The Sixth International *Aspergillus* Meeting

"Asperfest"

March 15-17, 2009
Asilomar Conference Center

Organized by the
Aspergillus Genomes Research Policy Committee

Aspergillus Genomes Research Policy Group (AGRPG)

An *Aspergillus* Genomics workshop was held at the March 2003 Asilomar Fungal Genetics meeting. From discussions in that workshop it was obvious that our community needed to organize to fully exploit genomics resources. A provisional *Aspergillus* Genomes Research Policy Committee (AGRPC) was conscripted and charged with creating a structure for community-wide coordination and organizing an annual meeting. The First *Aspergillus* Meeting was held in Copenhagen, April 21, 2004, as a satellite meeting of the European Congress on Fungal Genetics-7. In addition to scientific presentations, bylaws were approved, community research directions were discussed and the 2004 AGPRC was elected. The name *Aspergillus* Genomes Research Policy Group was adopted for the community. The objectives of the AGRPG are: (1) Provision of an educational and discussion forum for issues pertaining to *Aspergillus* genomics, in this widest sense, for the various *Aspergillus* research communities; (2) Influencing grant making bodies and other institutions on behalf of the various *Aspergillus* research communities; (3) Coordinating research activities internationally, as and when required, to strengthen the science base of the *Aspergillus* genus. For more information on the activities of the AGRPG and other *Aspergillus* news see our homepage at FGSC (http://www.fgsc.net/Aspergillus/asperghome.html).

2008 AGRPC

Gerhard Braus (*A. nidulans*), 2008- 2011; Georg-August-University Goettingen, Germany; gbraus@gwdg.de
Gary Payne (*A. flavus*), 2008-2011; North Carolina State University, USA; gary_payne@ncsu.edu
Scott Baker (*A. niger*), 2006-09; Pacific Northwest National Laboratory, USA; scott.baker@pnl.gov
Masayuki Machida (*A. oryzae*), 2006-09; Nat'l Inst of Biosci and Human Tech, Japan; m.machida@aist.go.jp
Michelle Momany, Chair (*A. nidulans* and *A. fumigatus*), 2006-09; University of Georgia, USA; momany@plantbio.uga.edu
Arthur Ram (*A. niger*), 2006-09; Leiden University, The Netherlands; a.f.j.ram@biology.leidenuniv.nl
Dr. J. W. Bennett (*A. flavus* and *A. parisiticus*) 2007-2010; Department of Plant Biology & Pathology, Rutgers; profmycogirl@yahoo.com
Gustavo H. Goldman, (*A. nidulans* and *A. fumigatus*) 2007-2010; University de Sao Paulo, Brazil; ggoldman@usp.br
Jennifer R Wortman (*A. fumigatus*) 2007-2010; Univ. of Maryland School of Medicine; jwortman@som.umaryland.edu
Kevin McCluskey (Ex officio); Curator, Fungal Genetics Stock Center; mccluskeyk@umkc.edu

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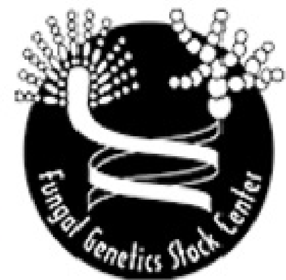

The Sixth International *Aspergillus* Meeting

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March 15-17, 2009
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All sessions in the Chapel

March 15, Sunday

3:00- 6:00 **Registration**

6:00 **Dinner**

7:00-9:00 **Welcome Reception—Sponsored by Verenium** **Chapel**

March 16, Monday

7:30-9:00 **Breakfast**

9:00-9:20 **Welcome, introductions and announcements**

AGRPC Chair: Michelle Momamy

Session I:
9:20-10:00 **Metabolic Pathways and the Metabolome**
Chair: Masayuki Machida

Jens Nielsen (Chalmers Biocenter, Chalmers University of Technology, Göteborg, Sweden)
"The metabolic networks of Aspergilli"

Jens C. Frisvad, Christian Rank and Thomas O. Larsen, (Department of Systems Biology, Technical University of Denmark)
"Aspergillus exometabolomics: a link to the perfect states?"

10:00-10:30 ***Coffee Break***

10:30-11:10 **Metabolic Pathways and the Metabolome (continued)**

Masanori Arita (Frontier Sciences, University of Tokyo)
"Metabolic Pathway Analysis using Wiki"

Berl Oakley and Clay Wang. (University of Kansas and University of Southern California)
"Secondary metabolism and metabolomics in *Aspergillus nidulans*"

Session II:
11:10-11:50 **Comparative Genomics and Databases**
Chair: Scott Baker

Jennifer Wortman and Gavin Sherlock (Univ. of Maryland and Stanford University)
"The *Aspergillus* Genome Database (AspGD), a curated database of *Aspergillus* gene, protein, and genomic sequence information for the fungal research community"

Jane Mabey Gilsenan (University Hospital of South Manchester, Manchester, UK)
"CADRE, *Aspergillus* Genomes and the *Aspergillus* Cloud"

12-1:00 ***Lunch***

Session III:

1:00-1:40 **Comparative Genomics and Databases (continued)**
Chair: Scott Baker

Mikael Andersen (Technical University of Denmark, Lyngby, Denmark)
"Systems biology- based analysis of the response of *Aspergillus niger* to ambient pH"

Kevin McCluskey (Fungal Genetics Stock Center, University of Missouri- Kansas City, MO, USA)
"Aspergillus at the FGSC"

Scott Baker (US DOE, Pacific Northwest National Laboratory, Richland, Washington, USA)
"A summary of *Aspergillus* genomics"

Session IV:

1:40-2:40 **Genetics and Cell Biology**
Chair: Gerhard Braus

Iran Malavazi (University of Sao Paulo, Brazil)
"Factors influencing the calcineurin-crzA pathway in the *Aspergillus fumigatus* pathogenesis and virulence."

Jo Strauss (University of Natural Resources and Applied Life Sciences, Boku Vienna, Austria)
"Epigenetics in Aspergillus"

S. Bergmann (Department of Molecular and Applied Microbiology, Leibniz Institute for Natural Product Research and Infection Biology - Hans-Knöll-Institute, Jena, Germany)
"Activation of silent gene clusters in *Aspergillus nidulans.*"

2:40-3:10 **Community Directions Discussion**
Chair: Michelle Momany

3:10-3:45 ***Coffee Break***

Session V:

3:45-4:45 **Talks selected from Abstracts**
Chair: Gustavo Goldman

Marcia Kress, Department of Molecular Microbiology and Genetics, Georg August University, Göttingen – Germany.
"The importance of E3 ubiquitin ligase SCF complexes for the development of the mold *Aspergillus nidulans"*

Srisombat Puttikamonkul (Department of Veterinary Molecular Biology, Montana State University, Bozeman, MT, USA)
"The Trehalose Pathway is critical for *Aspergillus fumigatus* virulence."

Natalie D. Fedorova, (J. Craig Venter Institute, Rockville, MD, USA)
"SMURF: a web tool for genomic mapping of secondary metabolite clusters"

Peter J. Punt, (TNO Quality of Life, P.O. Box 360, 3700 AJ Zeist, The Netherlands) "Characterization of the *Aspergillus niger prtT*, a unique regulator of extracellular protease encoding genes."

5:00-6:00 **Pontecorvo Lecture: Herbert N Arst, Imperial College, London**
"Calcium: A New Perspective"
Introduction: Jennifer Wortman

6:00-7:00 **Dinner**

7:00-10:00 **Posters and drinks**
Poster session and outstanding student poster -sponsored by Novozymes

March 17, Tuesday

7:30-9:00 **Breakfast**

Session VI:

9:00-10:00 **Medical Mycology**
Chair: Arthur Ram

Céline M. O'Gorman (University College Dublin, Ireland)
"Sex in Dublin City - Aspergillus fumigatus reveals its secret!"

Judith Behnsen (Leibniz Institute for Natural Product Research and Infection Biology, Jena, Germany)
"Interaction of *Aspergillus fumigatus* with the human complement system."

Gregory S. May (Division of Pathology and Laboratory Medicine, University of Texas MD Anderson Cancer Center Houston, TX USA)
"Fungal genetic response to the host."

10:00-10:15 **Elections**

10:15-10:30 **Coffee Break**

Session VII:
10:30-11:30 **What makes a pathogen? Chair: Joan Bennett**

Maren Klich (USDA/ARS, New Orleans, LA, USA)
"Medically important species of *Aspergillus"*

Robert Cramer (Montana State University, Bozeman, MT, USA)
"Aspergillus fumigatus metabolism: clues to mechanism of opportunistic pathogenesis"

Gary Payne (North Carolina State University, Raleigh, NC, USA)
"Possible virulence factors differentially expressed in *Aspergillus flavus* during infection"

11:30 **Announce election results and take any further discussion**
Announce winner of student poster award
--Sponsored by Novozymes

12:00-1:00 ***Lunch***

POSTER ABSTRACTS

BIOCHEMISTRY AND METABOLISM

1. Aspergillus exometabolomics: a link to the perfect states?

Jens C. Frisvad, Christian Rank and Thomas O. Larsen, Center for Microbial Biotechnology, Department of Systems Biology, Technical University of Denmark, Building 221, Søltofts Plads, DK-2800 Kgs. Lyngby, Denmark, e-mail: jcf@bio.dtu.dk

Aspergillus is a very diverse genus. Twelve perfect states of Aspergillus have been described: Chaetosartorya, Emericella, Eurotium (= Edyuillia), Fennellia, Hemicarpenteles, Hemisartorya, Neocarpenteles, Neopetromyces, Neosartorya, Petromyces, Sclerocleista, & Warcupiella. According to Peterson (2008) five genera: Hemicarpenteles, Sclerocleista and Warcupiella(and other members of the Trichocomaceae) are outside Aspergillus sensu stricto. Species hitherto regarded as having only the imperfect Aspergillus state, have recently been found to have a perfect state (e.g. Neosartorya fumigata and Petromyces parasiticus) and thus sclerotium or ascoma associated secondary metabolites are also potentially additions to the "full" profile of potential secondary metabolites anyone species can produce. Furthermore provisional annotation of full genome sequenced Aspergilli have shown that many more secondary metabolites are potential species specific metabolites than have already been discovered. We have analysed species of Aspergillus and Penicillium and associated teleomorphs for secondary metabolite profiles. Each teleomorph genus is a partially polythetic class regarding secondary metabolites, with few overlaps between teleomorphic genera of Aspergillus. This is in accordance with the large phylogenetic distance between some of these genera: For example Neosartorya (and Aspergillus section Fumigati) species produce a combination of fumigatins, viriditoxins, fumigaclavines, gliotoxins, trypacidins, fumitremorgins, fumiquinazolins, helvolic acids, fumagillins, neosartorin, cyclopiazonic acid while Petromyces (and Aspergillus section Flavi) produce kojic acids, aspergillic acids, cyclopiazonic acids, aflatoxins, asperfuran, oryzaechlorins, aflavinines, aflatrems, so comparing section Fumigati with section Flavi reveals that only cyclopiazonic acid is in common. Examples of the large differences in secondary metabolite combinations between Aspergillus sections will be presented in addition to a discussion on exometabolomics in general.

2. Global and local chemistry of the *Aspergillus flavus* group.

Christian Rank*, Jens Christian Frisvad, Thomas Ostenfeld Larsen, Center for Microbial Biotechnology, Department of Systems Biology, Technical University of Denmark, cr@bio.dtu.dk

Aspergillus flavus is the most thoroughly investigated fungi with respect to its chemistry. Many important metabolites have been reported from this species, of which the aflatoxins are the most important. The chemical diversity of the group has, however, never been comprehensively investigated and there are published results which do not fit the general chemical profile of *A. flavus*. To review the chemistry a global approach has been used to select isolates for local chemical analysis: Using direct injection of extracts into an ESI-MS, more than 300 isolates of *A. flavus*, *A. oryzae* and related species was compared for chemical profiles to be used for a chemotaxonomical clustering. From each cluster, selected isolates was investigated for unique chemistry. The results of this global and local chemical analysis will be presented with emphasis on important metabolic pathways and novel structures. *Student poster

3. Identification and Characterization of the Asperthecin Gene Cluster of *Aspergillus nidulans*.

Szewczyk, E., Chiang, Y.M., Oakley, C.E., Davidson, A.D., Wang, C.C. and Oakley, B.R.

The sequencing of *Aspergillus* genomes has revealed that the products of a large number of secondary metabolism pathways have not yet been identified. A likely reason is that most secondary metabolism gene clusters are expressed at very low levels under standard laboratory culture conditions. It is, therefore, important to discover conditions or regulatory factors that can induce the expression of these genes. We report that the deletion of *sumO*, the gene that encodes the small ubiquitin-like protein SUMO in *A. nidulans*, caused a dramatic increase in the production of the secondary metabolite asperthecin and a decrease in the synthesis of austinol/dehydroaustinol and sterigmatocystin. The overproduction of asperthecin in the *sumO* deletion mutant has allowed us, through a series of targeted deletions, to identify the genes required for asperthecin synthesis. The asperthecin biosynthesis genes are clustered and include genes encoding an iterative type I polyketide synthase, a hydrolase, and a monooxygenase. The identification of these genes allows us to propose a biosynthetic pathway for asperthecin. The project was supported by grants PO1GM084077 and RO1GM031837.

4. Cloning of Asparaginase gene, *ahrA*, from *Aspergillus nidulans*, and determination of specific enzyme activity using colorimetric methods.

Kyle Smith and Patricia M. Shaffer, Department of Chemistry and Biochemistry, University of San Diego, San Diego, CA, 92110, USA, kylesmith-08@sandiego.edu, shaffer@sandiego.edu.

L-Asparaginase is an aminohydrolase that catalyzes the hydrolysis of asparagine to aspartic acid and ammonia. The *ahrA* asparaginase gene (on chromosome VIII) produces a monomeric subunit that has 2 catalytic sites and a single tetrameric interface homology. Now, this gene was purified from previously transformed *E.coli*, and sequenced to verify the existence of *ahrA*. The plasmid DNA was used in a PCR reaction with new primers containing BamH1 and HindIII restriction endonuclease sites. The PCR product was ligated into an expression vector (pET-21a(+), Novagen) and was transformed into bacterial strain DH5alpha. A mini-prep and restriction digest was performed to verify the presence of the expected insert into the plasmid. The purified plasmid DNA was transformed into bacterial strain BL21(DE3) that was induced with IPTG to express the enzyme. A 6x His tag allowed for the purification on Ni chelating beads and cleavage from the beads using increasing concentrations of imidizole. The specific enzyme activity (the production of ammonia) was measured by a colorimetric assay using sodium pentacyanonitrosylferrate as the reactive agent. Asparaginase is used as part of the therapy and cure for childhood acute lymphoblastic leukemia (ALL). The success of this research may have medicinal significance. I am grateful to Graduate Women in Science and San Diego State University Research Foundation for funding and laboratory space.

5. Pigment production in response to deficiency in DNA repair

Jakob Blæsbjerg Nielsen, Christian Rank, Sameer Shamsuddin Mapari, Michael Lynge Nielsen, Thomas Ostenfeld Larsen, Jens Christian Frisvad & Uffe Hasbro Mortensen Center for Microbial Biotechnology, Department of Systems Biology, Technical University of Denmark, 2800 Kgs. Lyngby, Denmark jbn@bio.dtu.dk

Metabolites play an important role in many aspects of the fungal metabolism. We have observed that mutations in the homologous recombination (HR) apparatus in *Aspergillus nidulans*, which affect its ability to repair DNA damage such as double-strand breaks, result in the production of reddish pigment(s) coloring the fungal mycelium and surrounding media. The amount of pigment produced in the individual mutants of HR seems to correspond to the importance of the mutation in DNA repair. Colored fungal metabolites are often polyketides, a diverse group of secondary metabolites that has enormous interest due to their broad range of bioactivities in humans. By determining which compounds are involved in the response, we hope to understand how this fungus defends itself against DNA damage or replication stress via secondary metabolites.

