The program of the Tenth Annual Southeast Regional Collaborative Access Team (SER-CAT) Meeting and Structural Biology Symposium held at Florida State University’s Chemical Sciences Laboratory on Friday, March 15, 2013 between 8 AM and 6 PM.
SYMPOSIUM SPONSORS
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# Tenth Annual SER-CAT Meeting
## STRUCTURAL BIOLOGY SYMPOSIUM PROGRAM
### Symposium schedule

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<th>Start Time</th>
<th>End Time</th>
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<tr>
<td><strong>Friday, March 15, 2013</strong></td>
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<tr>
<td>Shuttle from Hotel</td>
<td>8:00 AM</td>
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<td>Coffee</td>
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<td>8:45 AM</td>
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<tr>
<td>Welcome Address</td>
<td>8:45 AM</td>
<td>8:55 AM</td>
<td>Ross Ellington, Associate VP Research</td>
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<td>Announcements</td>
<td>8:55 AM</td>
<td>9:00 AM</td>
<td>Hong Li</td>
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**Session 1: Interesting Structures** Chair: Beth Stroupe, FSU

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<tr>
<td>Steve White, St Jude Children's Research Hospital</td>
<td>9:00 AM</td>
<td>9:20 AM</td>
<td>Synchrotron radiation resolves an 80 year old question in medicinal chemistry - the mechanism of sulfa drugs</td>
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<td>Christine Dunham, Emory University</td>
<td>9:20 AM</td>
<td>9:40 AM</td>
<td>Structural studies of ribosome-dependent toxins active during the stringent response</td>
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<td>SER-CAT Outstanding Science Award</td>
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<td>Structural basis for iron piracy by pathogenic Neisseria</td>
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<td>Susan Buchanan, NIDDK-NIH</td>
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<tr>
<td>SER-CAT Young Investigator Award</td>
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<td>10:15 AM</td>
<td>Repair of 5'-topoisomerase II-DNA adducts by mammalian tyrosyl-DNA phosphodiesterase 2</td>
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<td>Matthew J. Schellenberg, NIEHS-NIH</td>
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<tr>
<td><strong>Coffee Break</strong></td>
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**Session 2: Interesting Structure Methods** Chair: Piotr Fajer, FSU

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<tr>
<td>Art Edison, UF</td>
<td>10:35 AM</td>
<td>10:55 AM</td>
<td>High Sensitivity NMR for Metabolomics and Natural Products</td>
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<tr>
<td>Alan Marshall, FSU</td>
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<td>11:15 AM</td>
<td>Understanding Drug Action and Drug Resistance from Their Effects on Protein Receptor Structure</td>
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<td>Scott Stagg, FSU</td>
<td>11:15 AM</td>
<td>11:35 AM</td>
<td>Progress Toward High-Throughput High-Resolution Electron Microscopy</td>
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<tr>
<td><strong>Lunch Break &amp; Group Photo</strong></td>
<td>11:30 AM</td>
<td>12:30 PM</td>
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<tr>
<td><strong>Posters &amp; Poster Award</strong></td>
<td>12:30 PM</td>
<td>2:00 PM</td>
<td>Poster and EM Facility Tour</td>
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**Session 4: Beamline Workshop** Albert Fu & John Chrzas

| Beamline Workshop 1                         | 2:00 PM    | 2:30 PM  | Beamline Alignment & Fluorescence Scan                                |
| Beamline Workshop 2                         | 2:30 PM    | 3:00 PM  | Sample Alignment & Sample Rastering                                  |
| Beamline Workshop 3                         | 3:00 PM    | 3:30 PM  | Helical Data Collection                                               |
| Beamline Workshop 4                         | 3:30 PM    | 4:00 PM  | Mini-Beam for Small Crystal or Special Case                           |
| **Coffee Break**                            | 4:00 PM    | 4:20 PM  |                                                                      |
| Beamline Workshop 5                         | 4:20 PM    | 4:45 PM  | Data Collection Strategy in a Larger View                             |
| Beamline Workshop 6                         | 4:45 PM    | 5:15 PM  | Data Reduction with Various Programs                                  |
| Beamline Workshop 7                         | 5:15 PM    | 5:30 PM  | Seeking Help with Remote Collection                                  |
| Shuttle to Mag Lab (NHMFL)                  | 5:30 PM    |          |                                                                      |

**Evening Program [NHMFL Lobby]**

| **Evening Program and Banquet**             | 6:00 PM    | 9:00 PM  | Jazz Concert and Dinner                                              |
| Shuttle back to Hotel                       | 8:45 PM    |          |                                                                      |
GROUP PHOTO
A group photo will be taken during the Lunch Break.

