was most important of it all. The genomic perspective for a obligatory biotrophic life cycle was very intriguing, thus the project began. The genome-sequencing project started during mid-2006 with EST sequencing. Immediately afterward, whole-genome shotgun sequencing was done. In 2007, BAC sequences were made available, followed by next-generation sequences with Illumina. With the addition of new data, the project started evolving with new and additional information.

A: Baxter: The project began as a collaboration between labs at Virginia Tech and Warwick University. The VBI lab had previously sequenced *Phytophthora sojae* and *P. ramorum*, and now we had the chance to sequence an obligate biotroph relative. A key issue is the need to understand how *Hpa* suppresses its host plant's immunity, and by sequencing the genome, we were better able to identify genes such as the so-called RXLRs that constitute the pathogen's arsenal of attack against plants. The project plan was reasonably straightforward—the Washington Genome Sequencing Centre was to shotgun sequence the genome, while the Sanger Centre would provide BAC sequences that we selected from our library at Warwick.



Laura Baxter (left) and Sucheta Tripathy (right)

We had new EST libraries made, which were also sequenced at Sanger. Once the data was available, it was up to us to then integrate it, do the automated annotation and manual refinements, and make the resource available for the research community. As it happened, a new collaborator's lab was later able to provide next-generation sequencing data, which we used to enhance the existing assembly.

Q: What were the human difficulties in running such a large project? How were people informed/motivated? How did you manage to maintain an overview?

A: Tripathy: Usually, this type of multi-organizational project starts off with an annotation jamboree, where interested groups get assembled and annotate sequence data. The sequence jamboree for *Hpa* was hosted at VBI, and we provided the basic annotations, such as gene call and primary and functional annotations. The VMD database provided a platform for users to deposit their information and browse the data. Users submitted their data/finding in a one-page format. With every subsequent assembly version, users/interest groups were informed about the availability of new data and most of the time a deadline was given for them to complete analysis. We maintained a mailing list and often had conference calls on a weekly basis. Long projects such as this required constant reminders and follow-ups to get everybody to send their piece. The real challenge was to incorporate all the information into the central theme of the paper. Most of the work that was not submitted by the user groups was done by the first authors. This paper took four long years of constant redos and repeats to come to the stage it is now.

A: Baxter: A great thing about the project was that researchers from so many labs (19 in total) on both sides of the Atlantic were involved—U.S.A., U.K., Canada, Netherlands, Germany, France, and China. But with so many people working on the project, communication was key. The jamboree was a great opportunity for everyone to meet up and work together for a whole week, when otherwise we had to rely on e-mails and conference calls. After the jamboree, people went away and worked on subsets of the data that they found interesting and sent us “vignettes” to gather together into the final paper. We were fortunate that so many people wanted to get involved and were usually very quick at getting detailed reports back to us.

Q: What were the big stumbling blocks? How did you manage to get them out of the way?

A: Tripathy: The stumbling blocks were many; the most prominent of which were the multiple assembly versions.

A: Baxter: There were eight major releases, and some additional minor releases in between! It was sometimes a battle to keep up with running the same computations on each new version, but Sucheta did a great job on this,

and importantly it meant that the research community had access to the most up-to-date data at each stage in the project.

A: Tripathy: Each time we incorporated extra information, such as BACs and Illumina reads, to close the gaps as much as possible, it led to redoing the entire annotation all over again. The story had to be reconstructed with every assembly version. It is like working with an entirely new genome. We did not have separate jamborees for each assembly version, so the core group took over redoing most of the work.

A: Baxter: Yes, integrating the data (Sanger reads, BACs, ESTs, and Illumina reads) as it became available was a challenge. We hadn't anticipated Illumina reads being available at the start of the project, but we were pleased to be able add the “next-gen” data from a new collaborator at the Sainsbury lab.

Q: What's next?

A: Tripathy: *Hpa* has revealed the streamlining of genomes from a necrotrophic life cycle to obligate biotrophy. We will continue to work in related genomes to further strengthen this hypothesis.

A: Baxter: Also, now that we've identified potential key effectors, the next step is to understand what they are doing in the host plant to suppress the immune network. It was great to be involved in such an interesting and varied project alongside a great international team of researchers. Everyone involved put in a huge amount of time and effort, but I think the end result was well worth it.

An Interview with Bart Thomma

Q: When did the first sparks for this study start?

A: The story took off rather slowly. **Matthieu Joosten**, a colleague-PI at the Laboratory of Phytopathology, was performing 2-D gel analyses of proteins present in apoplastic fluids harvested from the interactions of various *Cladosporium fulvum* strains with tomato. In these analyses, Matthieu kept stumbling over a very prominent protein spot on all the 2-D gels of apoplastic extracts from infected tomato leaves that were colonized by the fungus. The spot appeared not to be derived from tomato, and N-terminal sequencing revealed that it indeed concerned a *C. fulvum* protein, which was designated Ecp6 (for extracellular protein 6) as it was the sixth identified in planta-secreted proteins of the fungus. However, as Matthieu's interest is on defense in solanaceous plants, therefore more focused



Bart Thomma

on the plant than on the pathogen, the role of this protein was not pursued in his group.