6. The heme biosynthetic pathway in *Aspergillus niger*

A.C.W. Franken[1]*, B.C. Lokman[2] , A.F.J. Ram[1], C.A.M.J.J. van den Hondel[1,2], S. de Weert[1] 1) Institute of Biology Leiden, Leiden University, Molecular Microbiology, Kluyver Centre for Genomics of Industrial Fermentation, Wassenaarseweg 64, 2333 AL Leiden, The Netherlands, 2) HAN Biocentre, Laan van Scheut 2, 6525 EM Nijmegen, The Netherlands,

The incorporation of heme as a cofactor, is a putative limiting factor in the overproduction of heme-containing fungal peroxidases in *Aspergillus* species. Addition of hemin to growth medium has been reported to improve the production of peroxidase. However, hemin uptake and the effect of hemin addition on the transcriptional regulation of the heme-biosynthesis pathway genes have not been studied in *Aspergillus*. To gain more insight into the heme biosynthesis pathway, the genes encoding the eight different enzymes in the pathway were identified in the A. niger genome. Individual deletion of four genes in the pathway (*hemA, hemB, hemF* or *hemH*) showed that all four are essential. In contrast to the other deletion strains, growth of the ?*hemA* mutant could be restored by the supplementation of 50 µM 5'-aminolevulinic acid (ALA). Supplementation with hemin alone did not restore growth, but supplying limiting amounts of ALA together with hemin fully restored growth of the ?*hemA* strain and indicates either enhanced uptake of ALA in the presence of hemin, or uptake of hemin itself. ?*hemB*, ?*hemF* and ?*hemH* strains could only be propagated as heterokaryons. The ?*hemF* and ?*hemH* heterokaryons show auto-fluorescence upon exposure to UV-light due to the accumulation of specific porphyrins. Possible regulation at the transcriptional level of the four heme pathway genes mentioned above under various heme-limiting conditions might provide more information about the regulation of the pathway, and is currently in progress. *Student poster

7. AflJ may mediate stability of proteins necessary for aflatoxin biosynthesis

Kenneth C. Ehrlich[1], Brian Mack[2], Jeffrey W. Cary[1], and Shubha Kale Ireland[2], Southern Regional Research Center/ARS/USDA1 and Xavier University2, New Orleans LA Previous studies found that the protein AflJ is required for aflatoxin biosynthesis. AflJ is predicted to have at least three membrane spanning domains and a C-terminal microbody targeting signal. Interaction between AflR and AflJ was shown to be necessary for aflatoxin biosynthesis. In knockout transformants of *aflJ* in *A. parasiticus* or *A. flavus*, transcripts of biosynthesis genes were detected suggesting that AflJ acts post- transcriptionally. We now report, based on a yeast two-hybrid assay, that AflJ interacts with fungal homologs of subunits 5 (CsnE) and 6 (CsnF) of the COP9 signalosome complex, as well as a putative NEDD8 activating enzyme (UbaC). The latter homolog is predicted to activate ubiquitination while the former may affect ubiquitin-mediated protein degradation. The association of AflJ with components of the protein degradation pathway suggests a plausible mechanism for its action consistent with previously reported observations. AflJ may specifically prevent degradation of enzymes critical to aflatoxin biosynthesis. Thus, in *aflR* knockout transformants, the transcripts of aflatoxin biosynthesis genes are still made but some of the proteins may be swiftly degraded, thereby preventing buildup of sufficient enzyme pools to carry out the necessary biosynthetic steps.

8. Importance of the *acuM* gene for growth of *Aspergillus fumigatus* on non-fermentable carbon sources and its impact on virulence.

Matthias Brock and Ilse D. Jacobsen Hans-Knoell Institute, Beutenbergstr. 11a, 07745 Jena, Germany; E-mail: Matthias.brock@hki-jena.de

The *acuM* gene from *Aspergillus nidulans* was previously shown to be essential for growth on non- fermentable carbon sources by regulating the expression of gluconeogenic genes. Here we describe the construction of an *A. fumigatus* mutant containing a partial deletion of the *acuM* gene. The mutants were phenotypically characterised for their growth behaviour on different glycolytic and gluconeogenic carbon sources. As expected, the mutant was unable to grow on olive oil, acetate, ethanol and glutamate. Intermediate growth was observed on glycerol. Interestingly, the mutant was also unable to grow on albumin, whereas only a slight growth defect was observed on casamino acids and no phenotype was visible during growth on peptone. Even more, the ability to grow on albumin was restored by the addition of low amounts of glucose (2 – 5 mM) to the growth medium. The mutant was subsequently tested in a murine infection model using cortisone acetate treated mice. Survival curves revealed no significant differences between wild type and mutant and also the lesions within the lung tissues were highly similar. These results imply that non-hydrolysed proteins (such as albumin) cannot provide the sole carbon source during *A. fumigatus* pathogenesis in corticosteroid treated mice. It rather points to the (co-)metabolism of glucose or peptide fragments, which may represent major growth substituting nutrients during the infection process.

CELL BIOLOGY

9. Distinct and overlapping functions of the Rho-like GTPases RacA and CftA in the life cycle of *Aspergillus niger*.

Min-Jin Kwon, Eelke D. Roos, Mark Arentshorst, Cees A.M.J.J. van den Hondel and Arthur F.J. Ram Institute of Biology Leiden, Leiden University, Molecular Microbiology, Kluyver Centre for Genomics of Industrial Fermentation, Wassenaarseweg 64, 2333 AL Leiden, The Netherlands.

In all eukaryotic cells, Rho-related GTPases (Rho, Rac and Cdc42) are involved in controlling and organizing actin cytoskeleton and thereby controlling polarized cell growth. The genome of *A. niger* revealed the presence of four Rho-like GTPases (RhoA-D) and single copy Rac1 and CDC forty-two homologs, named RacA and CftA. To asses functions to the different Rho-like GTPases, null mutants have been generated. Of all the disruptants, the *racA* mutant displayed to most severe growth phenotype and was characterized by a hyperbranching phenotype. In addition, deletion of *racA*, *cftA*, *rhoB* and *rhoD* resulted in reduced formation of conidia. In general, the growth and morphology of the different disruption strains not severely affected, which could be explained by redundancy. The overlapping function of RacA and CftA was shown by trying to obtain a *racA*, *cftA* double deletion mutant. As all primary transformants containing both the *racA* and the *cftA* deletion were all heterokaryotic we conclude that the double mutant is lethal. The phenotypic consequences of the loss of *racA* or *cftA* on the actin cytoskeleton in germinating spores have been studied in detail. The localization of actin was examined using immunofluorescence in both the wild type and the *racA* or *cftA* deletion mutants. Actin patches in the *racA* mutant showed a severe condensation at the extreme apex at the hyphal tip while both in the *cftA* mutant and wild type strain a gradient of actin patches towards the tip was observed.

10. GDP-mannose transporter contributes to polarity establishment in *Aspergillus nidulans*.

Loretta Jackson-Hayes, Laura R. Johnson, Barbara S. Gordon, Terry W. Hill, Darlene M. Loprete. Departments of Biology and Chemistry, Rhodes College, 2000 N. Parkway, Memphis, TN 38112, jacksonhayesl@rhodes.edu

GDP-mannose transporters (Gmt) carry nucleotide sugars from the cytosol across the Golgi apparatus membrane in various eukaryotic organisms including plants and a variety of fungi. Some fungal species including *Saccharomyces cerevisiae* express a single Gmt, while others including *A. nidulans* express two orthologues (GmtA and GmtB) whose individual roles have not been elucidated. GmtA displays a punctate pattern of distribution indicative of localization within the Golgi apparatus. Here we show that GmtB localization is congruent with GmtA in mature hyphae. Real-time reverse transcriptase PCR shows a gradual decrease in GmtA expression during the first 12 hours of germination while GmtB expression remains high and unchanged during this period. Depletion of GmtB expression by the glucose repressible alcA promoter causes abnormalities in polarized growth. Taken together these observations suggest that GmtB plays an important role in establishment of polarized growth. Although GmtA and GmtB reside within the same Golgi compartments they appear to perform independent functions at different developmental stages. Funded by Research Corporation, the Merck/AAAS Undergraduate Science Research Program and NSF RUI

11. The *Aspergillus nidulans* orthologue of protein kinase C (PkcA) localizes to forming septa in a formin- and actin-dependent manner.

Terry W. Hill, Darlene M. Loprete, Loretta Jackson-Hayes, John L. Musgrove, Erinn A. Ogburn, Bianca D. Cathey[1], Jessica A. Lemon[2]. Departments of Biology and Chemistry, Rhodes College, Memphis TN USA and [1]Department of Biology, Rust College, Holly Springs MS USA, [2]Department of Biology, Tougaloo College, Jackson, MS USA.

The *calC*2 mutation in the *Aspergillus nidulans* orthologue of protein kinase C (PkcA) causes hypersensitivity to Calcofluor White along with other drug sensitivities which indicate a defect in cell wall integrity. Placing the PkcA gene under the control of the regulatable AlcA(p) promoter severely compromises hyphal morphogenesis and resistance to wall damaging drugs under repressed conditions. A PkcA::GFP chimera localizes to hyphal apices and growing septa, as well as to the conidiogenous apices of phialides. These observations indicate that PkcA plays roles in polarized wall growth, in cytokinesis, and in the cell wall integrity pathway. In order to investigate the mechanisms of PkcA targeting to hyphal tips and points of septation, we exposed a strain expressing PkcA::GFP under its native promoter to the F-actin disrupting agent cytochalasin A. After 15 minutes exposure, PkcA failed to localize to cortical rings (marking sites of incipient septation) or to hyphal apices. Instead, PkcA localized only to scattered cytoplasmic patches, possibly representing detachment of already formed contractile rings. In addition, we have generated a strain expressing Pkc::GFP in a temperature-sensitive *sepA*1 background (*A. nidulans* formin orthologue). When grown for several hours under restrictive conditions, PkcA still localized in the normal manner to hyphal apices, but no cortical rings were formed. This indicates that, even though PkcA and SepA occur together at both hyphal apices and septation sites, the relationship of PkcA to SepA is different at the two locations.

12. Analysis of SONB, a nuclear pore complex protein involved in the DNA damage response.

Jennifer R. Larson and Stephen A. Osmani. Ohio State University, Columbus, Ohio. larson.315@osu.edu, osmani.2@osu.edu

Nuclear pore complexes (NPCs) are composed of a set of proteins called nucleoporins (Nups) and allow the transport of proteins and nucleic acids across the nuclear envelope. Some nucleoporins appear to have additional functions aside from their role in transport. For example, components of the NUP84 subcomplex have previously been implicated in the response to DNA damage. NPCs have been associated with telomere anchoring/silencing, and telomere anchoring to the nuclear periphery is essential for efficient repair of DNA damage in subtelomeric regions. $\mathrm{SONB}^{\mathrm{Nup98}}$ is a conserved component of nuclear pore complexes and is essential in *Aspergillus nidulans*. The *sonB1* allele was identified in a screen for suppressors of the temperature sensitive *nimA1* allele of the mitotic NIMA kinase. Importantly, subsequent genetic analyses indicated SONB has a role in a novel response to DNA damage. *sonB1* mutant cells are hypersensitive to DNA damaging agents and show synthetic genetic interactions with $scaA^{\mathrm{Nbs1}}$, a breast cancer susceptibility gene. Mutations in NBS1 cause an increased sensitivity to DNA damage and shortened telomeres. We have extended these studies and found that *sonB1* shows synthetic genetic interactions with the same components of the NUP84 subcomplex previously implicated in the DNA damage response in yeast. We will examine the role of SONB in telomere anchoring and activation of a DNA damage checkpoint, and partially map out this novel DNA damage response pathway that involves components of the nuclear pore complex.

13. Two Golgi apparatus COG proteins are important to polarity establishment and maintenance in *Aspergillus nidulans.*

Sara Gremillion[1], Amira El-Ganiny[2], Darlene Loprete[1], Terry Hill[1], Susan Kaminskyj[2], and Steven Harris[3]. [1]Departments of Biology and Chemistry, Rhodes College, 2000 N. Parkway, Memphis, TN 38112, USA. [2]Department of Biology, University of Saskatchewan, Saskatoon, Saskatchewan, Canada. [3]University of Nebraska-Lincoln, Plant Science Initiative, Lincoln, NE 6858-0660, USA. gremillions@rhodes.edu

*swoP*1 (swollen cell) and *podB*1 (polarity defective) mutations in *Aspergillus nidulans* interfere with establishment and maintenance of polarity. At restrictive temperatures, conidia of *swoP*1 may swell to approximately 1.5 times the normal diameter, produce abnormally wide hyphae and/or establish multiple points of polarity, which grow isotropically before arrest. Conidia of *podB*1 never establish polarity at restrictive temperatures. Cell walls of both strains are as thick as 1 µm (TEM) compared to ca. 0.04 µm at 28C, and the cytoplasm contains numerous irregular membrane structures. Genes complementing the mutations of *swoP*1 and *podB*1 have strong sequence homology to COG4 (AN7462) and COG2 (AN8226), respectively. Sequencing of the respective loci reveals point mutations causing truncations near the C-terminus. In mammals and yeast, COG2 and COG4 are part of a multi-protein structure called the COG (conserved oligomeric Golgi) complex associated with retrograde transport within the Golgi apparatus. To provide evidence for a COG function of AN7462 and AN8226, we used a high-copy AMA1 plasmid to overexpress the COG homologues of *A. nidulans* COG1-4, COG6, COG7, as well as the functionally-related homologues YPT1 (Rab GTPase) and HOC1 (mannosyl transferase). High copy expression of COG2 corrected the *swoP*1 phenotype while the remaining proteins did not. High copy expression of COG3 and COG4 corrected the *podB*1 phenotype while the remaining proteins did not. Collectively, these results support a conclusion that the SwoP and PodB proteins function in a common complex including a predicted COG3, which is consistent with the structure of the “A lobe” of yeast and mammalian COG models.

14. The mitotic NIMA kinase shows synthetic lethal interactions with genes potentially involved in septation and cell tip growth in *Aspergillus nidulans*.

Meera Govindaraghavan[1]*, Sarah Lea McGuire[2] and Stephen A. Osmani[1] [1]Department of Molecular Genetics, The Ohio State University, Columbus, OH [2]Department of Biology, Millsaps College, Jackson, MS

In *Aspergillus nidulans* the transition from G2 into mitosis requires the NIMA kinase. In yeast, a synthetic lethal screen using *KIN3*, the non essential NIMA orthologue, identified twelve genes, not involved with the cell cycle (S.L. McGuire, unpublished). Since *KIN3* does not have mitotic functions, if these synthetic interactions where conserved in *A. nidulans* this might reveal additional non-mitotic roles for NIMA. We therefore determined if NIMA interacts with the orthologues of the yeast genes that are synthetically lethal with *KIN3*. Of the ten orthologues identified, synthetic lethal/sick temperature sensitive interactions with *nimA7* were identified for only four, An-*swd1*, An-*vps23*, An-*vps25* and An-*ypt7*. We characterized the nuclear morphology and septation of the double mutants which revealed temperature sensitive synthetic growth defects linked to abnormal branching, septation and DNA segregation. Because we additionally discovered a defect in the tip morphology of $nimA^{ts}$ cells, collectively the results indicate that NIMA plays roles in septation and tip growth. This hypothesis is strengthened by the observation that NIMA localizes to septa and cell tips (C. De-Souza, K-F. Shen and S.A. Osmani unpublished). Further characterization of these interactions will lead to a better understanding of these previously unrealized non mitotic functions of NIMA. *Student poster

15. Construction of autonomously replicating vectors for complementation analysis of disruption mutants in a *ku70* deletion background in *Aspergillus niger*.

Neuza Carvalho*, Mark Arentshorst, Cees van den Hondel and Arthur Ram. Institute of Biology Leiden, Leiden University, Molecular Microbiology, Kluyver Centre for Genomics of Industrial Fermentation, Wassenaarseweg 64, 2333 AL Leiden, The Netherlands.