TOUR OF FEI TITAN KRIOS & EM FACILITY
There will be an opportunity for the participants to take a tour of Biological Science Imaging Resources (http://bsir.bio.fsu.edu) during the Poster Session. All those interested in taking the tour are requested to assemble at the front lobby of Chemical Science Laboratory around 1PM. Dr Duncan Sousa (dsousa@fsu.edu | 850-645-9317) will be your tour guide.

SHUTTLE SERVICE
There will be shuttle service between the Double Tree Hilton Hotel and Symposium site (Chemical Sciences Laboratory Auditorium) beginning around 7:45 AM on Friday, March 15, 2013. There will be two runs: the first one will be at 7:45 AM starting at the Hotel and the second one will be around 8:10 AM at the Hotel. There will also be shuttle service between the Mag Lab and the Double Tree Hilton Hotel beginning around 8:45 PM on Friday, March 15, 2013. The first one will be at 8:45 PM starting at the Mag Lab lobby and the second one will be around 9:05 PM at the Mag Lab lobby.

EVENING PROGRAM
The evening program will be a Jazz Concert and Banquet at the National High Magnetic Field Laboratory. There will be a shuttle service between the Symposium site (CSL Auditorium) and Mag Lab for the evening program. The first shuttle will start at 5:45 PM at CSL and the second run will be around 6:05 PM at CSL.

MAP OF SYMPOSIUM SITE

Map of the local area surrounding Chemical Sciences Laboratory (CSL). Event Parking is also shown.
FSU CAMPUS & MAG LAB MAPS

FSU Campus Map: [http://parking.fsu.edu/Parking/Campus Parking Map/CampusMap2012-13.pdf](http://parking.fsu.edu/Parking/Campus Parking Map/CampusMap2012-13.pdf)

Mag Lab floor plan. [http://magnet.fsu.edu/about/visit.html](http://magnet.fsu.edu/about/visit.html)
A Non-Heme Iron-Dependent Redox Regulator of the NF-κB Transcription Factor
Shingo Esaki, Department of Chemistry, Georgia State University, Atlanta, GA 30303-2924, USA

Pirin is a nuclear non-heme Fe protein of unknown function present in all human tissues. Here, we describe that pirin acts as a redox sensor of the NF-κB transcription factor, a critical mediator of intracellular signaling that has been linked with cellular responses to pro-inflammatory signals and controls the expression of a vast array of genes involved in immune and stress responses. Our spectroscopic results show that the ferric, not ferrous, form of pirin substantially facilitates binding of NF-κB proteins to target κB genes, which suggests pirin performs a redox sensing role in NF-κB regulation. The molecular mechanism of such a metal identity and redox state-dependent regulation is revealed by our structural studies of pirin. The Fe center is shown to play an allosteric role on an R-shaped surface area that has two distinct conformations based on the identity and the formal redox state of the metal. We show that the R-shaped area is the active site of pirin responsible for modulating NF-κB DNA binding properties. We propose that the non-heme Fe protein pirin provides a reversible functional switch that enables pirin to activate NF-κB to respond to the changes of redox levels in cell nucleus.

Characterization of the activity of a CRISPR nuclease from S. epidermidis
Nancy F. Ramia, Li Tang and Hong Li Inst Mol Biophysics, Florida state university, Tallahassee, FL 32306

CRISPR is an adaptive immune system in Prokaryotes which protects them from invading phages and conjugative plasmids through RNA mediated cleavage of foreign nucleic acids. The methods of nucleic acids interference vary widely among the known CRISPR systems but all are ribonucleoprotein particles that cleave either RNA or DNA. Identification of the subunits having nucleic acid cleavage activities is the first step towards understanding the biochemical aspects of the interference mechanism. Staphylococcus epidermidis was shown to interfere with invading DNA by in vivo biochemical studies. However, the molecular mechanism responsible for this functional step is completely unknown. Staphylococcus epidermidis belongs to the CRISPR subtype III-B characterized by presence of the cas10 family of proteins member, Csm1. Cas10 family of proteins are predicted to be enzymatically active in type III CRISPR systems. Csm1 has a permuted HD, zinc finger, and a polymerase-like palm domain and is thus a possible candidate for the DNA cleaving subunit. In this work we report the first in vitro biochemical characterization of Staphylococcus epidermidis Csm1 protein. Our data show that Csm1 cleaves single-stranded DNA in a divalent-metal dependent manner. Csm1 cleaves ssDNA exonucleolytically in a 3'-5' direction, and does not cleave blunt-ends dsDNA. Mutations of the strictly conserved Aspartate residues in its polymerase-like palm but not its HD abolished DNA cleaving activity. This work suggests Csm1 might work with other Csm proteins in the context of an effector complex to degrade invading DNA.