Since my group is interested in the virulence function of effector proteins of plant-pathogenic fungi, Matthieu proposed that I should have a look at the role of this protein. Together with **Peter van Esse**, at that time a Ph.D. student in my group, the visiting scientist **Melvin Bolton** showed that Ecp6 is a virulence factor, as leaf colonization by *C. fulvum* was significantly hampered in Ecp6-silenced strains.

Two observations urged us to focus on Ecp6 rather than on any of the previously characterized effectors of which we didn't know their exact role in fungal virulence. First of all, there was the finding of **Jack Vossen**, who cloned the full-length gene, that this effector, in contrast to all the other *C. fulvum* effectors, had recognizable protein domains; LysM domains that had been implicated in carbohydrate binding in other proteins. Secondly, again in contrast to all the other *C. fulvum* effectors, clear Ecp6 homologs were found in the genomes of other fungi. This was all published in *Molecular Microbiology* in 2008.

Q: Who did what?

A: At the start of the functional analysis of the Ecp6 protein, I did not have funding to hire someone to work on this effector and different people in the lab performed part of the analysis. Peter, who became a post-doc in my group and attempts to identify the race 1 elicitor of the vascular fungus *Verticillium dahliae*, and **Ronnie de Jonge**, a Ph.D. student who works on the annotation of the *V. dahliae* genome, supervised two M.Sc. students that wanted to work on Ecp6. Results of the students who showed that the protein actually binds chitin but does not protect fungal cell walls against chitinases inspired both Peter and Ronnie. This was in contrast to the previously identified chitin-binding *C. fulvum* effector Avr4, which protects hyphae against hydrolysis by plant chitinases. At that point, we started to believe that Ecp6 works mechanistically distinct from Avr4 and could be a protein that sequesters chitin fragments such that these fragments do not activate the host chitin receptor and thus activate host immunity. Especially Ronnie dug in deeper, and he left the bioinformatics and his computer to go into the lab and set out to prove the immune-suppression hypothesis. Once we found that Ecp6 was able to suppress chitin-triggered immunity by challenging tomato suspension cells with chitin fragments in the absence and presence of Ecp6, the next step was to demonstrate that Ecp6 was able to compete with host chitin receptors. Since the rice CEBiP receptor was the only convincingly reported chitin receptor at that time, I asked Dr. **Naoto Shibuya**, who cloned the gene encoding that receptor, to help us out. He immediately got enthusiastic and his lab members swiftly demonstrated that Ecp6 was able to specifically compete with CEBiP for chitin binding. That closed our story.

Q: What were the big stumbling blocks? How did you manage to get them out of the way?

A: Whereas in cell suspensions of tomato and *Nicotiana benthamiana*, as well as in leaf discs of *N. benthamiana* we could easily activate host defenses upon chitin treatment, chitin appeared not to be a very easy trigger of host defenses in tomato leaf tissue. It took some time to set up a reproducible system to activate and measure a chitin-triggered ROS burst in tomato leaf discs, but in the end we managed. We also spent quite some time to set up similar assays in Arabidopsis. However, we never succeeded in this. Fortunately, *C. fulvum* is a strict tomato pathogen so we did not need the Arabidopsis assay.

Q: What's next?

A: One of the major challenges will be to unravel how LysM domains exactly bind chitin. Although we assume that the three LysM domains of Ecp6 are the three chitin-binding moieties, the experimental evidence for this is lacking. We tried to generate a number of Ecp6 mutants but were faced with unstable proteins.

Furthermore, the zigzag model predicts the existence of tomato accessions that have learned to recognize Ecp6. One of the M.Sc. students that was involved in the initial experiments to reveal the role of Ecp6, **Anja Kombrink**, has now become a Ph.D. student in my group and she has indeed been able to identify a number of exciting tomato accession.

An Interview with Nuria S. Coll

Q: When did the first sparks for this study start?

A: In the lab of **Jeff Dangl**, we study different aspects of plant-pathogen interactions. One of the research areas is to understand the molecular mechanisms that regulate programmed cell death during the hypersensitive response. Key in deciphering this process was the identification of LSD1 (lesion simulating disease 1), a negative regulator of cell death that prevents cell death from spreading beyond the site of pathogen invasion and keeps the hypersensitive response local. Once **Bob Dietrich** had cloned LSD1 in 1996, it became clear that LSD1 contained a novel zinc finger domain. Based on the then-available EST database, he and **Mike Richberg** identified just a few other Arabidopsis proteins with a homologous zinc finger domain. Among these proteins was AtMC1, which we at that time completely



Nuria Coll (right) and Petra Epple (left)

neglected to study any further, as it seemed too different from LSD1 and some of the other LSD1-like proteins we were working on (LOLs; Epple et al., 2003). However, once the paper by Uren et al. was published in 2000, it was evident that AtMC1 belongs to the metacaspase family, distant relatives of animal caspases. In animals, caspases are cell death regulators essential for proper development and immune system function. Plant metacaspases surmised to control cell death based on their structural homology to animal caspases, but since their discovery in 2000, very few functional data existed assigning them a function in plant cell death. The possibility that AtMC1 acted as a cell death effector was very fascinating.