Mutants with a defective Non-Homologous-End-Joining (NHEJ) pathway are a very powerful tool for fungal genetic engineering. Several reports over the last few years have shown that mutants in the NHEJ–pathway (ku70/ku80 mutants) are very efficient recipients for gene targeting and achieve homologous targeting efficiencies up to 100%. To prove that a phenotype is associated with the deletion of a certain gene, the gene of interest is transformed back to the gene deletion strain which will ectopically integrate into the genome. However, phenotype complementation becomes difficult in a *ku70* deletion background because ectopic integration frequencies are low and the gene will preferably integrate via homologous recombination, thereby replacing again the disrupted gene. One way to circumvent this problem is to clone the gene for complementation into an autonomously replicating plasmid containing a selection marker. Under selective pressure the plasmid is maintained, giving the wild type phenotype; once the selective pressure is removed, the plasmid is gradually lost and the mutant phenotype is again observed. We have constructed autonomously replicating vectors containing either the *pyrG* or Hygromycine as selection markers and a unique *NotI* site for easy cloning of complementing genes. These vectors have been successfully used for complementation analysis of gene deletion mutants in *A. niger*. *Student poster

16. Complementation of the *Aspergillus nidulans wetA* mutant with the apparent *Neurospora crassa* ortholog NCU01033

Srijana Upadhyay, Daniel J. Ebbole, Heather H. Wilkinson, and Brian D. Shaw Department of Plant Pathology and Microbiology, Program for the Biology of Filamentous Fungi, Texas A&M University, College Station TX 77843

The asexual life cycle of *Aspergillus nidulans* begins and ends with the production of uninucleate haploid spores called conidia. *A. nidulans* genes *brlA*, *abaA* and *wetA* are considered key regulators of conidiophore development and conidium maturation. In *A. nidulans wetA* is required for synthesis of cell wall layers that make spores impermeable. The *wetA* mutant of *A. nidulans* displays defective conidia that fail to mature and later undergo lysis. The *Neurospora crassa* deletion mutant of the *wetA* ortholog (NCU01033) does not show any defect in conidiation. This study assesses the ability of these genes to cross complement. A chimeric fusion construct with the *A. nidulans wetA* promoter and the *N. crassa* NCU01033 coding sequence restored the conidiation defect of the *A. nidulans wetA* mutant. Complementation analysis of *wetA* mutant with *N. crassa* NCU01033 expressed by its endogenous promoter is ongoing.

17. Comparitive analysis of *fluG* function in *Aspergillus nidulans* and *Neurospora crassa*

Srijana Upadhyay, Daniel J. Ebbole, Heather H. Wilkinson, and Brian D. Shaw Department of Plant Pathology and Microbiology, Program for the Biology of Filamentous Fungi, Texas A&M University, College Station TX 77843

Asexual reproduction, conidiation, is an important phase in the life cycle of fungi. In *Aspergillus nidulans* the process initiates with the emergence of an aerial conidiophore stalk, followed by the development of an elaborate multicellular conidiophore that produces uninucleate haploid spores called conidia. Genetic regulation of conidiation is well characterized in *A. nidulans*, but comparative analysis of the function of these genes in other fungi is limited. In *A. nidulans fluG* is involved in generation of an extracellular signal required for activation of asexual development. In *Neurospora crassa*, deletion of the *fluG* ortholog (NCU04264) produced a mutant that displays no detectable phenotype. Surprisingly we found that complementation with the NCU04264 gene restored the conidiation defect of the *A. nidulans fluG* mutant. Overexpresion of NCU04264 in *A. nidulans* via the *alcA* promoter also conferred the ability to initiate conidiation in submerged culture, a phenotype similar to that exhibited by the *A. nidulans* overexpression strain in published studies. Our results demonstrate conservation of biochemical function of the *fluG* product between the two species, even though the *fluG* ortholog apparently is not required for conidiation in *N. crassa*.

18. Cross-species complementation of *Aspergillus nidulans* conidiation regulators with orthologs from *Neurospora crassa*

Dawoon Chung*, Srijana Upadhyay, Daniel J. Ebbole, Heather H. Wilkinson, and Brian D. Shaw Department of Plant Pathology and Microbiology, Texas A&M University, College Station TX 77843

Conidiation is a crucial process for survival and dissemination of filamentous fungi. To study functional conservation of general regulators for conidiation, we are comparing two model systems, *Aspergillus nidulans* and *Neurospora crassa*. Three *A. nidulans* mutants (*abaA, medA,* and *stuA*) were tested by transforming with the orthologs from *N. crassa*. The *abaA* ortholog of *N. crassa* has little sequence similarity (34%), but is likely the ortholog of *A. nidulans abaA* with microsynteny. In contrast, *N. crassa acon-3* and *ama-1* have higher similarity with *A. nidulans medA* and *stuA* (51% and 60%). Expression of the *N. crassa abaA* orthologs by its endogenous promoter or via an *alcA* promoter in the *A. nidulans abaA* mutant did not complement the developmental phenotypes. Over-expressed *acon-3* and *ama-1* via *alcA* promoter partially complemented conidiophore morphology of the *A. nidulans* mutants. Expression of *acon-3* and *ama-1* by their native promoters fully complemented conidiophore morphology and conidiation of *A. nidulans* mutants. GFP- labeled AbaA, MedA and StuA localized in nuclei in *A. nidulans* live cells. These results suggest the function of conidiation regulators is both conserved (MedA and StuA) and diverged (AbaA) in two filamentous fungi. *Student poster

19. A putative eisosomal protein, SurA is involved in development of *A. nidulans*

Dawoon Chung*, and Brian D. Shaw Program for the Biology of Filamentous Fungi Department of Plant Pathology and Microbiology, Texas A&M University, College Station TX 77843

Eisosomes are immobile protein complexes at the plasma membrane, which mark sites of endocytosis in *Saccharomyces cerevisiae*. Sur7, Pil1 and Lsp1 are components of eisosomes. Sur7 is also a multicopy suppressor of mutations in *rvs167* that encodes an actin binding protein. Deletion of *sur7* altered sphingolipid metabolism and ascospore production in yeast. Sequence analysis showed that *A. nidulans* had one putative ortholog of Sur7 (27% identity and 47% similarity to Sur7 in *S. cerevisiae*). We generated a deletion mutant of *surA*, and this mutant displayed radial growth defect that was more severe under a restrictive temperature. In addition, the *surA* mutant showed reduced production of asexual spores, but increased sexual development including Hülle cells and cleistothecia. FM4-64 uptake and filipin staining did not exhibit significant differences between the *surA* knockout mutant and wild type when they were grown at a permissive temperature. Characterization of the mutant at a restrictive temperature is under way. A SurA::GFP fusion protein localized at immobile cortical patches consistent with localization in yeast. Our results suggest that a putative component of endocytic eisosome is associated with development of *A. nidulans*. *Student poster

20. Assessing the roles of striatin orthologs in fungal growth, development and virulence

Chih-Li Wang*, Won-Bo Shim, and Brian D. Shaw Department of Plant Pathology and Microbiology, Program for the Biology of Filamentous Fungi, Texas A&M University, College Station, TX 77843-2132, USA

Proteins of the striatin family contain a caveolin binding domain, a coiled-coil motif, and a calmodulin binding domain in the N-terminus and a WD40 repeat domain in the C-terminus. Three members of the striatin family, striatin, SG2NA and zinedin, have been identified in multicellular animals while filamentous fungi encode only one homolog. In mammals, they are mainly expressed in neuronal somatodendrites and have been characterized as cytosolic and membrane-bound proteins which play roles in signal transduction and vesicular trafficking. The striatin orthologs in filamentous fungi are associated with virulence of maize stalk rot and perithecium development in *Fusarium verticillioides* (*Fvfsr1*), and *F. graminearum* (*Fgfsr1*). In *Sordaria macrospora*, PRO11 is membrane bound and associated with sexual development. We generated *Cgfsr1* deletion mutant in *Colletotrichum graminicola*, the maize stalk rot and leaf anthracnose pathogen, to characterize its roles. The mutant showed reduced growth and reduced conidiation. The virulence of the mutant will be discussed. Additionally, the *fsrA* deletion mutant in *Aspergillus nidulans* also showed restricted colony growth with red pigment accumulation and altered sexual development. The subcellular localization of a fsrA::GFP fusion in *Aspergillus nidulans* was consistent with localization to the endoplasmic reticulum and the nuclear envelope. The detailed characters of mutants and further experimental identification of the subcellular localization will be disscussed. *Student poster

21. The design of genetic screens to study the molecular basis of microtubule-based cargo transport

Kaeling Tan, Michael Wu and Samara Reck-Peterson Department of Cell Biology, Harvard Medical School, Seeley G Mudd Building, Rm529, 240 Longwood Ave,Boston, MA, 02115

In eukaryotic cells, molecular motors transport cellular material, such as organelles, chromosomes and mRNAs, by moving along the microtubule (MT) cytoskeleton. MT motors fall into two classes: dynein, which transports cargo towards the MT minus-end (from the cell periphery towards the nucleus) and kinesin, which generally moves cargo in the opposite direction. While much is known about how these motors work in vitro, much less is known about how these motors move cargo in cells and how these events are regulated both spatially and temporally. We are designing genetic screens in *Aspergillus nidulans* to identify factors that are required for MT-based transport. *A. nidulans* is an ideal model system for studying this problem because it has four MT-based motors that function in transport, in addition to having a sequenced genome, a high efficiency of homologous recombination, and a life-cycle that is suitable for high-throughput screening. As a basis for our screens we are identifying all organellar cargo transported by each of the four motors; we expect cargo to mislocalize in the background of motor deletions. As a starting point for identifying cargo we are tagging all of the Rab GTPases in the *A. nidulans* genome with EGFP in strains lacking each of the four motors. After identifying organelle mis-distribution phenotypes we will perform genome-wide high-throughput microscopy-based screens to identify novel components required for dynein and kinesin-based transport. We expect to identify new conserved paradigms about the mechanism of cargo transport as well as some processes that will be specific to the *Aspergilli*, which could serve as therapeutic targets for fungal infections.

22. A split CandA regulates ubiquitin ligases in *Aspergillus nidulans*

Elke U. Schwier[1], Martin Christmann[1], Kerstin Helmstaedt[1], Krystyna Nahlik[1], Mieke Westermann[2], Stephanie Grond[2], Silke Busch[1], Gerhard H. Braus[1] Georg-August-Universität Göttingen, [1]Institut für Mikrobiologie und Genetik, [2]Institut für Organische und Biomolekulare Chemie, Göttingen, Germany, khelmst@gwdg.de Cullins are a class of scaffold proteins, which are part of the SCF (Skp1/Cullin/F-box protein) ubiquitin ligases. The activity of these ligases is positively regulated by the neddylation of the cullin while binding of the cullin to Cand1 (cullin-associated Nedd8-dissociated protein 1) favors the disassembly and impairs the reassembly of the complex. Cand1 blocks the neddylation site in the C-terminal part of cullin as well as the Skp1 adaptor binding site in the N-terminal region of cullin. In *Aspergillus nidulans*, the gene encoding the putative homolog of Cand1 is split into two genes (*candA-N* and *candA-C*) encoding two proteins each with one cullin binding site. Deletion of either gene causes an impairment of the asexual cycle, a block of the sexual cycle and the production of brownish-red pigments. These defects can be complemented by a *candA-N::C* fusion construct. Both CandA proteins interact with each other, but only CandA-C associates with cullins in a yeast two-hybrid assay indicating that CandA-N binding to cullin is mediated by CandA-C. *In vivo*, CandA-N requires CandA-C for transport into the nucleus. Binding of only CandA-C to cullin might reflect transient states in the disassembly/reassembly cycle of SCF ubiquitin ligases which could be adopted independently of cullin deneddylation.

23. The importance of E3 ubiquitin ligase scf complexes for the development of the mold *Aspergillus nidulans*

Marcia Kress, Rebekka Harting, Gerhard H. Braus Department of Molecular Microbiology and Genetics, Georg August University, Göttingen – Germany

Targetting to the proteasome often requires the phosphorylation of substrates and subsequent ubiquitination by a pathway involving the enzymes E1 activating, E2 conjugating and several types of E3 ligases. The largest class of E3 ligases is the cullin RING ligase (CRL), which the scaffold protein, CulA, is the target of Nedd8/RubA, an Ub like protein. We have previously shown that the deneddylation is necessary for fungal development (Busch et al., 2003; 2007). For further exploration of the role of RubA during fungal development, we have tagged RubA (TAP and/or S- tag) to identify the association partners of neddylated cullins. Neddylated Cul1/CulA recruits the counterparts of Skp1/SkpA, Rbx1/RbxA and various F-box proteins, which are core parts of CRLs. In addition, proteins required for the RubA linkage pathway were identified. A genetic analysis of the identified interacting proteins showed that the corresponding genes often are essential for *A. nidulans* growth. We showed this by heterokaryon rescue for the RubA encoding gene. Experiments with higher eukaryotes are difficult, because mutations often result in embryonic death of the organism. A deletion analysis revealed that not even parts of the *rubA* encoding gene can be deleted. CulA, RbxA, and RubA-E2 conjugating enzyme, UbcL, also seem to be essential. Busch et al., Mol. Microbiol. 49, 717-730, 2003. Busch et al., PNAS USA. 104, 8125-8130, 2007.

COMPARATIVE AND FUNCTIONAL GENOMICS

24. Proteome maps of total cell, mitochondrial and secreted proteins of *Aspergillus fumigatus*. Martin Vödisch[1]*, Olaf Kniemeyer[1], Dirk Wartenberg[1], Daniela Albrecht[1], Robert Winkler[2], Axel A. Brahhage[1], [1]Leibniz Institute for Natural Product Research and Infection Biology (HKI) / Friedrich-Schiller-Universität Jena, Germany, [2]Tecnologico de Monterrey, Departamento de Biotecnologia e Ing. de Alimentos, Mexico, martin.voedisch@hki-jena.de

Aspergillus fumigatus is a ubiquitously distributed saprophytic mould. Upon inhalation, *A. fumigatus* spores germinate, undergo hyphal growth and spread in the lungs causing deadly invasive aspergillosis in immunocompromised patients. In comparison to other fungi, *A. fumigatus* possesses obviously higher stress tolerance and better mechanisms to adapt to the host environment, since this Aspergillus species is the prominent cause for such severe lung infections. As a basis for comparative proteomic studies, we established a reference proteome map. Using MALDI-TOF-MS/MS we identified 392 protein spots representing 344 proteins separated on 2D-gels. Proteins involved in primary metabolism, protein synthesis, cell cycle regulation, DNA processing and transcription, cellular organisation and stress response were most abundant. Since mitochondria are involved in many essential processes such as energy metabolism, cellular differentiation and cell death, we established a protocol for the isolation of mitochondria of *A. fumigatus* mycelium and constructed a 2-D reference-gel for this organelle as well. 136 proteins represented by 196 spots have been identified so far. To complete the analysis of the mycelial proteome, we analysed the secreted proteins (secretome) under in vitro conditions. In many cases, proteins secreted by pathogens play an important role for their virulence. For *A. fumigatus* only little information about secreted proteins and their contribution to virulence is available. About 80 different proteins, represented by 272 spots, were detected of which approximately 45% were predicted as secreted proteins by bioinformatic analysis. Proteases and proteins involved in cell wall metabolism were most abundant. *Student poster

25. An archeological exploration of fungal production strains: Analysis of *Aspergillus niger* Glucoamylase- Producing Strains Using Comparative Genome Hybridization (CGH) and Quantitative Real-Time PCR (qPCR).
Barbara Cherry[1], Elena Bashkirova[1], Qiming Jin[1], Hiroaki Udagawa[2], Alfredo Lopez De Leon[1], Shinobu Tagaki[2], Randy Berka[1], and Debbie Yaver[1]. [1]Novozymes, Inc., Davis, CA and [2]Novozymes, Ltd., Japan.