Recognition and Cleavage of a Nonstructured CRISPR RNA by Its Processing Endoribonuclease Cas6
Yaming Shao1, and Hong Li1,2 Inst Mol Biophysics, 2Department of Chemistry and Biochemistry, Florida State University, Tallahassee, FL 32306 USA

Bacteria and archaea employ a strategy based on clustered regularly interspaced palindromic repeats (CRISPRs) to defend themselves against harmful nucleic acids. CRISPRs are genetic loci of identical repeats interspaced by unique spacer sequences derived from past Infections. The step of crRNA biogenesis depends on the activity of the endonuclease Cas6 that cleaves within the repeat region of the repeat-spacer array transcript to yield individual spacer RNA flanked by portions of the repeat. Mature crRNA subsequently assembles with other Cas proteins into effector complexes. These complexes target invading DNA or RNA for destruction. We solved the complex structure of a processing endoribonuclease bound with a noncleavable RNA. The enzyme stabilizes a short RNA stem-loop structure near the cleavage site and cleaves the phosphodiester bond using an active site comprised of arginine and lysine residues.

Structural Characterization of the CRISPR-Associated Cmr Effector Complex
Alexis Cocozaki,a Yaming Shao, Michael Spilman,a Nancy Ramia,a Rebecca Ternsb, Michael Ternsb, Scott Stagg,a and Hong Li,a Institute of Molecular Biophysics, Florida State University, 91 Chieftan Way, Tallahassee FL 32304, U.S.A.b, Departments of Biochemistry and Molecular Biology, and Genetics, University of Georgia, Athens GA 30602, U.S.A.

The Clustered Regularly Interspaced Short Palindromic Repeat (CRISPR) immunity system defends bacteria and archaea against viruses and plasmids through RNA-guided invader DNA or RNA cleavage. Short sequences are acquired from the invader and inserted as spacers into an array of repeat-spacer units. The CRISPR array is transcribed into a long RNA that is processed by CRISPR-associated proteins into individual guide units (crRNA).
Effect of ribonucleoprotein complexes use crRNAs to recognize and cleave the invader’s DNA or RNA. Although all CRISPR systems appear to share the same biological function of RNA-mediated defense, the ribonucleoprotein complexes involved are highly diverse in composition and their mechanisms of activity are beginning to be elucidated. The Cmr RNP complex of *P. furiosus* was the first example of a CRISPR effector complex that targets RNA exclusively. The Cmr complex is composed of six proteins (Cmr1-6). Although the Cmr complex’s crRNA-guided RNA cleavage activity was demonstrated biochemically, the details of the assembly of the complex and the recognition and cleavage of the target are largely unknown. The largest component of the Cmr complex, Cmr2, contains an HD domain and a metal and nucleotide-binding site. Contrary to previous predictions, the HD domain of Cmr2 and its nucleotide-binding site do not play essential roles in the cleavage activity of the Cmr complex. This raised the question of the role of Cmr2 and other Cmr proteins. We solved the structure of Cmr2 bound to Cmr3. Both Cmr2 and Cmr3 are essential for the Cmr complex’s crRNA-guided cleavage activity. Mutagenesis experiments and docking of this structure into a low-resolution structure of the full Cmr RNP complex suggest an essential role for Cmr2 and Cmr3 in the assembly of the full RNP complex.