Another surprise came when we found that AtMC2, the closest homolog of AtMC1, had an anti-cell death function, antagonizing AtMC1. The lack of AtMC1 suppressed cell death in *lsd1* mutants. In contrast, the absence of AtMC2 in an *lsd1* background had the complete opposite effect: it enhanced and accelerated *lsd1* cell death. Most importantly, a triple mutant lacking LSD1 and both AtMC1 and AtMC2 exhibited essentially no cell death. The existence of a cell death network antagonistically regulated by these two metacaspases motivated us to start an in-depth study of their mode of action and their involvement in plant cell death after a pathogen attack.

Q: What is the composition of the group involved?

A: Petra Epple had a major role in this project. She is a senior post-doc in the lab with great expertise and started this project several years ago. I joined Jeff's lab in 2006 as a post-doc and started working closely with her as a team on this project. My interest in the role of metacaspases in cell death started during my Ph.D. in the ETH-Zürich and I found this project very attractive.

In addition, two undergraduate students have participated in the published research. **Charles Clover**, who worked with Petra before I joined and has since graduated from UNC medical school. **Andie Smidler** started working with me in 2009 and will be part of the group until she graduates this year.

The lab is a very diverse mixture of people from different nationalities, backgrounds, and expertise, who have been extremely helpful in the development of this project. We all work together in a big shared space, which encourages productive discussions between lab members. All the lab, including Jeff and his wife **Sarah Grant**, contribute to the great working atmosphere. We work hard but also have a lot of fun.

The work has also been fruit of a collaborative effort with **Frank van Breusegem** and his former post-doc **Nick Vercammen** at Ghent University, who provided us with some of the transgenic lines and with whom we are still collaborating.

Q: What were the big stumbling blocks? How did you manage to get them out of the way?

A: This project has not been an easy one. First, it was quite difficult to clone AtMC1 and AtMC2 under their native promoters. It was also challenging to study their expression patterns. Using quantitative PCR, we couldn't get any informative data. Finally, we used the promoter::GUS lines, which provided us with a clear picture of what was going on in terms of expression during cell death. AtMC1 expression was confined to a narrow zone around the cell death site front, whereas AtMC2 was more diffusely expressed in the surrounding areas, giving a spatial explanation of their antagonistic roles.

Another challenge came from the fact that AtMC1 and AtMC2 are proteases that are prone to self-digest and aggregate, so we had to be extremely careful when doing biochemistry. In fact, we have been unable to obtain stable and specific antibodies for any of them, and at that point, we had to create transgenic lines expressing epitope-tagged versions of the proteins under the control of their native promoters. It took quite a long time to obtain all the transgenic lines used in this study because we had to combine many different transgenes in several genetic backgrounds in order to get the tools needed to figure out how AtMC1 and AtMC2 function.

Q: Any great moments? Any downs? How did you get over them?

A: We got tough reviews from *Science* the first time we submitted our manuscript, but at the end they improved the quality of our work tremendously. One of the critiques was that we didn't show any pathology phenotype for the *atmc1* and *atmc2* mutants. When we tested them, it was great to observe that they confirmed all the previous findings: after infection, cell death was suppressed in *atmc1* mutants and enhanced in *atmc2* when compared to wild type.

At that time, we also found that, although all the mutants and transgenic lines had cell death phenotypes, they were as resistant as wild type to the different avirulent pathogens tested. This was very exciting, because it proves that cell death and effective disease resistance can be separated. So, we have found two proteins that specifically regulate cell death without affecting disease resistance.

Q: What's next?

A: One of the current focuses of our research is to try to identify AtMC1 and AtMC2 substrates, which we are doing still in collaboration with the group of Frank van Breusegem. We are also performing a mutagenesis screen to try to identify new components in the cell death pathway regulated by the AtMC1-AtMC2 tandem. Another aim is to find the interacting partners of AtMC1 and AtMC2 during cell death. We suspect they may be part of bigger protein complexes that form once cell

death is triggered, constituting a “deathosome” analogous to cell death complexes in animals that would allow signal amplification. We are also intrigued by the possible role of the remaining metacaspases of the family in the hypersensitive response.

With this work, we have added two new components to the cell death pathway triggered by the plant immune system and we have shown that they control similar processes as animal caspases. However, we are still far from getting the big picture of how programmed cell death works in plants, which is the chain of events downstream of pathogen recognition leading to cell death, and which mechanisms lead to the two separable responses integrated in the hypersensitive response, namely cell death and defense. ■

Have You Read the Top 10 Most Popular MPMI Articles in 2010?