Over the past few decades, *Aspergillus niger* has been improved for glucoamylase (AMG) production by classical mutagenesis. In order to begin characterizing these strains to determine what changes are associated with increased productivity, microarray-based comparative genome hybridization (CGH) analysis was used to obtain information regarding genomic changes that occurred during successive rounds of mutagenesis. This analysis revealed both deletions and amplifications of discrete chromosomal segments ranging in size from less than one kb to as much as 400 kb. Most importantly, we observed successive amplifications of a 216 kb region that included the *glaA* (glucoamylase) gene in the AMG strain lineage. In addition, CHEF gel analyses suggest that the amplified DNA segment has also undergone several non-reciprocal translocations in the AMG strain lineage. Quantitative PCR analyses confirmed the CGH results, leading us to conclude that at least some of the incremental improvements in AMG titers obtained from mutants derived by successive rounds of mutagenesis and screening may be associated with increases in the number of chromosomal *glaA* gene copies.

26. Dissection of polyketide biosynthesis pathways in *Aspergillus nidulans*
Michael Lynge Nielsen, Katrine Husted Brogaard, Christian Rank, Jakob Blæsbjerg Nielsen, Thomas Ostenfeld Larsen, Jens Christian Frisvad & Uffe Hasbro Mortensen Center for Microbial Biotechnology, Department of Systems Biology, Technical University of Denmark, mln@bio.dtu.dk

Genome sequencing and subsequent annotation of several *Aspergillus* genomes has revealed the presence of a large number of previously unknown putative gene clusters. Many of the clusters contain an interesting class of multi domain enzyme called polyketide synthases (PKS). The synthases are able to catalyze the polymerization of acetyl and propionyl subunits into large molecules that are subsequently modified by neighboring enzymes encoded within each cluster. The result is the formation of complex molecules with a high degree of structural diversity and often a very specific biological activity. These activities have been applied in a range of pharmaceuticals such as antibiotics, cholesterol lowering agents and anti cancer agents. Although, a few polyketide biosynthesis pathways have been elucidated, most clusters still remain to be mapped and characterized. Moreover, very little is known about how individual tailoring enzymes coordinate their activities. In the prese! nt study, we use a combination of reverse genetics and chemical analyses to identify the products of uncharacterized polyketide gene clusters in *Aspergillus nidulans*. This is specifically done by a combination of promoter replacements and gene deletions. For selected clusters we tag genes with fluorescent proteins to asses the spatial and temporal coordination of the biosynthesis. Examples of this approach are presented.

27. Improving gene targeting in *Aspergillus nidulans* for high through-put experiments

Jakob Blæsbjerg Nielsen, Michael Lynge Nielsen, Bjarne Gram Hansen and Uffe Hasbro Mortensen Center for Microbial Biotechnology, Department of Systems Biology, Technical University of Denmark, jbn@bio.dtu.dk

Gene targeting in filamentous fungi has until recently been cumbersome due to difficulties with gene-targeting substrate construction and low efficiencies demand laborious screening and verification of correctly targeted candidates. Recently, progress has been presented to diminish both bottlenecks. Hence, PCR based methods for substrate construction has been presented, and strains designed for efficient gene-targeting have been developed by eliminating the non-homologous end-joining pathway (NHEJ). Nevertheless, to facilitate high through-put gene targeting experiments, further improvements will be desirable. We will present new tools to facilitate easy production of substrates for construction of deletions, point mutations, GFP taggings etc. In addition, since elimination of NHEJ still produces significant background, success rates are typically in the range of 50-90 %, high through-put experiments will still benefit if this efficiency can be further improved. Since the me! chanism for the residual random integration is unknown, we have determined its genetic requirements in *Aspergillus nidulans*. Interestingly, the *RAD52* homolog, *radC*, appears to play a crucial role in this type of random integrations indicating that microhomology-mediated end-joining could be a possible integration pathway. To test this model, we are currently investigating the DNA sequences around double strand breaks, which have been repaired in the absence of NHEJ and not by traditional HR. Moreover, since RadC also plays a key role in gene targeting, no straight forward genetic manipulation can be done to channel more gene targeting substrates into the HR pathway. We have therefore evaluated different types of gene targeting substrates to address whether substrate design can influence the gene targeting efficiency in NHEJ deficient strains.

28. SMURF: genomic mapping of fungal secondary metabolite pathways

Natalie D. Fedorova[1], Nora Khaldi[2], Fayaz T. Seifuddin[1], Dan Haft[1], Geoff Turner[3], Kenneth Wolfe[2], William C. Nierman[1]. [1]J. Craig Venter Institute, Rockville, MD, USA. [2]Trinity College, Dublin 2, Ireland. [3]University of Sheffield, Sheffield S10 2TN, UK

Filamentous fungi produce an impressive array of secondary metabolites (SMs) including mycotoxins, pigments, antibiotics and pharmaceuticals. The genes responsible for SM biosynthesis, export, and transcriptional regulation are often found in SM gene clusters. To search systematically for such clusters, we developed the web-based software SMURF (Secondary Metabolite Unknown Regions Finder) available at www.jcvi.org/smurf. SMURF's predictions are based on the domain content of genes located close to backbone genes that often encode the first steps in SM pathways. In tests, SMURF accurately recovered all known SM clusters and detected 8 additional potential clusters in *Aspergillus fumigatus*. We applied SMURF to catalog putative SM clusters in 25 publicly available fungal genomes. Among these taxa, the Aspergilli appear to have the coding capacity for the largest numbers of these metabolites. The genome of *A. niger* alone encodes 61 backbone enzymes and 58 clusters. Further comparison reveals that SM pathways are very unevenly distributed among fungal taxa suggesting an evolutionary pattern of rapid pathway gain and loss. It also confirmed the correlation between unicellularity and the absence of SMs, and the prominent role of gene duplication in the creation of new pathways.

29. The *Aspergillus carbonarius* genome: Analysis of potential secondary metabolite biosynthetic gene clusters.

Scott E. Baker[1], Giancarlo Perrone[2], Antonia Gallo[2], Giuseppina Mulè[2], Antonia Susca[2], Antonio Logrieco[2], [1]Pacific Northwest National Laboratory, Richland, Washington, [2]National Research Council, Institute of Science of Food Production Bari, Italy

A draft genome sequence for *Aspergillus carbonarius* strain ITEM5010 has been recently generated in collaboration with the US Department of Energy Joint Genome Institute. *Aspergillus carbonarius* is closely related to *Aspergillus niger*, an important model organism as well as an industrial enzyme and organic acid producer. Like *A. niger*, the genome sequence for *A. carbonarius* encodes numerous glycoside hydrolases. In addition, the *A. carbonarius* genome sequence will aid in annotation of *A. niger*. *Aspergillus carbonarius* is also an economically significant organism as the main agent of ochratoxin A contamination of wine. We have characterized the putative ochratoxin A biosynthetic cluster and have initiated manual annotation of the draft genome sequence. While *A. niger* has been recently shown to produce fumonisin B2, the biosynthetic cluster for fumonisin biosynthesis is absent from the *A. carbonarius* genome. We have initiated a detailed analysis of the polyketide synthase (PKS) genes encoded within the *A. carbonarius* genome. Preliminary phylogenetic analysis of ketosynthase domains indicates a substantial diversity within predicted PKS encoding genes.

30. High-throughput sequencing of the *A. nidulans* transcriptome.

Mark X Caddick, Chris Sibthorp, Neil Hall, Dan Rigden and Prudence Wong.The University of Liverpool, School of Biological Sciences, Liverpool L69 7ZB, UK

The true complexity of the *A. nidulans* transcriptome is poorly defined at present. The genome sequence has been used to identify putative genes but it is likely that a significant number have been missed. The experience of many researches is that the precise coordinates of transcript 5' and 3' ends, splice sites and even the coding regions are quite frequently miss-assigned. Currently we have very little knowledge about variation in promoter use, differential splicing or 3' heterogeneity. Finally, the role of antisense and other non-coding RNAs has not been extensively assessed, although their potential importance to gene expression and regulation may be profound. As a first step to rectifying this we are about to initiated transcriptome sequencing utilising ABI SOLiD sequencer technology. The specific aims of this work, the potential of high throughput sequencing in fungal research and our preliminary data will be discussed.

31. Transitive RNA Interference in the Fungi *Aspergillus oryzae* and *Aspergillus niger*

Evee Fernandez, Suchindra Maiyuran, Amanda Fischer, Donna Moyer and Howard Brody. Novozymes Inc., 1445 Drew Avenue, Davis, CA 95618. HowB@novozymes.com

RNA interference (RNAi), moderates gene expression by means of cleavage of double stranded RNA by Dicer, producing 21-25 nucleotide silence inducing RNAs (siRNAs). In association with Argonaut containing complexes, these siRNAs target sequence specific degradation of the homologous single-stranded messenger RNA. In the majority of eukaryotes the nuclease degradation occurs within the boundaries of the dsRNA target. In *Arabidopsis thaliana* and *Caenorhabditis elegans* gene silencing can also take place transitively, spreading into regions adjacent to the double-stranded target. Here we demonstrate endogenous Transitive RNAi in the ascomycetes *Aspergillus oryzae* and *Aspergillus niger*. A 500 bp fragment of the *A. oryzae wA* gene encoding a Polyketide Synthase, involved in spore color development, or its *A. niger* ortholog was inserted directly 5' of an inverted repeat derived from the *E. coli* Hygromicin gene. Transformants of both Aspergilli produced spores lighter in color than wild type. Southern analysis performed on white spored transformants confirmed that gene disruption did not account for the phenotype. Real-time RT-PCR demonstrated a direct correspondence of steady-state mRNA level and spore color. In a strain lacking the *A. oryzae* ortholog of the *N. crassa* QDE1 RNA dependent RNA polymerase (RdRP) gene, transformants obtained having the *wA* fragment inserted upstream of the Hygromicin-derived inverted repeat produced only wild type spore color, which indicates that RdRP is necessary to achieve transitive RNAi.

32. The *Aspergillus* Genome Database (AspGD), a curated database of *Aspergillus* gene, protein, and genomic sequence information for the fungal research community.

Martha Arnaud[1], Gail Binkley[1], Marcus Chibucos[2], Maria Costanzo[1], Jonathan Crabtree[2], Stuart Miyasato[1], Prachi Shah[1], Marek Skrzypek[1], Jennifer Russo Wortman[2], and Gavin Sherlock[1] [1]Department of Genetics, Stanford University, Stanford, CA 94305-5120 [2]Institute for Genome Sciences, University of Maryland School of Maryland, Baltimore, MD

We have recently obtained funding to create a resource for the *Aspergillus* research community, AspGD, which will be a multispecies genomics database modeled on the *Candida* and *Saccharyomyces* Genome Databases. Our goal is to facilitate research on Aspergilli and on other medically and economically important fungal pathogens by providing a first-stop reference for *Aspergillus* genomics and molecular biology, with up-to-date, high-quality, curated scientific information and web-based research tools. We will implement an optimized annotation pipeline across all *Aspergillus* genomes, to maintain a set of current and consistent gene boundary annotations, and will incorporate links between genomes based on orthology and synteny data. Gene function, localization, and mutant phenotype annotations will be manually curated from the *Aspergillus* literature. We will provide web-based tools for sequence visualization and retrieval, and for analysis of sequence information and gene product annotations. All of the data in AspGD will be freely available to the public from http://www.aspgd.org/. In addition, we will maintain a colleague registry by which *Aspergillus* research community members may share contact information and research interests, to facilitate collaboration. We strive to be responsive to the needs of the research community, and we welcome your feedback and suggestions. AspGD curators may be reached by email at aspergillus-curator@genome.stanford.edu AspGD is supported by grant RO1 AI077599 from the NIAID at the NIH.

33. Proteome analysis of the response of *Aspergillus fumigatus* to voriconazole

Nansalmaa Amarsaikhan*, Olaf Kneimeyer , Zumrut Ogel Department of Biotechnology, Middle East Technical University, Ankara, Turkey Department of Molecular and Applied Microbiology, Hans-Knoll Institute, Jena, Germany nansa89@yahoo.com

Aspergillus fumigatus is the most important airborne fungal pathogen which can cause invasive aspergillosis in immunocompromised individuals, where the number of available antifungal drugs is limited due to its eukaryotic origin. Aspergillosis is usually treated by the administration of antifungal drugs, in most cases by the azole group drugs such as voriconazole. Recently, there has been increasing evidence for antifungal drug resistance in Aspergillus. For this reason, the research focus has shifted to investigating the key proteins involved in drug resistance. Commonly, it is known that development of antifungal drug resistance is associated with the upregulation of general stress response pathways. Thus, studies focusing on the transcriptional and proteomic profiles are of great importance to address these general mechanisms. In this study, we are studying the change of the protein expression level of *A. fumigatus* in response to voriconazole, an important azole group drug. As a result of this study, we will be able to compare the proteome data with transcriptome data released by Ferreira et al (2006).This study would be the first proteomic study directed to antifungal drug resistance mechanisms and would lead to the comprehensive evaluation of key proteins involved in this process. A greater understanding of azole resistance in *A. fumigatus* could lead to an improved antifungal therapy in future. *Student poster

34. Cell wall degrading enzymes in *Aspergillus flavus* and fungal virulence

Jiujiang Yu[1], Natalie Fedorova[2], William C. Nierman[2,3], Gary A. Payne[4], Joan W. Bennett[5], Deepak Bhatnagar[1], and Thomas E. Cleveland[1] [1]USDA/ARS, Southern Regional Research Center, New Orleans, LA, USA. [2]J. Craig Venter Institute, Rockville, MD, USA. [3]The George Washington University School of Medicine, DC, USA. [4]North Carolina State University, Raleigh, NC, USA . [5]Rutgers University, School Environmental and Biological Sciences, New Brunswick, NJ, USA.

The primary objective of our *Aspergillus flavus* genomics program is to understand the global regulation of toxin production by this fungus with a view to reduce or even eliminate aflatoxin contamination in food and feed, as well as control fungal infection in preharvest crops. An *A. flavus* EST and a whole genome sequencing project for this fungus have been completed. Genes that are potentially involved in aflatoxin formation and fungal infection have been identified. Data mining of the *A. flavus* genome has demonstrated that *A. flavus* possesses a whole array of genes encoding cell wall degrading enzymes. Gene expression studies in *A. flavus* indicate that these enzymes are highly expressed. These fungal enzymes could play an important role in the virulence of *A. flavus* and its ability to infect the crops. These enzymes probably contribute to the saprophytic property of *A. flavus* and can be explored for its potential benefit in biofuel production.

GENE REGULATION

35. *Aspergillus fumigatus* GATA factor AfNsdD affects hyphal growth and secondary metabolism.

Yoon-Hee CHEON[1], Dong-Soon OH[1], Dong-Min HAN[2], and Kap-Hoon HAN[1]. [1]Department of Pharmaceutical Engineering, Woosuk University, 565-701, Korea and [2]Division of Life Sciences, Wonkwang University, 570-749, Korea. khhan@woosuk.ac.kr.

Aspergillus fumigatus is a primary causative agent of aspergillosis, which is an opportunistic infectious fungal disease mainly localized in the respiratory system of human and animals. Although, sexual developmental process is recently found in *A. fumigatus*, its¡¯ downstream gene regulation process after the mating remains to be identified. To compare whether *A. fumigatus* has conserved sexual developmental process in molecular level, we identified an ortholog of the *nsdD* gene, encoding a GATA type transcription factor required for sexual development in a homothallic fungus *A. nidulans*. Gene expression study showed that the *A. nidulans nsdD* gene is highly expressed in the hypoxic condition as well as sexual stage. Similarly, the putative ortholog of *nsdD* in *A. fumigatus*, named *AfnsdD*, behaves very similar to *A. nidulans nsdD*. Knock-out of the *AfnsdD* gene resulted in highly melanized colony phenotype and retarded hyphal growth, which is very similar to *A. nidulans nsdD* mutant. This result indicates that the NsdD GATA factor- mediated molecular mechanism is probably conserved in Aspergilli although many of them have no known sexual development. This work was supported by grant from KOSEF (R1-2006-000-11204-0).