1. Complete Genome Sequence of the Fire Blight Pathogen *Erwinia amylovora* CFBP 1430 and Comparison to Other *Erwinia* spp.
2. Understanding the Plant Immune System
3. Getting the Most from the Host: How Pathogens Force Plants to Cooperate in Disease
4. Methods to Study PAMP-Triggered Immunity Using Tomato and *Nicotiana benthamiana*
5. Intracellular Transport of Viruses and Their Components: Utilizing the Cytoskeleton and Membrane Highways
6. Phytoalexin Accumulation in the Interaction Between Rice and the Blast Fungus
7. Identification of *Nicotiana benthamiana* Genes Involved in Pathogen-Associated Molecular Pattern-Triggered Immunity
8. Emerging Viral Diseases of Tomato Crops
9. Effector-Triggered and Pathogen-Associated Molecular Pattern-Triggered Immunity Differentially Contribute to Basal Resistance to *Pseudomonas syringae*
10. All Hands on Deck—The Role of Chloroplasts, Endoplasmic Reticulum, and the Nucleus in Driving Plant Innate Immunity ■

Call for Papers! MPMI Focus Issue on Plant-Microbe Symbioses

Molecular Plant-Microbe Interactions (MPMI) invites original research manuscripts for a special focus issue on plant-microbe symbioses. This focus issue will be edited by **Krzysztof Szczyglowski**, Agriculture and Agri-Food Canada, London, Ontario, Canada, and **Michael Sadowsky**, University of Minnesota, St. Paul, MN, U.S.A.. Articles should focus on the molecular biology and molecular genetics/genomics of symbiotic interactions of microbes (prokaryotes and eukaryotes) with plants. The editors intend that this issue will bring added attention to areas of research that are of critical importance or that have shown significant progress in recent years. The target date for the special issue is November 2011.

Deadlines

- Submission of mini-reviews: May 1, 2011 (by personal invitation only)
- Submission of original research manuscripts: June 1, 2011
- Final acceptance of all relevant manuscripts: August 1, 2011

Submission Format

Authors should use the ScholarOne Manuscripts website for submission. For more details, please visit and upload submissions at <http://mc.manuscriptcentral.com/aps/mpmi> or e-mail sadowsky@umn.edu or Krzysztof.Szczyglowski@AGR.GC.CA with reference to the focus issue on plant-microbe symbioses. ■

What Can You Learn in 140 Characters?

Where do you get your most recent news on molecular plant-microbe interactions and related sciences? For some members, it comes in the form of a tweet. IS-MPMI's Twitter followers have continued to grow, as well as the amount and quality of scientific information posted on Twitter. Other organizations and individuals tweeting include premier institutions and the top researchers in our area of science.

Visit <http://twitter.com/ismpmi> to start connecting to other “tweeters” who share information on the latest breakthroughs and research on molecular science in general and in your unique area. If you prefer not to visit Twitter, the latest tweets from IS-MPMI are available on the IS-MPMI *net* homepage. ■



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November 2010, Volume 23, Number 11

CURRENT REVIEW—All Hands on Deck—The Role of Chloroplasts, Endoplasmic Reticulum, and the Nucleus in Driving Plant Innate Immunity.

CURRENT REVIEW—Intracellular Transport of Viruses and Their Components: Utilizing the Cytoskeleton and Membrane Highways.

CURRENT REVIEW—A Four-Partner Plant–Virus Interaction: Enemies Can Also Come from Within.

CURRENT REVIEW—Plasmodesmata: Gateways to Local and Systemic Virus Infection.

CURRENT REVIEW—Interactions Between Tobamovirus Replication Proteins and Cellular Factors: Their Impacts on Virus Multiplication.

A Host-Factor Interaction and Localization Map for a Plant-Adapted Rhabdovirus Implicates Cytoplasm-Tethered Transcription Activators in Cell-to-Cell Movement.

Effects of Amino-Acid Substitutions in the *Brome mosaic virus* Capsid Protein on RNA Encapsidation.

Enhanced Glutathione Metabolism Is Correlated with Sulfur-Induced Resistance in *Tobacco mosaic virus*-Infected Genetically Susceptible *Nicotiana tabacum* Plants.

Involvement of the P1 Cistron in Overcoming eIF4E-Mediated Recessive Resistance Against *Clover yellow vein virus* in Pea.

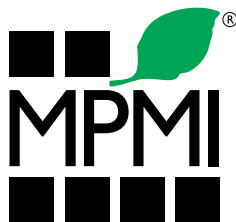
An h-Type Thioredoxin Functions in Tobacco Defense Responses to Two Species of Viruses and an Abiotic Oxidative Stress.

The N-Terminal Domain of PMTV TGB1 Movement Protein Is Required for Nucleolar Localization, Microtubule Association, and Long-Distance Movement.

Turnip mosaic virus (TuMV) Is Able to Use Alleles of Both *eIF4E* and *eIF(iso)4E* from Multiple Loci of the Diploid *Brassica rapa*.

Direct Interaction Between the *Rice yellow mottle virus* (RYMV) VPg and the Central Domain of the Rice *eIF(iso)4G1* Factor Correlates with Rice Susceptibility and RYMV Virulence.

Characterization of the Interactions Between *Cucumber mosaic virus* and *Potato virus Y* in Mixed Infections in Tomato.



December 2010, Volume 23, Number 12

CURRENT REVIEW—Understanding the Plant Immune System.

TECHNICAL ADVANCE—The Nod Factor-Independent Symbiotic Signaling Pathway: Development of *Agrobacterium rhizogenes*-Mediated Transformation for the Legume *Aeschynomene indica*.

Genetic Dissection of Basal Resistance to *Pseudomonas syringae* pv. *phaseolicola* in Accessions of *Arabidopsis*.

Conservation in Function of a SCAR/WAVE Component During Infection Thread and Root Hair Growth in *Medicago truncatula*.

A Yeast *STE11* Homologue *CoMEKK1* Is Essential for Pathogenesis-Related Morphogenesis in *Colletotrichum orbiculare*.

A Putative RNA-Binding Protein Positively Regulates Salicylic Acid-Mediated Immunity in *Arabidopsis*.

Limitation of Nocturnal ATP Import into Chloroplasts Seems to Affect Hormonal Crosstalk, Prime Defense, and Enhance Disease Resistance in *Arabidopsis thaliana*.

Lack of Galactose or Galacturonic Acid in *Bradyrhizobium japonicum* USDA 110 Exopolysaccharide Leads to Different Symbiotic Responses in Soybean.

A Combined ¹H Nuclear Magnetic Resonance and Electrospray Ionization–Mass Spectrometry Analysis to Understand the Basal Metabolism of Plant-Pathogenic *Fusarium* spp.

Morphological and Molecular Analyses of Host and Nonhost Interactions Involving Barley and Wheat and the Covered Smut Pathogen *Ustilago hordei*.

Stem Rust Spores Elicit Rapid RPG1 Phosphorylation.

Sinorhizobium meliloti Requires a Cobalamin-Dependent Ribonucleotide Reductase for Symbiosis With Its Plant Host.

January 2011, Volume 24, Number 1

CURRENT REVIEW—Perspectives on Remorin Proteins, Membrane Rafts, and Their Role During Plant–Microbe Interactions.

AVR1-CO39 Is a Predominant Locus Governing the Broad Avirulence of *Magnaporthe oryzae* 2539 on Cultivated Rice (*Oryza sativa* L.).

Functional Characterization of *fst1* in *Fusarium verticillioides* During Colonization of Maize Kernels. Cell-Penetrating Peptides Derived from Viral Capsid Proteins.

Mutations in the P3 Protein of *Soybean mosaic virus* G2 Isolates Determine Virulence on *Rsv4*-Genotype Soybean.

Biological Activity of the *Agrobacterium rhizogenes*-Derived *trnC* Gene of *Nicotiana tabacum* and Its Functional Relation to Other *plast* Genes.

The Transcription Factor FgStuAp Influences Spore Development, Pathogenicity, and Secondary Metabolism in *Fusarium graminearum*.

The Hypersensitive Induced Reaction and Leucine-Rich Repeat Proteins Regulate Plant Cell Death Associated with Disease and Plant Immunity.

TaDAD2, a Negative Regulator of Programmed Cell Death, Is Important for the Interaction Between Wheat and the Stripe Rust Fungus.

Comparative Analysis of the Capacity of Tombusvirus P22 and P19 Proteins to Function as Avirulence Determinants in *Nicotiana* species.

BAK1 Is Not a Target of the *Pseudomonas syringae* Effector AvrPto.

Genetic Basis for the Hierarchical Interaction Between *Tobamovirus* spp. and *L* Resistance Gene Alleles from Different Pepper Species.

A Novel Transcriptional Factor Important for Pathogenesis and Ascosporegenesis in *Fusarium graminearum*.

VdSNF1, the Sucrose Nonfermenting Protein Kinase Gene of *Verticillium dahliae*, Is Required for Virulence and Expression of Genes Involved in Cell-Wall Degradation.

Identification of Genes Involved in the Response of Banana to Crown Rot Disease.

February 2011, Volume 24, Number 2

The Role of Catalase-Peroxidase Secreted by *Magnaporthe oryzae* During Early Infection of Rice Cells.

Characterization of *Geminivirus* Resistance in an Accession of *Capsicum chinense* Jacq.

Callose Deposition: A Multifaceted Plant Defense Response.

Functional Analysis of the Asian Soybean Rust Resistance Pathway Mediated by *Rpp2*.

The Evolutionary History of *Beet necrotic yellow vein virus* Deduced from Genetic Variation, Geographical Origin and Spread, and the Breaking of Host Resistance.

Response to Environmental Stresses, Cell-wall Integrity, and Virulence Are Orchestrated Through the Calcineurin Pathway in *Ustilago hordei*.

Regulation and Symbiotic Role of *nirK* and *norC* Expression in *Rhizobium etli*.

Genomic and Evolutionary Features of the SPI-1 Type III Secretion System That Is Present in *Xanthomonas albilineans* but Is Not Essential for Xylem Colonization and Symptom Development of Sugarcane Leaf Scald.