36. Deletion of the *Aspergillus nidulans* nitrogen regulatory gene *areB* reveals pleiotropic phenotypes.

Koon Ho Wong[1], Meryl A. Davis[1], Michael J. Hynes[1] and Richard B. Todd[1,2] [1]Department of Genetics, The University of Melbourne, 3010 AUSTRALIA. [2]Department of Plant Pathology, Kansas State University, Manhattan KS 66506 USA.

The *Aspergillus nidulans* transcription activator AreA is a key regulator of nitrogen metabolic gene expression. AreA transcription capacity is highly regulated in response to nitrogen nutrient quality and/or availability by autogenous transcriptional activation, differential areA mRNA stability, interaction with the NmrA corepressor and the TamA coactivator, and regulated AreA nuclear export. AreA contains a C-terminal GATA zinc finger DNA binding domain that is highly conserved in areA homologues in other filamentous fungi. In *A. nidulans*, another GATA factor AreB containing an N-terminal GATA domain and a C-terminal leucine zipper domain has been implicated in nitrogen regulation. Evidence that AreB acts in nitrogen regulation was provided by gain-of-function mutations in *areB*. AreB and NreB, the *Penicillium chrysogenum* ortholog, are highly conserved and NreB overexpression suggested that NreB acts negatively possibly by competing with AreA for DNA binding. However, AreB and NreB are likely orthologous to the *Neurospora crassa* sexual development regulator Asd4, which does not function in nitrogen regulation. In order to investigate the role of AreB in nitrogen regulation, we determined the *areB* deletion phenotype. Gene replacement mutations in *areB* or *nreB* were not previously reported. AreB antagonizes AreA activation and functions in growth, asexual development and conidial germination but not sexual development. Overexpression of AreB prevents AreA-dependent gene expression and confers severe growth inhibition. Both the DNA-binding domain and the leucine zipper of AreB are required for AreB functions. These data indicate that AreB has a wide domain of action including but not limited to nitrogen metabolic genes.

37. Mechanisms of action of transcriptional regulators involved in fatty acid catabolism.

Andrea Prynych[1]*, Sandra Murray[1], Meryl Davis[1], and Michael Hynes[1]. [1]Department of Genetics, University of Melbourne, Parkville, Australia.

FarA, FarB and ScfA are Zn(II)2Cys6 transcription factors which upregulate genes required for growth on fatty acids in *Aspergillus nidulans*. FarA and FarB are highly conserved and bind to CCGAGG core sequences in the promoters of their target genes. My project focuses on investigating how they work together to control gene expression. *farA* overexpression strains show an increase in reporter gene activity in the presence of oleate but a decrease is seen when *farB* is overexpressed. This led to a proposed model in which a FarA homodimer activates genes when a long-chain fatty acid is present, but a FarA-FarB heterodimer (with ScfA potentially interacting) increases expression during growth on short-chain fatty acids. Protein interactions are being determined using yeast two hybrid analysis. In the heterodimer, FarB might receive the short-chain fatty acid signal, while FarA provides the activation ability. FarB-FarA fusion proteins were created in which different putative activation domains of FarA were inserted into the C-terminus of FarB, creating FarB proteins that have activation capability. These proteins were tested in a *farA?* background with growth significantly increased compared to wildtype *farB*. The activation ability of FarA may be its main contribution to short-chain fatty acid dependent expression, but it may also have other roles. *Student poster

38. Effects of stress stimuli on "transposability" and post-transcriptional modifications of mRNAs from DNA transposon *Crawler* in *Aspergillus oryzae*.

Hironobu Ogasawara[1], Yoji Hata[2], Saori Takahashi[1], and Katsuya Gomi[3]. [1]Institute for Food and Brewing, Akita Pref. Agric. Forest. and Fish. Res. Center, Akita, Japan. [2]Research Institute, Gekkeikan Sake Co. Ltd., Kyoto, Japan. [3]Graduate School of Agricultural Science, Tohoku University, Sendai, Japan.E- mail:hironobu@arif.pref.akita.jp

An active DNA transposon *Crawler* isolated from the genome of industrially important fungus *Aspergillus oryzae* transposes under extreme stress conditions. The mRNA analysis of *Crawler* in the conidia revealed that cryptic splicing and premature polyadenylation of the mRNA occurred in the normal culture condition. In the present study, we analyzed the relationship between transposition events and proportional change in post- transcriptional modifications of the *Crawler* mRNA by RT-qPCR under various stress stimuli. These results suggested that post-transcriptional regulation for *Crawler* tends to be inhibited by stress treatment of $CuSO_4$ and heat shock, which stimulated the transposition events in conidia allowing the full-length and active transposase to be produced. Even extreme stress such as UV, H_2O_2 or metal ions except Cu^{2+} could not lead to the transposition of *Crawler* resulting from insufficient changes for mature mRNA molecules. Moreover the effects of stress stimuli inducing the transposition events of *Crawler* on the splicing for several genes responsible for metabolism, cellular structure or RNA processing were also examined to understand the control mechanism against the active transposon.

39. Cell wall integrity MAP kinase signalling pathway in the human-pathogenic fungus *Aspergillus fumigatus*

Radhika Jain[1,2]*, Vito Valiante[1], Thorsten Heinekamp[1], Axel.A.Brakhage[1,2]. [1]Leibniz Institute for Natural Product Research and Infection Biology - HKI, Jena, Germany. [2]University of Jena, Germany.

Mitogen-activated protein kinase (MAPK) cascades are evolutionary conserved signalling modules transducing stimuli from the cell surface to the nucleus. MAPK pathways control key virulence functions and are involved in the regulation of various cellular responses in eukaryotes. In *A. fumigatus*, three putative MAP kinases are involved in the cell wall integrity (CWI) signalling pathway: *bck1* (MAPK kinase kinase), *mkk2* (MAPK kinase) and *mpkA* (MAPK).To investigate whether *bck1*, *mkk2* and *mpkA* are involved in CWI signalling of *A. fumigatus*, mutant strains of respective genes were constructed and phenotypically analysed. In addition, transcriptional regulation of *mpkA*, and the function of the corresponding protein was studied. An *A. fumigatus* strain was generated carrying the *lacZ*-reporter gene fused to the *mpkA*-promoter. Post transcriptional modification of MpkA was monitored by western blot analysis.Functional analysis of Bck1, Mkk2 and MpkA in *A. fumigatus* revealed their involvement in hyphal growth and filamentation. The sensitivity of these mutants against cell wall inhibitors demonstrates that the genes are acting in the CWI signalling pathway and mediate cell wall damage responses. Furthermore, new insights into the regulation of MAPK signalling in *A. fumigatus* were gained. It was observed that *mpkA* is regulated transcriptionally in response to cell wall disrupting agents. By contrast, regulation of MpkA following treatment with oxidative stress agents occurs at the post transcriptional level. Interestingly, delta *mpkA* mutants are still virulent in a mouse infection model. *Student poster

40. Regulation by light of conidiation genes in *Aspergillus nidulans*

C Ruger-Herreros*, M Olmedo, R Fernández-Barranco, LM Corrochano, D Cánovas Depto de Genetica, Avenida Reina Mercedes,6 Universidad de Sevilla, Spain

The ascomycete fungus *Aspergillus nidulans* is a model organism for the research on conidiation. Red and blue light stimulate conidiation in *A. nidulans,* but mutations in the *veA* gene allow conidiation in the dark. Conidiation in *A. nidulans* is controlled by a master regulator, the product of the *brlA* gene. Many gene products act upstream of *brlA*, presumably allowing the synthesis of chemicals or allowing the transduction of environmental signals, to trigger *brlA* transcription and conidiation. The regulation of conidation by light may occur through activation of the expression of some regulatory genes. We have shown that the expression of several conidiation genes, including *brlA, fluG flbA, flbB* and *flbC,* is activated by light. The level of photoinduction was moderate, between 4-10 fold, and changed with the age of the culture and the genetic background. Some genes, like *brlA,* are activated by light regardless of age, but *flbA* was only induced by light in 18 h-mycelia, while *flbC* was only induced in 25 h-mycelia. Mutation in *veA* did not prevent photoactivation of gene expression, and an increase in light-dependent mRNA accumulation was observed for *flbA* and *flbC.* The *veA* mutant did show an increase in the accumulation of some conidiation mRNAs in the dark. The activation by light of conidiation genes would provide increased amounts of regulatory proteins for conidiation clarifying the role of light on conidation. *Student poster

41. Activation of silent gene clusters in *Aspergillus nidulans*

S. Bergmann[1]*, V. Schroekh[1], K. Scherlach[2], J. Schuemann[2], H.-W. Nuetzmann[1], C. Hertweck[2,3] and A.A. Brakhage[1,3]. [1]Department of Molecular and Applied Microbiology and [2]Department of Biomolecular Chemistry, Leibniz Institute for Natural Product Research and Infection Biology -Hans-Knöll-Institute, Jena, Germany. [3]Friedrich-Schiller-University, Jena, Germany. sebastian.bergmann@hki-jena.de

Microorganisms as bacteria and fungi produce important low-molecular weight molecules that show different biological activities. Genome mining of available fungal genomes indicated that their potential to produce these compounds designated secondary metabolites (SMs) is greatly underestimated. Fungi encode the genetic information for the biosynthesis of many more compounds which still await discovery. The vast amount of DNA sequence in the public database represents only the beginning of this new genomics era. Most of the fungal secondary metabolism gene clusters are silent under laboratory conditions. Despite this limitation, to get access to the vast number of unknown compounds encoded by silent gene clusters, mixing genomic data, genetic engineering and analytical techniques provides a new avenue to discover novel and potentially bioactive natural products. Bioinformatic analysis of the published *A. nidulans* genome sequence led to the identification of 48 putative SM gene clusters. By overexpressing a pathway-specific transcription factor, we were able to induce a silent gene cluster containing the gene for a mixed PKS/NRPS system. This approach is rendered feasible by the fact that all of the genes encoding the large number of enzymes required for the synthesis of a typical secondary metabolite are clustered and that in some cases, a single regulator controls the expression of all members of a gene cluster to a certain extent. By this way, we were able to isolate novel compounds. In addition, we will discuss further attempts to activate silent gene clusters and to identify physiological conditions under which gene clusters are active under natural conditions. *Student poster

42. Characterization of the *Aspergillus niger prtT*, a unique regulator of extracellular protease encoding genes.

Peter J. Punt[1], Frank H. J. Schuren[1], Jan Lehmbeck[2], Tove Christensen[2], Carsten Hjort[2], and Cees A.M.J.J. van den Hondel[3]. [1]TNO Quality of Life, P.O. Box 360, 3700 AJ Zeist, The Netherlands; [2]Novozymes A/S, Denmark; [3]Institute of Biology, Leiden University, The Netherlands Peter.punt@tno.nl

Expression of several *Aspergillus niger* genes encoding secreted, but not vacuolar, protease genes was affected in the previously isolated *A. niger* protease mutant, AB1.13. Complementation cloning of the corresponding protease-regulatory gene affected in this mutant was accomplished using a screening approach based on the use of the *A.nidulans amdS* selection marker driven by the *A.niger pepA* promoter. Complementation was achieved by introduction of a self-replicating cosmid library into the mutant strain carrying the *PpepA::amdS* marker. The complementing sequences obtained from complementing cosmid clones contained a gene encoding a member of the fungal-specific Zn2Cys6-binuclear cluster protein family. Different from several other pathway specific regulators present in Aspergillus species no PrtT orthologues could be found in any other non-*Aspergillus* species, but also not in *Aspergillus nidulans*. In all *Aspergillus* species the *prtT* gene is tightly clustered to a syntenic region carrying the amylolytic regulator *amyR*. The *prtT* gene is transcribed from two upstream promoters, resulting in mRNA species carrying one or more short upstream open reading frames. Both this finding and the identification of differentially spliced EST clones suggest the presence of post-transcriptional gene regulation for *prtT*.

43. Engineering intracellular metabolism by altering gene expression of *Aspergillus oryzae*

Hideaki Koike[1], Tomomi Toda[1], Tomoko Ishii[1], Junichiro Marui[1], Sumiko Ohashi[1], Hiroko Hagiwara[1], Kenichiro Matsushima[2], Tadashi Takahashi[2], Akira Ohyama[3], Motoaki Sano[4], Shinichi Ohashi[4], Marie Nishimura[5], Yasuji Koyama[2] and Masayuki Machida[1] [1] Natl. Inst. Adv. Indus. Sci. Tech. (AIST), [2] Noda Inst. Sci. Res., [3] Insilico Biology, [4] Kanazawa Inst. Tech., [5] Natl. Inst. Agrobiol. Sci.

Aspergillus oryzae is one of the most important organisms in Japanese fermented food industry. Though it hardly produces secondary metabolites, related organisms are producers of diverse metabolites. A major objective of our project is to develop a system using *A. oryzae* to generate diverse metabolites. Combinatorial biochemistry approach by introducing exo-genous genes and fermentation techniques are combined. To reveal regulation of metabolic pathways in *A. oryzae* under various conditions, gene expression profiles under condition of submerged culture, solid media and so on were analyzed by DNA micro array. A series of deletion mutants of transcription factor have been obtained by systematic gene targeting. More than 200 mutants were tested for their ability to produce some metabolites. Several mutants showed lack of production or different dependency on environmental nutrient conditions. The factors affecting expression level of metabolic genes are being studied. To develop the system, novel vectors and host strains of *A. oryzae* have been constructed. We have replaced promoter regions of some metabolic genes and successfully altered the level of some metabolite productions. To introduce genes, the correct detection of splicing position is important. We have analyzed motifs of splicing in *A. oryzae* by comparing EST and genome sequences.

44. Identification of putative polyadenylation signals in *Aspergillus oryzae* using EST data.

Mizuki Tanaka[1], Yoshifumi Sakai[1], Osamu Yamada[2], Takahiro Shintani[1], and Katsuya Gomi[1] [1]Div. Biosci. Biotech. Future Bioind., Grad. Sch. Agric. Sci., Tohoku Univ., [2]Natl. Res. Inst. Brewing, Japan.

In the previous study, we showed that premature polyadenylation within the ORF of AT-rich heterologus genes generally occurred in *Aspergillus oryzae*, and it was prevented by codon optimization (*Appl. Environ. Microbiol.*, **74**, 6538-6546 (2008)). This observation suggested that low mRNA levels of heterologus genes were resulted by the degradation process of mRNA lacking translation termination codon, which is called chr(34)nonstop mRNA decaychr(34) pathway. In eukaryotes, poly(A) addition site was determined by several sequence elements, and those sequence element have been extensively studied in mammals and yeast. Among those, hexanucleotide AAUAAA and its related sequences located in 10-30 nt upstream of poly(A) site are known as most highly conserved sequence element, which is called polyadenylation signal. However, there were no AAUAAA or its related sequence within ORF of heterologus genes occurred premature polyadenylation. This suggested that another sequence could function as polyadenylation signal(s) in *A. oryzae*. In this study, we constructed a dataset of nucleotide sequences around poly(A) site from EST (expressed sequence tag) data to elucidate the sequence elements that can function as 3'-end processing signals in *A. oryzae*. Furthermore, we examined the 3'-UTR length and nucleotide profile around the poly(A) site by using this dataset.

45. Profiling of gene expression during polar growth of *Aspergillus fumigatus*

Ken Oda[1], Andrew Breakspear[2], Susan Cowden[1], Michelle Momany[1]. [1]Dept. of Plant Biology, Univ. of Georgia, Athens, USA., [2]current address: Dept. of Plant Pathology, Univ. of Minnesota, St. Paul, USA, e-mail: koda@plantbio.uga.edu

A. fumigatus is the most common airborne pathogen causing fatal mycoses in immunocompromised patients. Polarized growth is one of the critical factors for establishing fungal pathogenesis, but little is known about the genes involved in early polar growth and their regulation. The purpose of this study was to find polar growth related genes in *A. fumigatus*. *A. fumigatus* Af293 was cultured in complete medium and total RNA was extracted at set time points. DNA microarray experiments were performed comparing dormant cells (0hr) with isotropicly growing cells (4hr), isotropicly growing cells with cells showing emerging germtubes (6hr), and with more mature hyphae (8hr). Expression of selected genes was confirmed by RT-PCR. Comparison of altered genes in the three array experiments shows that the change of gene expression is most prominent at the switch from dormant to isotropic growth and that many genes show significant expression changes during early growth in *A. fumigatus*.