Germinating Spore Exudates from Arbuscular Mycorrhizal Fungi: Molecular and Developmental Responses in Plants and Their Regulation by Ethylene.

The *Pseudomonas* Secondary Metabolite 2,4-Diacetylphloroglucinol Is a Signal Inducing Rhizoplane Expression of *Azospirillum* Genes Involved in Plant-Growth Promotion. ■



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People

In Memory



Adam Kondorosi

Adam Kondorosi, former board director and president-elect of our society and a member of the Hungarian Academy of Sciences and the Academia Europaea, died on January 21, 2011, at the age of 64 in Szeged, Hungary. We had been aware of his deteriorating health for six years and, lately, of his declining physical condition, but now, after the inevitable happened, we are appalled that he passed away. Fate

denied him the opportunity to return to the lab after stepping down from his administrative posts, or to see the recent recognition of his wife Eva, or the chance to see their daughter Fanny graduate, marry, and have children.

Adam took his inspiration for science from his family. He was a relative of the Nobel laureate **Richard Zsigmondy**, but his interest in biology came from his grandfather, **Bela Johan**, who was a great microbiologist, member of the Hungarian Academy of Sciences, and the first Rockefeller scholar from Hungary. It was Johan who dramatically improved the life prospects of Hungarian citizens by establishing the public health and epidemiology system, introducing compulsory vaccination against infectious diseases and initiating industrial production of vaccines, antibiotics, and vitamins. Nevertheless, he was set aside from public health work and even imprisoned, first by the Nazis, then by the Communists. Later, being the only one in Hungary who could produce penicillin, he was allowed to work in the fermentation departments of pharmaceutical companies until his age of 90. Adam, thus, became acquainted with microbiology from his childhood and studied biology at the Eötvös Lóránd University in Budapest. As an undergraduate student, he started his experimental work at the Institute of Genetics led by **Barna Gyórfy** during an era when the shadow of Lysenko still haunted geneticists in the communist countries. After this institute closed, Adam was recruited from the pool of the most talented young scientists to the newly formed Biological Research Center of the Hungarian Academy of Sciences in Szeged. It was here that he initiated research on symbiotic nitrogen-fixation, rapidly gaining international recognition for his fundamental research establishing a genetic system for *Sinorhizobium meliloti*. This led to the construction of its first genetic map, the discovery of the nodulation (*nod*) and the nitrogen fixation (*fix*, *nif*) genes on a megaplasmid, and the identification of the genes determining host-specific nodulation. After the description of the nodulation genes, he analyzed their regulation as well as the synthesis and function of the Nod proteins and their product, the Nod factors. In 1982, he was invited by **Jeff Schell** as a Humboldt Foundation visiting professor to initiate and direct *Rhizobium*-legume symbiosis research at the Max Planck Institute für Züchtungsforschung (Cologne,

Germany), which resulted in a fruitful cooperation and deep friendship. In 1988, Adam was recruited by the French National Centre for Scientific Research (CNRS) to establish a new research institute on plant molecular biology. He created and directed the Institute des Sciences Végétales (ISV, Gif-sur-Yvette, France) for over 13 years and made it a great success. At ISV, his interest turned toward the eukaryotic partner of the symbiosis. He initiated projects, for example, on the physiology of Nod factor signaling and the identification of plant genes required for nodulation and nitrogen fixation, first by biochemical methods, then with the emerging genomics technologies, making a major effort for establishing the *Medicago* genetic tools. As a result, we know a lot about the development of symbiotic cells, such as the endoreduplication of both the plant and bacterial genomes or plant peptides governing bacteroid differentiation. In addition, a large collection of insertion mutants in *M. truncatula* is available because Adam encouraged and supported the generation of this mutant platform at ISV. Despite premature interruption of his career by illness, Adam published more than 250 scientific articles, including several that were true benchmarks that led the field and which remind us of him when we cite the great results he achieved.

Adam was not only an exceptionally great scientist but also a true gentleman who coupled his fine intelligence to friendly and dignified behavior and to the care of others. Throughout his career, Adam was supported by his wife, Eva, an independent scientist in her own right and many saw their interaction as a symbiosis that greatly helped their research on symbiotic nitrogen fixation. This background made Adam a wonderful colleague, and a boss with great empathy. Though he was awarded by numerous top prizes (for example, the Max Planck Research Award, the Carlos J. Finlay Prize of UNESCO, the Széchenyi Prize), he was humble, having more pride in the success of his colleagues and students than his own, supporting us whoever and wherever we were. We were taught not only science, but also humanity and how one might be the smallest part of the machine, but be respected as much as the whole engine. Most of us who went to the ISV for discussions and experiments, visited Adam and Eva in their home and were warmly welcomed like a family member. Indeed, we have been. As a close-standing member of this extended family, I was asked by Eva to let the friends/colleagues know the sad news and received a number of wonderful messages about Adam. I believe that all of us will remember Adam as described by Andy Johnston, his friend of more than 30 years: "He was always a prince among men, both for his brilliance as a scientist, and, more importantly, as a human being of the very highest quality." God bless his soul.