OTHER TOPICS

46. *Aspergillus fumigatus* gene expression in experimental murine lung infections
William C. Nierman[1], Natalie D. Fedorova[1], Charlie Cairns[2], and Elaine Bignell[2] [1] J. Craig Venter Institute, Rockville, MD, USA [2] Imperial College, London, UK

Aspergillus fumigatus is the most frequent cause of invasive aspergillosis in immune suppressed human patients. We have developed a murine model for analyzing the early stages of A. fumigatus colonization and progression to invasive disease. The model incorporates instillation of conidia into mouse lungs and subsequent harvesting of bronchoalveolar lavage fluid (BALF) samples for analysis. Validated mRNA amplification and analysis protocols have allowed transcriptome analysis of the fungal mRNAs present in the BALFs. Expression profiling of *A. fumigatus* germlings at 12-14 hours after instillation into neutropenic mouse lungs reveals dramatically altered gene expression relative to growth in laboratory culture. Up-regulated genes are often found in secondary metabolism and other accessory gene clusters such as the gliotoxin, pseurotin, and siderophore biosynthesis clusters. We found also significant concordance between the observed host-adapted changes in the transcriptome and those resulting from *in vitro* iron limitation, nitrogen starvation, and loss of the LaeA methyltransferase. To further elucidate the role of LaeA in *A. fumigatus* virulence, we analyzed temporal gene expression profiles of a wild type and an isogenic *laeA*-deleted strain, which misregulates gene expression at secondary metabolite gene clusters and is avirulent in a murine model. Growth and differentiation during initiating phases of murine infection were compared between parental and mutated isolates at 4, 8, and 14 hours post-infection in neutropenic mice. Transcriptome analysis of the *laeA* mutant revealed a major *in vivo* regulatory deficit of a few secondary metabolite biosynthetic gene clusters and more than thirty accessory gene clusters. In our continuing studies employing this murine early infection model, we will undertake analysis of hypervirulent *A. fumigatus* mutants, *laeA* proteome analysis, and the murine host response to the fungal pathogen.

47. Impact of Homocitrate Synthase on *Aspergillus fumigatus* pathogenesis
Felicitas Schöbel* and Matthias Brock. Leibniz Institute for Natural Product Research and Infection Biology – Hans Knöll Institute, Jena

Fungi, such as *A. fumigatus*, are able to synthesize lysine de novo via the alpha-aminoadipate pathway. In contrast, lysine is an essential amino acid for humans and must be obtained from the diet. Therefore, enzymes of this pathway might represent potential targets for new antifungals. However, until now it is unclear, whether *A. fumigatus* can satisfy its need for lysine from the degradation of the surrounding host tissue, e.g. from the degradation of proteins. This assumption is supported by the virulence attenuation of an *A. fumigatus* methylcitrate synthase mutant in murine infection models (Ibrahim-Granet et al 2008). Such a mutant accumulates toxic amounts of propionyl-CoA, which most likely derive from the degradation of proteins during pathogenesis. Our major interest was to verify, whether the de novo synthesis of lysine is only essential for the onset of an invasive aspergillosis or also during later stages of infection. To prove this assumptions, we deleted the homocitrate synthase, the first enzyme of the alpha-aminoadipate pathway, from the genome of *A. fumigatus*. The mutant revealed that the de novo lysine biosynthesis plays a critical role for conidia germination on unhydrolysed proteins. In vivo studies confirmed an importance of lysine biosynthesis especially during the onset of infection, whereas the phenotype was partially complemented by feeding mice with elevated levels of lysine. Ibrahim-Granet O., et al. (2008) Methylcitrate synthase from *Aspergillus fumigatus* is essential for manifestation of invasive aspergillosis. Cell Microbiol.; 10(1):134-48. *Student poster

48. Conidiation color mutants of *Aspergillus fumigatus* are highly pathogenic to the heterologous insect host *Galleria mellonella*.
Jennifer Jackson, Laura Higgins, and Xiaorong Lin (xlin@mail.bio.tamu.edu)

Invertebrates have been increasingly viewed as a valid model for virulence studies of human fungal pathogens as their virulence traits are likely conserved among different hosts. The caterpillar *Galleria mellonella* has been used as a heterologous host for a number of yeast pathogens. Here we have evaluated the possibility of applying this heterologous insect model to investigate the virulence trait of the filamentous fungal pathogen *Aspergillus fumigatus*: melanization. Melanization in *A. fumigatus* confers bluish-grey color to conidia and is a known virulence factor in mammal models. Surprisingly, conidial color mutants with deletions in the defined melanin biosynthesis gene cluster in B5233 background caused enhanced insect mortality. Insertional mutants in Af293 background producing conidia of previously identified colors and of novel colors were isolated and they also displayed a higher level of pathogenicity in the insect model, confirming the relationship between fungal melanization defects and enhanced virulence to the caterpillar. Exacerbated insect immune response induced by increased exposure of PAMPs and elevated levels of fungal secreted metalloproteinases may cause the increased mortality of the larvae infected with the color mutants. Our study underscores the importance of the knowledge about the insect innate immunity status in understanding fungal pathogenicity in insect models. This study also shows that the G. mellonella is a reproducible model for *A. fumigatus* that could become a valuable tool for studying fungal traits that are required for infections in both mammals and the insect. Additionally, our observations indicate the potential of using melanization defective mutants of natural insect fungal pathogens in the biological control of insect populations.

49. Interaction of *Aspergillus fumigatus* with the human complement system

Judith, Behnsen[1]*, Andrea, Hartmann[2], Axel A., Brakhage[1], Peter F., Zipfel[2]. Department of Molecular and Applied Microbiology[1], Department of Infection Biology[2], Leibniz Institute for Natural Product Research and Infection Biology - Hans Knoell Institute (HKI) and Friedrich Schiller University Jena, Germany, Judith.Behnsen@hki-jena.de

The complement system plays a crucial role in the defense against pathogenic microorganisms. This study describes two mechanisms of complement evasion of the opportunistic human-pathogenic fungus *Aspergillus fumigatus*. The binding of complement regulators to different developmental stages of *A. fumigatus*, as well as to a pigmentless *pksP* mutant, was analyzed and distinct binding patterns were observed. Conidia showed strong binding of three regulators of complement activation: factor H, FHL-1, and plasminogen. In contrast to conidia, hyphae showed only weak binding of complement regulators. The pigmentless *pksP* mutant, which produces white conidia and which proved to be attenuated in virulence in a mouse infection model, was found to bind no factor H and plasminogen. Remarkably, hyphae inactivate complement in a factor H independent way. Culture supernatant of *A. fumigatus* showed complement cleavage activity. Key components of the complement activation cascade (C3, C5 and C4) were efficiently degraded by a secreted protease. These two independent mechanisms might help *A. fumigatus* to evade the attack of the human complement system. *Student poster

50. Alcohol fermentation and hypoxia adaptation in the pathogenic mold *Aspergillus fumigatus*

Nora Grahl[1]*, Jeffrey M. Macdonald[2], Michael P. Gamcsik[2], and Robert A. Cramer Jr[1]. [1]Department of Microbiology, Montana State University, Bozeman, MT 59717, USA, [2]Joint Department of Biomedical Engineering, University of North Carolina Chapel Hill and North Carolina State University Raleigh, NC.

Recent studies have shown that tolerance to hypoxia may be a virulence attribute of fungal pathogens. Currently, our knowledge of *Aspergillus fumigatus's* (*Af*) ability to survive in hypoxic conditions during fungal pathogenesis is limited. Using a murine model of invasive aspergillosis and ^{1}H-NMR metabolomics, we have found ethanol and lactate in the lungs of *Af* infected mice. This result suggests that *Af* utilizes fermentation pathways *in vivo* to cause disease. During ethanol fermentation, pyruvate, the end product of glycolysis, is metabolized by pyruvate decarboxylase (Pdc) to acetaldehyde, which is subsequently reduced to ethanol by alcohol dehydrogenase III (*alcC*). Under growth conditions containing glucose as a carbon source and low oxygen levels we observed that pyruvate decarboxylase activity in *Af* cell-free extracts is elevated. We identified 3 potential pyruvate decarboxylase genes (*pdcA*, *pdcB*, and *pdcC*) in *Af* and observed that all 3 *pdc* genes were induced under hypoxic conditions. Generation of null mutants in these genes and the alcohol dehydrogenase *alcC* has allowed us to determine the role of fermentation in hypoxia adaptation in this pathogenic mold. Our results indicate that PdcA is the pyruvate decarboxylase primarily responsible for alcohol fermentation in *Af*. The loss of *pdcA* results in a decrease of Pdc activity of ~80% and a reduction of ethanol production below the detection limit of 0.01%. Loss of *alcC* results in elimination of 95% of EtOH production. Surprisingly, we did not observe a growth defect of the *pdcA* or *alcC* mutant under hypoxic conditions. We hypothesize that either residual Pdc activity from PdcC or reduction of pyruvate to lactate by lactate dehydrogenase is enough to allow survival under hypoxic conditions. We further hypothesize that either the elimination of all *pdc* genes or a *pdcA pdcC* double knockout will affect the ability of *Af* to survive under hypoxic conditions and cause disease. *Student poster

51. The Trehalose Pathway is critical for *Aspergillus fumigatus* virulence

Srisombat Puttikamonkul*, Sven D. Willger, and Robert A. Cramer Jr. Department of Veterinary Molecular Biology, Montana State University, Bozeman, MT, USA.

Trehalose, a disaccharide sugar, accumulates in *Aspergillus* conidia and plays a role in protection against stress including: high temperature, high osmolarity and ROS. In this study, we have characterized the trehalose pathway in *A. fumigatus* for the first time. Phylogenetic analyses revealed multiple copies of the trehalose biosynthesis gene *tps1* (*tpsA* and *tpsB*), a single copy of the trehalose-6-phosphate (T6P) phosphatase *tps2* (*orlA*), and single copies of *tps3* and *tsl1* orthologs. We have generated single and double null mutants of *tpsA* and *tpsB* and show that both genes are required for trehalose biosynthesis. Generation of a single *tpsA* or *tpsB* mutant did not alter trehalose accumulation, while generation of a *tpsA tbsB* double mutant completely abolished trehalose biosynthesis. Generation of an *orlA* (*tps2*) null mutant revealed a role for *orlA* in asexual conidiation when cultured on glucose minimal media. However, the defect in conidiation could be recovered on both sorbitol and glycerol minimal media. The *orlA* and *tpsA tpsB* mutants displayed sensitivity to growth at high temperatures (50°C). Surprisingly, lack of *orlA* did not affect the production of trehalose at 37°C suggesting an alternate pathway for trehalose biosynthesis exists in *A. fumigatus*. Of particular interest, the *orlA* null mutant was avirulent in two murine models of invasive aspergillosis. Our results suggest that trehalose and T6P are important components of the biology of *A. fumigatus*. Importantly, our results suggest that T6P phosphatase contributes to the ability of this mold to cause lethal disease in immunocompromised patients. Thus, as others have suggested, the trehalose pathway is worth exploiting as an antifungal drug target given its conservation in other pathogenic fungi and absence in humans. *Student poster

52. A sterol-regulatory element binding protein is required for cell polarity, hypoxia adaptation, azole drug resistance, and virulence in *Aspergillus fumigatus*.

Sven D. Willger[1], Srisombat Puttikamonkul[1], Kwang-Hyung Kim[2], James B. Burritt[3], Nora Grahl[1], Laurel J. Metzler[4], Robert Barbuch[4], Martin Bard[4], Christopher B. Lawrence[2] and Robert A. Cramer Jr. [1]. [1]Department of Veterinary Molecular Biology, [3] Department of Microbiology, Montana State University, Bozeman, MT 59717, USA. [2]Virginia Bioinformatics Institute, Virginia Polytechnic Institute and State University, Blacksburg, VA 24061, USA. [4]Department of Biology, Indiana University-Purdue University, Indianapolis, IN 46202, USA

Aspergillus fumigatus is a saprophytic fungus commonly found in soil and compost piles. In immunocompromised patients it takes on a sinister form as a potentially lethal opportunistic human pathogen. At the site of infection, the significant influx of immune effector cells and the necrosis of tissue by the invading pathogen generates a hypoxic microenvironment in which both the pathogen and host cells must survive. Currently, whether hypoxia adaptation is an important virulence attribute is unknown. Here we report the characterization of a sterol-regulatory element binding protein, SrbA, in *A. fumigatus*. Loss of SrbA results in a mutant strain of the fungus that is incapable of growth in a hypoxic environment and consequently incapable of causing disease in two distinct murine models of invasive pulmonary aspergillosis (IPA). Transcriptional profiling and annotation of genes that are affected by loss of SrbA function implicated that SrbA is involved in maintaining sterol biosynthesis and cell polarity. Further examination of the SrbA null mutant phenotype revealed that SrbA plays a critical role in ergosterol biosynthesis, resistance to the azole class of antifungal drugs, and in maintenance of cell polarity in *A. fumigatus*. Significantly, the SrbA null mutant was highly susceptible to fluconazole and voriconazole. These findings present a new function of SREBP proteins in filamentous fungi, and demonstrate for the first time that hypoxia adaptation is an important virulence attribute of pathogenic molds.

53. Medically important species of *Aspergillus*

Maren A. Klich, USDA/ARS/SRRC, New Orleans, LA, USA. email: Maren.Klich@ars.usda.gov

Aspergilli cause a variety of health problems ranging from mild allergies to life-threatening systemic mycoses. The incidence of mycoses caused by *Aspergillus* (aspergilloses) has risen rapidly as chemotherapy, organ transplants and diseases such as AIDS have increased the immunosuppressed population. The most common *Aspergillus* human pathogens, *A. fumigatus, A. flavus, A. niger* , and *A. terreus,* are also the most frequently occurring species in soils, indicating that the clinical population reflects the natural population. About 40 of the 250 species of *Aspergillus* have been reported as human pathogens. The pathogenic species are not clustered in any one subgeneric taxonomic group. The common trait of most of the pathogenic species is that they grow well at 37 C. Many new species have been described recently based on molecular and physiological data. Some of these are clinically important because they respond differently to therapeutic drugs. The most notable species in this regard is *A. lentulus*, a sibling species of *A. fumigatus*.

54. *nepA* as a possible pathogenicity factor in *A. flavus*

Andrea Dolezal[1], Charles Woloshuk[2], Gary Payne[1]. [1]North Carolina State University, Raleigh, NC [2]Purdue University, West Lafayette, IN

Infection of maize kernels by *Aspergillus flavus*, a pathogen of maize, results in kernel deterioration and contamination with aflatoxin. To identify putative pathogenicity genes, a custom-designed Affymetrix GeneChip DNA microarray was used to follow gene expression in *A. flavus* during infection of maize kernels in the field. Nearly 1,500 fungal genes were more highly expressed in infected living kernels when compared to expression in colonized autoclaved kernels at the same developmental stage. Of these, *nepA* was expressed on average 8 times higher in living kernels. *nepA* belongs to the necrosis inducing protein superfamily (NPP1), which several members in other plant pathogens are known to be involved in pathogenicity. To determine if *nepA* has a role in pathogenicity, the gene was deleted, and the mutant was used in pathogenicity tests. Growth and conidiation of the mutant on the kernel surface appeared sporadic and varied. In contrast, consistent differences in growth within kernels were observed between the mutant and wildtype . The *nepA* deletion mutant appeared to be impeded in growth in the endosperm, while wild type caused necrosis of kernel tissues. Additional experiments are being performed with beta-glucuronidase- expressing strains and histological stains to better define mycelium within kernel tissues. Our initial findings suggest that *nepA* has a role in the pathogenicity of *A. flavus*.

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Yu, Jiujiang 34
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Participants

Nansalmaa Amarsaikhan
Biotechnology
Midle East Technical Universit
Ankara 06531 Turkey
Phone: 903122105641
Fax: 903122102767
Email: nansa89@yahoo.com

Mikael R Andersen, PhD
Systems Biology - CMB
Tech. University of Denmark
Building 223
Søltofts Plads
Kgs Lyngby 2800 Denmark
Phone: 4545252675
Email: mr@bio.dtu.dk

Alex Andrianopoulos, PhD
Dept Genetics
Univ Melbourne
Dept Genetics, Univ Melbourne
Melbourne VIC 3010
VIC Australia
Phone: 61 3-9344-5164
Fax: 61 3-9344-5139
Email: alex.a@unimelb.edu.au

Masanori Arita, PhD
Computational Biology
University of Tokyo
CB05
Kashiwanoha 5-1-5
Kashiwa 277-8561
Chiba Japan
Phone: (81)4 7136 3988
Email: arita@k.u-tokyo.ac.jp

Martha Beasley Arnaud, PhD
Dept Gen
Stanford Univ Sch Med
S201B Grant Bldg, 300 Pasteur
Stanford CA 94305-5120
Phone: (650) 736-0075
Fax: (650) 724-3701
Email: arnaudm@genome.stanford.edu

Herbert N Arst, PhD, AB
Microbiology
Imperial College London
Flowers Bldg, Armstrong Rd
London SW7 2AZ
United Kingdom
Phone: 44 20 7594-2073
Fax: 44 20 7594-3095
Email: h.arst@imperial.ac.uk

Scott E Baker, PhD
Chem & Biol Process Dev
Pacific Northwest Natl Lab
902 Battelle Blvd
Richland WA 99352
Phone: (509) 372-4759
Fax: (509) 372-4732
Email: scott.baker@pnl.govll

Elena V Bashkirova, PHD
Integrative Biology
Novozymes.Inc
1445 Drew Avenue
Davis CA 95618
Phone: (530)750 57 70
Fax: (530)758-0317
Email: ebas@novozymes.com

Ozgur Bayram, PhD
Mol.Microbiology & Genetics
Inst Microbiology & Genetics
Grisebachst 8
Goettingen 37077
Germany
Phone: 49 5513-93818
Fax: 49 5513-93820
Email: obayram@gwdg.de

Judith Behnsen
Hans-Knoell-Institut
11 a
Beutenbergstr.
Jena 07745
Thuringia Germany
Phone: +49364153211003
Fax: +495320802
Email: judith.behnsen@hki-jena.del

Joan W Bennett, PhD
Dept Plant Biol & Pathology
Rutgers Univ
Foran Hall, Rm 296C
59 Dudley Rd
New Brunswick NJ 08901
Phone: (732) 932-9375 X386
Fax: 732-932-9441
Email: profmycogirl@yahoo.com

Sebastian C. Bergmann
Molecular and Applied Microbio
Hans Knoell Institute
11 a Beutenbergstrasse
Jena 07745
Thuringia Germany
Phone: +4936415321003
Fax: +4936415320802
Email: sebastian.bergmann@hki-jena.de

Randy M Berka, PhD
Dept Integrative Biol
Novozymes, Inc
1445 Drew Ave
Davis CA 95618
Phone: (530) 757-4974
Fax: (530) 758-0317
Email: ramb@novozymes.com

Gerhard H Braus, PhD
Microbiol/Gen, Molec Microbiol
Georg-August Univ
Grisebachstr 8
Gottingen D-37077 Germany
Phone: 49 551-39-3771
Fax: 49 551-39-3330
Email: gbraus@gwdg.de

Susanna A Braus-Stromeyer, PhD
Microbiology & Genetics
Georg-August-University
Grisebachstr. 8
Goettingen 37075
Niedersachsen Germany
Phone: (0551) 393817
Email: sbraus@gwdg.del

Matthias Brock
Microbiell Biochemistry
Hans-Knoell-Institut
11 a Beutenbergstrasse
Jena 07745
Thuringia Germany
Phone: +4936415321710
Fax: +4936415320802
Email: matthias.brock@hki-jena.de

Daren W Brown, PhD
Mycotoxin Research Group
USDA/ARS
1815 N University Ave
Peoria IL 61604
Phone: (309) 681-6230
Fax: (309) 681-6689
Email: browndw@ncaur.usda.gov

Kenneth S Bruno, BS PhD
Fungal Biotech, Team, CBPDG
Pacific Northwest National Lab
MSIN P8-60
902 Battelle Blvd
Richland WA 99352
Phone: (509) 375-4438
Fax: (509) 372-4732
Email: kenneth.bruno@pnl.govl

Mark X Caddick, BS PhD
Dept Biological Sci
Univ Liverpool
Biosciences Bldg, Crown St
Liverpool L69 7ZB
United Kingdom
Phone: 44 151-795-4566
Fax: 44 151-795-4410
Email: caddick@liv.ac.ukll

David Canovas
Genetics
Universidad de Sevilla
Avda de Reina Mercedes, 6
Sevilla 41012 Spain
Phone: + 34 954 55 59 47
Fax: + 34 954 55 71
Email: davidc@us.es

Neuza Carvalho
Molecular Microbiology
Clusius Laboratory
64 Wassenarseweg
Leiden 2333 AL
Netherlands
Phone: 0031615185921
Email: n.carvalho@biology.leidenuniv.nll

Yun C Chang, PhD
NIADI/DIR/LCI/MMS
NIH
10 Center Dr,Bldg10 11N234
Bethesda MD 20892
Phone: (301) 496-8839
Fax: (301) 480-3458
Email: yc3z@nih.gov

Yi-Ming Chiang, PhD
Pharmaceutical Sciences
USC School of Pharmacy
PSC406A
1985 Zonal Avenue
Los Angeles CA 90033
Phone: (626) 348-9156
Email: ymchiangster@gmail.com

Marcus C Chibucos, Ph.D.
Institute for Genome Sciences
University of Maryland
801 W. Baltimore Street
Baltimore MD 21201
Phone: (410) 706-0885
Email: mchibucos@som.umaryland.edu

Dawoon Chung, MS
Plant pathology
Texas A&M University
2132 Peterson Bldg. 120
2132 TAMU
College Station TX 77843
Phone: (979) 845-7547
Fax: (979) 845-6483
Email: dwchung@tamu.edu

Robert A Cramer, PhD
Veterinary Molec Biol
Montana State Univ
PO Box 173610
Bozeman MT 59717
Phone: (406) 994-7467
Fax: 406-994-4303
Email: rcramer@montana.edu

Meryl A Davis, PhD
Dept Genetics
Univ Melbourne
Grattan St, Parkville
Melbourne 3010
Australia
Phone: 613 8344-6266
Fax: 613 8344-5139
Email: m.davis@unimelb.edu.au

Junxin DUAN, Ph.D.
Novozymes China
14 Xinxi Road, Shangdi Zone
Beijing 100085 China
Phone: +861062987888X191
Fax: +861062980085
Email: jdua@novozymes.com

Nigel S Dunn Coleman Prof, PhD
AlerGenetica SL
Instituto Technology Canarias
Plaza de Sixto Machado , 3
Santa Cruz 38360
Tenerife Spain
Phone: 34 629 088 832
Email: ndunncoleman@alergenetica.com

Kenneth C Ehrlich, PhD
Dept Food & Feed Safety
Southern Reg Res Ctr/USDA
1100 RE Lee Blvd, Box 19687
New Orleans LA 70179
Phone: (504) 286-4369
Fax: (504) 286-4419
Email: ken.ehrlich@ars.usda.gov

Natalie D Fedorova, PhD
Infectious Disease
Inst Genomic Research
9704 Medical Ctr Dr
Rockville MD 20850
Phone: (301) 795-7756
Fax: (301) 795-7070
Email: natalief@jcvi.org

Evee Q Fernandez, BS
Integrative Biology
Novozymes
1445 Drew Avenue
Davis CA 95618
Phone: (530) 757-8100
Fax: (530) 758-0317
Email: evfz@novozymes.com

Angelique C Franken, MSc
Molecular microbiology
Leiden University, IBL
Clusius, DK06
Wassenaarseweg 64
Leiden 2333 AL
Netherlands
Phone: +31715274826
Email: a.c.w.franken@biology.leidenuniv.nl

Jens Frisvad, PhD
CMB
Department of Systems Biology
B 221 Soeltofts Plads
Kgs. Lyngby 2800
Denmark
Phone: 45 45252626
Fax: 45 45884922
Email: jcf@bio.dtu.dkl

Graeme S Garvey, Ph.D.
Med. Micro. and Immunology
University of Wisconsin-Madiso
1550 Linden Dr.
Madison WI 53706
Phone: (608) 882 1958
Email: gsgarvey@wisc.edul

Gustavo H Goldman, PhD
Fac Cienc Farm Ribeirao Preto
Univ De Sao Paulo
Av Do Cafe S/N, Ribeirao Preto
Sao Paulo, CEP 14040-903
Brazil
Phone: 55 16-3602-4280
Fax: 55 16-3602-1092
Email: ggoldman@usp.br

Meera Govindaraghavan
The Ohio State University
1519 Neil Avenue Apt F
Columbus OH 43201
Phone: 614 787 7117
Email: govind.meera@gmail.com

Nora Grahl
Veterinary Molecular Biology
Montana State University
PO Box 173610
Bozeman MT 59717
Phone: (406) 994-7468
Email: nora.grahl@montana.edul

Fabrice N Gravelat, Ph.D.
Microbiology and Immunology
McGill University
3775 University st
Montreal H3A2B4
Quebec Canada
Phone: (514) 398-4434
Email: fabrice.gravelat@mcgill.cal

Sara K Gremillion, Ph.D.
Biology
Rhodes College
2000 N. Parkway
Memphis TN 38112
Phone: 901-843-3699
Fax: 901-843-3565
Email: gremillions@rhodes.edu

Dong-Min Han, PhD
Div Life Sci
Wonkwang Univ
344-2 Shinyong-dong, Iksan
Chunbuk 570-749 Korea
Phone: 82 653-850-6220
Fax: 82 653-853-2516
Email: dmhan@wonkwang.ac.kr

Kap-Hoon Han, PhD
Pharmaceutical Engineering
Woosuk Univ
Rm 7226, Science Bldg
Wanju, Jeonbuk 565-701
Korea
Phone: 82 63-290-1427
Fax: 82 63-290-1436
Email: khhan@woosuk.ac.kr

Bjarne G Hansen
Dept. Systems Biology, CMB
DTU
Building 223
Søltoft Plads
Kgs Lyngby 2800
Denmark
Phone: 0045 45252703
Email: bgha@bio.dtu.dkl

Steven D Harris, PhD
Plant Sci Initiative
Univ Nebraska
E126 Beadle Ctr
Lincoln NE 68588-0660
Phone: (402) 472-2938
Fax: (402) 472-3139
Email: sharri1@unlnotes.unl.edu

Rebekka Harting, PhDStudent
Mol. Microbiology and Genetics
Georg August University
Grisebachstr. 8
Goettingen 37077
Germany
Phone: +49 551 393780
Email: rhartin@gwdg.del

Kerstin Helmstaedt, PhD
Inst Microbiology & Genetics
Georg-August University
Grisebachstr 8
Goettingen D-37077
Germany
Phone: 49 551-393772
Fax: 49 551-393820
Email: khelmst@gwdg.de

Terry W Hill, PhD
Department of Biology
Rhodes College
2000 North Parkway
Memphis TN 38112
Phone: (901) 289-9562
Fax: (901) 843-3565
Email: hill@rhodes.edu

Gerald Hofmann, PhD
Fermentation Optimization
Novozymes A/S
BD3.44
Hallas Alle 1
Kalundborg 4400
Denmark
Phone: 0045 44460279
Email: ghof@novozymes.com

Michael J Hynes, PhD
Dept Gen
Univ Melbourne
Grattan
Parkville VIC 3010
Australia
Phone: 61 03-83446239
Fax: 61 03-83445139
Email: mjhynes@unimelb.edu.aul

Loretta Jackson-Hayes, PhD
Dept Chemistry
Rhodes Col
2000 N Parkway
Memphis TN 38112
Phone: (901) 843-3510
Fax: (901) 843-3497
Email: jacksonhayesl@rhodes.edu

Radhika Jain
Molekular and Applied Microbio
Hans Knoell Institute
11 a Beutenbergstr.
Jena 07743
Thuringia Germany
Phone: +4936415321003
Fax: +4936415320802
Email: radhika.jain@hki-jena.de

Qiming Jin, PhD
Novozymes Inc
1445 Drew Ave
Davis CA 95616
Phone: (530) 757-0827
Fax: (530) 758-0317
Email: qmji@novozymes.com

Thomas R Joergensen
Molecular Microbiology
Leiden University
Wassenaarseweg 64
Leiden 2333AL
Netherlands
Phone: 0031715275056
Email: t.r.jorgensen@biology.leidenuniv.nll

Annette H Johansen, Ph.D
Protein Screening
Novozymes A/S
Smørmosevej 11
Bagsvaerd 2880 Denmark
Phone: (0045)44460736
Fax: (0045)44464515
Email: ahjo@novozymes.com

Etta Kafer
23233 52nd Ave
Langley V2Z 2P8
BC Canada
Phone: 604 533-7671
Email: ekafer@sfu.ca

Margaret E Katz, PhD
Dept Molec & Cellular Biol
Univ New England
Trevenna Rd
Armidale 2351
NSW Australia
Phone: 61 2-6773-3016
Fax: 61 2-6773-3267
Email: mkatz@une.edu.aul

Hye-Ryun Kim, MS
Life science
Wonkwang University
Shin-young dong 344-2
Iksan 570-749
Jeonbuk Korea
Phone: 82-19-434-1759
Fax: 82-63-853-2516
Email: etaaa@hanmail.net

Maren Klich
USDA ARS
1100 Robert E. Lee Blvd
New Orleans LA 70124
Phone: 504-286-4361
Email: Maren.Klich@ars.usda.govll

Hideaki Koike, Dr
Res Inst Cell Engineering
AIST Tsukuba Center 6-9
Higashi 1-1-1
Tsukuba 305-8566
Ibaraki Japan
Phone: 81 29-861-6679
Fax: 81 29-861-6174
Email: hi-koike@aist.go.jp

Sven Krappmann, PhD
Res. Center Infect. Diseases
University of Wuerzburg
Roentgenring 11
Wuerzburg D-97070
Germany
Phone: 49 931 31-2153
Fax: 49 931 31-2578
Email: sven.krappmann@uni-wuerzburg.del

Marcia Regina Kress
Mol. Microbiology and Genetics
Georg August University
Grisebachstr. 8
Goettingen 37077
Germany
Phone: +49 551 393780
Email: mkress@gwdg.del

Thanyanuch Kriangkripipat, MS
Dept Plant Biol
Univ Georgia
120 Carlton St
Athens GA 30602
Phone: (706) 542-6026
Fax: (706) 542-1805
Email: tkriang@plantbio.uga.edu

Jennifer R Larson, PhD
Molecular Genetics
Ohio State Univ
984 Biological Sciences Bldg.
496 W 12th Ave
Columbus OH 43210
Phone: (614) 247-6873
Fax: (614) 247-6845
Email: larson.315@osu.edu

Kuan-Han Lee, Ph.D.
Department of Pharmacology and
University of Southern Califor
PSC 406A
1985 Zonal Ave
Los Angeles CA 90089-9121
Phone: (626)6881848
Fax: (323)4421390
Email: kuanhanl@usc.edu

Aalexander Lin, M.S. stude
Pharmaceutical Sciences
USC School of Pharmacy
PSC406A
1985 Zonal Avenue
Los Angeles CA 90033
Phone: (310)310-1833
Email: linalexa@usc.edu