Submitted by Attila Kereszt, BayGen Institute, Szeged, Hungary. ■

Welcome New Members

The following members joined IS-MPMI between September 1 and December 31, 2010.
Please join us in welcoming them to the society!

Angela Chaparro-Garcia

The Sainsbury Laboratory
Norwich, United Kingdom

Fouad Daayf

Univ of Manitoba
Winnipeg, MB, Canada

Xinran Du

Moscow, ID, U.S.A.

Jessie Fernandez

University of Nebraska-Lincoln
Lincoln, NE, U.S.A.

Rosa J. Gil

Univ of California
Davis, CA, U.S.A.

Susana K. Gomez

The University of Texas
Tyler, TX, U.S.A.

Ralph Hueckelhoven

Technische Univ Munchen
Freising, Germany

Hiroyuki Iyozumi

Shizuoka Prefectural Inst of Agriculture &
Forestry
Iwata, Shizuoka, Japan

Florian G. Jupe

Scottish Crop Research Inst
Invergowrie, Dundee, United Kingdom

Julietta Jupe

Univ of Dundee
Dundee, United Kingdom

Nora Ludwig

Utrecht Univ
Utrecht, the Netherlands

Shankar Manoharan

Madurai Kamaraj University
Madurai, India

Vania Passo

Univ of Warwick
Warwick, United Kingdom

Lisa Sanchez

Université de Reims Faculté des Sciences
Reims Cedex 2, France

Ken-Taro Sekine

Iwate Biotechnology Research Center
Kitakami-shi, Iwate, Japan

Remco Stam

Univ of Dundee
Dundee, United Kingdom

Melanie G. Tuffen

Univ of Nottingham
Loughborough, United Kingdom

Frank White

Kansas State University
Manhattan, KS, U.S.A.

Alicia Zelada

INGEBI
Buenos Aires, Argentina

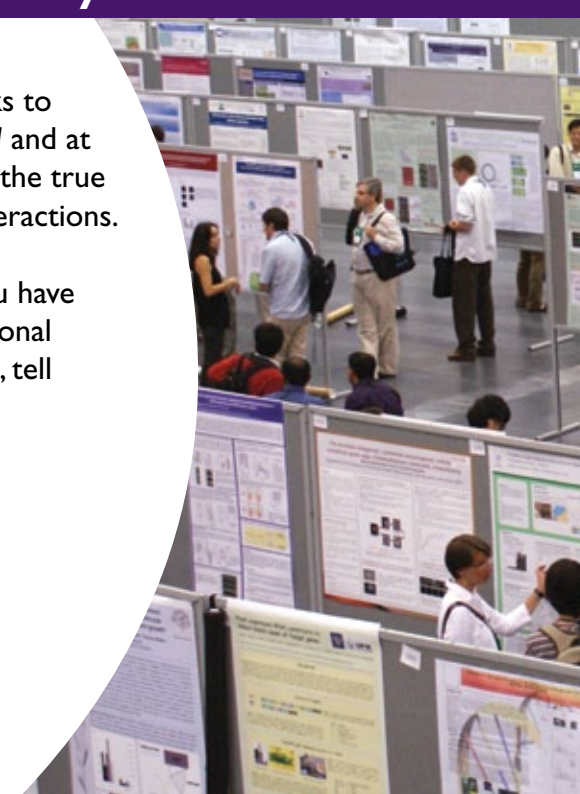
A Growing and Vibrant Community

IS-MPMI is a growing and vibrant community thanks to members like you. The research published in *MPMI* and at the congress continues our excellence. IS-MPMI is the true home for scientists in molecular plant-microbe interactions.

Share this unique community with others! If you have colleagues interested in attending the XV International Congress on Molecular Plant-Microbe Interactions, tell them to join to save on congress registration.



www.ismpminet.org



Employment

Post-Doctoral Positions at the Boyce Thompson Institute for Plant Research and NCSU-Mountain Horticultural Research and Education Center

Several post-doctoral positions are available for a new NSF-funded, multi-investigator project focused on leveraging genomics resources and wild species of tomato in order to identify, characterize, and use new sources of disease resistance. Preference will be given to candidates who have completed coursework in plant-microbe interactions, have research experience using a well-established experimental system, have a record of peer-reviewed publications, and are able to work as a productive member of a team of scientists. A Ph.D. degree is required in one of the following disciplines: biochemistry, bioinformatics, cell biology, genetics, microbiology, molecular biology, plant biology, plant breeding, or plant-microbe biology. Excellent writing and oral communication skills are required. Send a cover letter, a statement of research interests, and a CV, including the names of three references, to one (*only*) of the following individuals, depending on your research interests: Sorina Popescu (scp78@cornell.edu; protein microarrays), Zhangjun Fei (zf25@cornell.edu; bioinformatics), Gregory Martin (gbm7@cornell.edu; protein kinases), or Dilip Panthee (dilip_panthee@ncsu.edu; molecular plant breeding). Applications will be reviewed beginning January 25, 2011, and the positions will remain open until filled.