Xiaorong Lin, PhD
Department of Biology
Texas A & M Univeristy
3258 TAMU
BSBW 435
College Station TX 77843
Phone: 979-845-7274
Fax: 979-845-2891
Email: xlin@mail.bio.tamu.edu

Hong Liu, Ph. D
Infectious Diseases
LA Biomedical Research
RB2 #227
1124 West Carson
Torrance CA 90502
Phone: 310-222-6427
Email: hliu@labiomed.orgl

Ye Liu
Novozymes China
14 Xinxi Road, Shangdi Zone
Beijing 100085
China
Phone: 0086-10-62987888-221
Fax: 0086-1062980085
Email: liuy@novozymes.com

Zheng Liu Dr., Ph.D.
Fungal Diversity
Novozymes China
14 Xinxi Lu, Shangdi Zone
Beijing 100085
China
Phone: +861062987888X253
Fax: +861062980085
Email: lizg@novozymes.com

Hsien-Chun Lo, MS
Pharmaceutical Sciences
USC
PSC 406
1985 Zonal Ave
Los Angeles CA 90033
Phone: (626)689-5883
Email: usckenlo@gmail.com

Darlene Margaret Loprete, PhD
Chemistry
Rhodes College
2000 N Parkway
Memphis TN 38112
Phone: (901) 843-3905
Fax: (901) 278-4403
Email: loprete@rhodes.edu

Patrick Lorenz, PhD
R&D
AB Enzymes
Feldbergstrasse 78
Darmstadt 64293
Hessen Germany
Phone: +49-6151-3680-380
Email: patrick.lorenz@abenzymes.com

Mette Lübeck, PhD
Biotechnology and Bioenergy
Aalborg University Copenhagen
Lautrupvang 15
Ballerup 2750
Denmark
Phone: +45 9940 2589
Email: mel@bio.aau.dk

Peter S Lübeck, Ph.D.
Biotechnology and Bioenergy
Aalborg University Copenhagen
15 Lautrupvang
Ballerup 2750
Denmark
Phone: +45 99 40 25 90
Email: psl@bio.aau.dk

Jane E Mabey Gilsenan, PhD
School of Medicine
University of Manchester
Education & Research Centre
Wythenshawe Hospital
Manchester M23 9LT
United Kingdom
Phone: (+44)161 291 5901
Email: jane.gilsenan@manchester.ac.ukl

Masayuki Machida, Dr
Inst Biol Res Func
Natl Inst Advanced Sci Tech
Higashi 1-1, Tsukuba
Ibaraki 305-8566 Japan
Phone: 81 298-61-6164
Fax: 81 298-61-6174
Email: m.machida@aist.go.jp

Iran Malavazi, PhD
Pharmacy
FCFRP University of Sao Paulo
Bloco Q
Av do Cafe s/n Monte Alegre
Ribeirao Preto 14040-903
Sao Paulo Brazil
Phone: 55 16-36024311
Fax: 55 16-36024280
Email: imalavazi@gmail.com

Gregory S May, PhD
Dept Lab Med, Unit 54
Univ Texas/MD Anderson CA Ctr
1515 Holcombe Blvd
Houston TX 77030
Phone: (713) 745-1945
Fax: (713) 792-8460
Email: gsmay@mdanderson.org

Kevin McCluskey, PhD
Fungal Genetics Stock Center
Univ Missouri, Kansas City
5007 Rockhill Rd
Kansas City MO 64110
Phone: (816) 235-6484
Fax: (816) 235-6561
Email: mccluskeyk@umkc.edu

Petter Melin, PhD
Dept Microbiology
SLU
Genetikvagen 5
Uppsala S-750 07
Sweden
Phone: 46 18673398
Fax: 46 18673392
Email: petter.melin@mikrob.slu.sell

Bruce L Miller, PhD
Microbiol,Molec Biol,Biochem
Univ Idaho
P.O. Box 3052
Ash St
Moscow ID 83844-3052
Phone: (208) 885-7247
Fax: (208) 885-6518
Email: bmiller@uidaho.edu

Michelle C Momany, PhD
Dept Plant Biol
Univ Georgia
2502 Plant Sci
Athens GA 30602
Phone: (706) 542-2014
Fax: (706) 542-1805
Email: momany@plantbio.uga.edu

Uffe H Mortensen, PhD
Dept. of Systems Biology - CMB
Tech. University of Denmark
b223
Søltofts Plads
Lyngby 2800
Denmark
Phone: 45 4525 2701
Email: um@bio.dtu.dkl

Jakob B Nielsen, PhD
CMB
Dept. of Systems Biology - DTU
Soeltofts Plads
Kgs Lyngby 2800
Denmark
Phone: 45 45252657
Fax: 45 45884148
Email: jbn@bio.dtu.dkl

Michael L Nielsen, PhD
Dept. of Systems Biology - CMB
Tech. University of Denmark
b223
Soeltofts Plads
Kgs Lyngby 2800
Denmark
Phone: 45 4525-2700
Fax: 45 4588-4148
Email: mln@bio.dtu.dkl

Morten Thrane Nielsen Mr, Master
Department of Systems Biology
Technical University of Denmar
Lyngby 2800
Denmark
Phone: 004532680001
Fax: 004545884922
Email: mthrane82@gmail.com

William C Nierman, PhD
Infectious Diseases
J Craig Venter Inst
9704 Med Ctr Dr
Rockville MD 20850
Phone: (301) 795-7559
Fax: (301) 838-0209
Email: wnierman@jcvi.orgl

Marie Nishimura, PhD
Plant-Microb Interact Unit
Natl Inst Agrobiol Sciences
2-1-2, Kan-non dai, Tsukuba
Ibaraki 305-8602
Japan
Phone: 81 298-38-8461
Fax: 81 298-38-7408
Email: marie@affrc.go.jp

Céline M O'Gorman Miss, BSc (Hons)
UCD School of Biology
University College Dublin
Belfield
Dublin 4 Dublin 4
Ireland
Phone: +353-1-7162350
Fax: +353-1-7161153
Email: celine.ogorman@ucd.ie

Berl R Oakley, PhD
Dept Molec Biosci
Univ Kansas
Haworth Hall
1200 Sunnyside Ave
Lawrence KS 66045
Phone: (785) 864-8170
Email: boakley@ku.edul

Ken Oda, PhD
Plant Biol
Univ Georgia
1505 Miller Plant Sci
Athens GA 30602-7271
Phone: (706) 542-6026
Fax: (706) 542-1805
Email: koda@plantbio.uga.edu

Hironobu Ogasawara, PhD
Dept Enz & Microbiol
Res Inst Food & Brewing/Akita
4-26,aza-sanuki,Araya-machi
Akita 010-1623 Japan
Phone: +81-18-888-2000
Fax: +81-18-888-2008
Email: hironobu@arif.pref.akita.jp

Carsten Lillelund Olsen Sr, PhD
Fungal Gene Technology
Novozymes A/S
Brudelysvej 26, 1U2.26
Bagsvaerd 2880 Denmark
Phone: +045 44466013
Email: cols@novozymes.com

Stephen Osmani
Molecular Genetics
Ohio State University
Columbus OH 43210
Phone: 614 247 6791
Email: osmani.2@osu.edu

Thomas Ostenfeld Larsen, PhD
Department of Systems Biology
Technical University of Denmar
Søltofts Plads
Kgs. Lyngby 2800 Denmark
Phone: +45 45252632
Fax: +45 45884922
Email: tol@bio.dtu.dk

Gary A Payne, Ph. D.
Dept Plant Pathology
North Carolina State Univ
Box 7567
Raleigh NC 27695-7567
Phone: (919) 515-6994
Fax: (919) 513-0024
Email: gary_payne@ncsu.edu

Andrea M Prynych, BSc (Hons)
Genetics
University of Melbourne
Melbourne 3010
Victoria Australia
Phone: +61 3 8344-9878
Fax: +61 3 8344-5139
Email: a.prynych@pgrad.unimelb.edu.au

Peter J Punt, Dr
Dept Microbiology
TNO Quality of Life
PO Box 360, Utrechtseweg 48
Zeist 3700 AJ Netherlands
Phone: 31 30-6944-463
Fax: 31 30-6944-466
Email: peter.punt@tno.nl

Arthur F Ram
Molecular Microbiology
Institute of Biology Leiden
Clusius building
Wassenaarseweg 64
Leiden 2333 AL Netherlands
Phone: 31 71 5274914
Email: a.f.j.ram@biology.leidenuniv.nl

Christian Rank, MSc
System Biology
CMB
Søltofts Plads
Kgs. Lyngby 2800 Denmark
Phone: +4545252725
Email: cr@bio.dtu.dkl

Samara L Reck-Peterson, Ph.D.
Cell Biology
Harvard Medical School
240 Longwood Avenue
Boston MA 02115
Phone: 617-432-71798
Fax: 617-432-7193
Email: reck-peterson@hms.harvard.edu

Carmen Ruger-Herreros, Graduate
Genetica
Universidad de Sevilla
Lab 105
Avenida Reina Mercedes, 6
Sevilla 41012 Spain
Phone: +34954556473
Fax: +34954557104
Email: carmenruger@us.esl

Margarita Salazar, MSc
Chemical & Biological Eng.
Chalmers University of Technol
10 Kemivägen
Gothenburg SE-412 96
Sweden
Phone: 46 (31)7723876
Fax: 46 (31) 7723801
Email: margarita.salazar@chalmers.se

James F Sanchez, B.S, M.S
Pharmaceutical Sciences
University of Southern California
PSC 716
Los Angeles CA 91030
Phone: (562) 343-3815
Email: jfsanche@usc.edu

Claudio Scazzocchio, Ph. D.
Inst. Génétique Microbiologie
Univ Paris 11
Orsay 91405 France
Phone: int 33 1 69070121
Email: scazzocchio@igmors.u-psud.fr

Felicitas Schoebel
Hans Knoell Institute
11a Beutenbergstrasse
Jena 07745
Thuringia Germany
Phone: +4936415321003
Fax: +4936415320802
Email: felicitas.schoebel@hki-jena.de

Patricia M. Shaffer, Ph.D.
Chemistry & Biochemistry
University of San Diego
FH#102
5998 Alcala Park
san Diego CA 92110-2492
Phone: (619)260-4034
Email: shaffer@sandiego.edu

Brian Douglas Shaw, PhD
Plant Pathology & Microbiology
Texas A&M University
2132 TAMU
College Station TX 77843
Phone: (979) 862-7518
Fax: (979) 845-6483
Email: bdshaw@tamu.edul

Gavin Sherlock, PhD
Dept Gen
Stanford Univ
300 Pasteur Dr
Stanford CA 94305-5120
Phone: (650) 498-6012
Fax: (650) 724-3701
Email: sherlock@genome.stanford.edu

Kyle Smith, BS
Chemistry & Biochem
Univ San Diego
FH#102
5998 Alcala Park
San Diego CA 92110-2492
Phone: (619) 260-4034
Email: shaffer@sandiego.edul

Annette Sorensen, MSc
Center for Biotech &Bioenergy
Cph Institute of Techn., AAU
Lautrupvang 15
Ballerup DK-2750
Denmark
Phone: 509-987-4893
Email: aso@bio.aau.dkl

Joseph Strauss, Dr
Fungal Genomics Unit
ARC & BOKU University, Vienna
Muthgasse 18
Vienna 1190
Bangladesh
Phone: 43 1-360066720
Email: joseph.strauss@boku.ac.at

Janyce A Sugui, PhD
NIAID/NIH
Bldg.10-room 11N234
9000 Rockville Pike
Bethesda MD 20892
Phone: (301) 496-8946
Email: jsugui@niaid.nih.govl

Asa Svanstrom
Microbiology
Swedish Univ of Agricult Sci
PO Box 7025
Uppsala S-750 07 Sweden
Phone: 4618673327
Email: ana.svanstrom@mikrob.slu.sell

Edyta Szewczyk, PhD
Res. Center Infect. Diseases
University of Wuerzburg
Roentgenring 11
Wuerzburg 97070
Germany
Phone: +49 (0)931312153
Email: edyta.szewczyk@uni-wuerzburg.del

Shinobu Takagi
Research & Dev/Strain Dev
Novozymes Japan, Ltd
CB-6, 3 Nakase 1-chome, Mihama
Chiba 261-8501
Japan
Phone: 81 43-296-6770
Fax: 81 43-296-6760
Email: st@novozymes.com

Kaeling Tan, PhD
Cell Biol
Harvard Medical
Rm529
240 Longwood Ave
Boston MA 02115
Phone: (617) 432-7177
Email: kaeling_tan@hms.harvard.edu

Richard B Todd, PhD
Department of Plant Pathology
Kansas State University
4024 Throckmorton Hall
Manhattan KS 66506
Phone: 785-532-0962
Fax: 785-532-5692
Email: rbtodd@ksu.edu

Srijana Upadhyay, Master
Department of Plant Pathology
Texas A&M University
2132 peterson building room120
2132 TAMU
College station TX 77843
Phone: 979-845-7547
Fax: 979-845-6483
Email: srijanaupadhyay@tamu.edu

Amber D Valencia
Chemistry
University of Southern Califor
Los Angeles CA 90089
Phone: (562) 480-8195
Email: amber.valencia@gmail.com

Hans van den Brink
Enzymes
Chr Hansen A/S
Boge Alle 10-12
Horsholm DK-2970
Denmark
Phone: 45 4574-8455
Fax: 45 4574-8994
Email: dkhvb@chr-hansen.com

Cees A M J J van den Hondel, Prof Dr
Dept Mol. Microbiol.,Inst Biol
Univ Leiden
Wassenaarseweg 64
Leiden 2333 AL
Netherlands
Phone: 31 71 527-4745
Email:
c.a.m.van.den.hondel@biology.leidenuniv.nl

Martin Voedisch
Molecular and Applied Microbio
Hans Knoell Institute
11 a
Beutenbergstrasse
Jena 07745
Thuringia
Germany
Phone: +4936415321003
Fax: +4936415320802
Email: martin.voedisch@hki-jena.del

Chih-Li Wang, MS
Plant Pathology&Microbiology
Texas A&M University
Peterson Building
2132 TAMU
College Station TX 77843
Phone: 979-845-7547
Fax: 979-845-6483
Email: chih-tamu@tamu.edu

Clay Wang, PhD
Pharma. Sci. and Chemistry
Univ. of Southern California
1985 Zonal Ave PSC 406A
Los Angeles CA 90089
Phone: 323-442-1670
Email: clayw@usc.edu

Srisombat Wannaying
Veterinary Molecular Biology
Montana State University
PO Box 173610
Bozeman MT 59717
Phone: (406) 994-7468
Email:
srisombat.wannaying@myportal.montana.edu

Aric E Wiest, MS
Fungal Genetics Stock Center
University of Missouri, KC
5007 Rockhill Rd
KANSAS CITY MO 64110
Phone: 816-235-6485
Fax: 816-235-6561
Email: wiesta@umkc.edu

Sven D Willger, PhD
Veterinary Molecular Biology
Montana State University
PO Box 173610
Bozeman MT 59717
Phone: (406)994-7468
Email: sven.willger@montana.edu

Jennifer R Wortman
Institute for Genome Sciences
Univ. of MD School of Medicine
801 W. Baltimore St.
Baltimore MD 21201
Phone: 410-706-6784
Email: jwortman@som.umaryland.edu

Yi Xiong
Molec Gen
Ohio State Univ
484 W 12th Ave
Columbus OH 43210
Phone: (614) 316-0635
Email: xiong.29@osu.edu

Debbie Sue Yaver, PhD
Fungal Expression
Novozymes Inc
1445 Drew Ave
Davis CA 95618
Phone: (530) 757-4993
Fax: (530) 758-0317
Email: dsy@novozymes.com

Jiujiang Yu, Ph.D.
USDA/ARS
Southern Regional Res Ctr
1100 Robert E Lee Blvd
New Orleans LA 70124
Phone: (301) 760-9157
Fax: (504) 286-4419
Email: jiujiang.yu@ars.usda.gov