Post-Doctoral Positions in Molecular Mechanisms of Plant-Bacterial Interactions at the Boyce Thompson Institute for Plant Research

Post-doctoral positions are available for several projects focused on the interaction of tomato with *Pseudomonas syringae* or *Salmonella*. Projects are focused on AvrPto C-terminal virulence activity and its cognate R protein, Rpa (Yeam et al., *Plant J.* 61:16); AvrPtoB N-terminal virulence determinants and its E3 ligase domain (Rosebrock et al., *Nature* 448:370); characterization of tomato protein kinases involved in the immune response (Oh et al., *Plant Cell* 22:260); and the role of the tomato immune response in suppressing growth of food-borne pathogens. Candidates must have completed coursework in molecular host-microbe interactions, have research experience using a well-established experimental system, and have a record of significant peer-reviewed publications. A Ph.D. degree is required in biochemistry, genetics, microbiology, molecular biology, plant biology, plant pathology, or structural biology. Excellent writing and oral communication skills are required. For more information about the lab, see <http://bti.cornell.edu/GregoryMartin.php>. To apply, send a cover letter, a statement of research interests, and a CV, including the names of three references, to Gregory Martin by e-mail at gbm7@cornell.edu. Applications will be reviewed beginning January 10, 2011, and the positions will remain open until filled.

MAP Kinase Signal Transduction Pathways in Fungi: ARIADNE ITN Network—Marie Curie Ph.D. Positions

Thirteen Marie Curie research fellowships (three years) are open at one of the nine participants: Carlsberg Laboratory, Copenhagen, Denmark; Universidad de Córdoba, Córdoba, Spain; The University Court of the University of Aberdeen, Aberdeen, Scotland; The University of Exeter, Exeter, United Kingdom; Max Planck Institute for Terrestrial Microbiology, Marburg, Germany; Institute for Natural Products Research and Infection Biology, Hans Knoell Institut, Jena, Germany; Bayer CropScience SA, Lyon, France; Centro Nacional de Biotecnología, Madrid, Spain; and INRA, Paris, France. Marie Curie fellowships are offered with the objective to gain a Ph.D. degree. EU restrictions for Marie Curie fellows apply. Additionally, applicants must hold a master's degree or equivalent education. The degree should be in the area of biological sciences, medical sciences, or biotechnology. The applicant should have less than four years of research experience. The ARIADNE network aims at developing basic research projects on signal transduction in human and plant-pathogenic fungi to increase our knowledge on fungal pathogenesis and signal transduction. ARIADNE uses a comparative biological approach at the systems-biology level to identify and validate signaling target genes that play an essential causative role in fungal virulence on both plant and human hosts. Seven pathogenic species are studied, including the two major fungal pathogens of humans *Aspergillus fumigatus* and *Candida albicans*, as well as the plant pathogens *Fusarium oxysporum*, *Magnaporthe grisea*, *Mycosphaerella graminicola*, *Aschyta gossypii*, and *Ustilago maydis*. The central scientific and technological challenges for ARIADNE are to integrate parallel transcriptomics, proteomics, and chemical genetics for a comprehensive set of major fungal pathogens; identify evolutionarily conserved and species-specific signaling effectors that act as key players in fungal virulence; and validate these proteins as candidate targets for small molecule inhibitors. This groundbreaking scientific knowledge will facilitate the discovery of novel antifungals.

There is an open ARIADNE Ph.D. position at Bayer CropScience, Lyon, France. The ARIADNE project at Bioger aims at studying the MAP kinase pathway Mps1 of the fungal rice pathogen *Magnaporthe grisea* to identify the targets of this MAP kinase, which is required for infection. The project will integrate transcriptomics of existing conditional MAP kinase mutants and existing mutants of candidate downstream transcription factors (Swi4, Swi6, Rlm1), chemical genetics analysis of modified MAP kinase, and the comparative analysis of these different transcriptional regulatory networks. These analyses will lead to the identification of a comprehensive network of genes controlled by these MAP kinase pathways and will benefit from parallel studies in other fungi studied within ARIADNE. Contact labo mixt bayercropscience/cnrs at nathalie.poussereau@bayercropscience.com. ■

Date:
August 2 (Tue)
–6 (Sat), 2011

Abstract submission:
January 12 (Wed)
– April 6 (Wed), 2011

Venue:
Kyoto International
Conference Center
(Kyoto, Japan)

Chair:
Ko Shimamoto
(Nara Institute of Science and Technology)

Confirmed speakers

- Jeff Dangl (USA)
- Jean Dénarié (France)
- Maria Harrison (USA)
- Sheng Yang He (USA)
- Jonathan Jones (UK)
- Sophien Kamoun (UK)
- Gregory Martin (USA)
- Paul Schulze-Lefert (Germany)
- Brian J Staskawicz (USA)
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- Jian-min Zhou (China)



XV International Congress on Molecular Plant-Microbe Interactions

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