The Power of Two: The Contribution of Arg51 and Arg239* to the Catalytic Mechanism in a Zinc-dependent Decarboxylase

Lu Huo, Ian Davis, Lirong Chen, and Aimin Liu*, Dept Chemistry, Georgia State University, Atlanta, GA 30303

α-Amino-β-carboxymuconic-ε-semialdehyde decarboxylase (ACMSD) is proposed to be a prototypical member of a new protein subfamily in the amidohydrolase superfamily representing a novel non-hydrolytic C-C bond breaking activity. In a previous study, we reported the structure of ACMSD from *Pseudomonas fluorescence* (PfACMSD) as a dimer. Two conserved arginine residues in the putative substrate binding pocket of ACMSD, Arg51 and Arg239*, have attracted our attention. Arg239* intrudes into the active site from the neighboring protomer chain. The two arginine residues seem properly positioned to stabilize the substrate by providing ionic interactions with its two carboxyl groups. However a new structure of the human enzyme bound with a glycolic metabolic intermediate shows a monomeric structure. This observation opens a new question as to whether or not dimerization is required for ACMSD activity. This work seeks to answer this question and elucidate the roles of the Arg residues. The elution profile of PfACMSD from size exclusion chromatography shows that the protein is in equilibrium between monomer and dimer forms in solution. The specific activity of PfACMSD is seen to increase with greater stock protein concentrations. In this study, Arg51 and Arg239* were each mutated to alanine and lysine, and all four mutants were purified as soluble, inactive proteins. However, when R51A and R239A* were mixed, decarboxylation activity slowly recovered. The heterodimer hybridization experiment strongly supports that the active state of PfACMSD is its dimer form. The crystal structure of a heterodimer obtained by mixing R51A and R239A* mutants was solved to 2.0 Å resolution and will be presented at the conference. We also determined structure of human ACMSD and it is in the dimeric state. Together, these data reveal the precise role of the two arginine residues in the catalytic mechanism.

Enzymes in the Kynurenine Pathway

Ian Davis, Fange Liu, Lu Huo, and Aimin Liu, Georgia State University, Department of Chemistry, Atlanta, GA 30303

In mammals there are two separate pathways by which tryptophan can be metabolized, the serotonin and the kynurenine. Though the serotonin pathway is well studied, it accounts for only a small portion of tryptophan metabolic flux. The bulk of tryptophan not used for protein synthesis is catabolized though the kynurenine pathway (KP), a pathway with neuroactive intermediates such as kynurenic acid and quinolinic acid (QA). The kynurenine pathway is of interest to study because it is the sole source of endogenous QA, a compound which has been shown to be an agonist of N-methyl-D-aspartate receptors, and elevated levels of QA have been associated with several disease states: Alzheimer’s disease, anxiety, depression, epilepsy, AIDS dementia, and Huntington’s disease. This research focuses on the activity of a unique trio of enzymes, the QA Trio, in the tryptophan kynurenine pathway: 3-hydroxyanthranilic acid 2,3-dioxygenase (HAD), α-amino-β-carboxymuconic-ε-semialdehyde decarboxylase (ACMSD), and α-amino-β-muconic-ε-semialdehyde dehydrogenase (AMSDH). This proposed metabolic trio converts 3-hydroxyanthranilic acid (3-HAA) to 2-aminomuconate (2-AM) by an oxidative ring cleavage followed by a non-oxidative decarboxylation and finishing with an NAD⁺ dependent oxidation of the terminal aldehyde to a carboxylic acid. Both intermediates of this trio, α-amino-β-carboxymuconic-ε-semialdehyde (ACMS) and α-amino-β-muconic-ε-semialdehyde (AMS), are unstable and spontaneously cyclize to form QA and picolinic acid (PA) respectively. The third enzyme of the trio was purified, and its crystal structure was solved and will be presented. A CE-MS method was developed to separate and quantitate the substrate and products of the
first two enzymes in this trio. Computer simulations in MATLAB were employed to analyze the data obtained by
MS, and preliminary works indicate a possible new feedback inhibition mechanism in the KP.

**An Ultra High Resolution Structure of CTX-M β-Lactamase to study catalysis and discover novel inhibitors**

Derek Nichols, Yu Chen University of South Florida College of Medicine: Dept of Molecular Medicine, 12901 Bruce
B.Downs Blvd , MDC07 Tampa, Fl 33612

The resistance to second and third-generation cephalosporins due to the production of CTX-M beta-lactamase
from bacteria continues to pose a serious threat to various regions of the world, particularly in nosocomial
settings. The hydrolysis reaction catalyzed by CTX-M proceeds through a pre-covalent complex, a high-energy
tetrahedral acylation intermediate, a low-energy acyl-enzyme complex, a high-energy tetrahedral deacylation
intermediate after attack via a catalytic water, and lastly, the hydrolyzed beta lactam ring product which is
released from the enzyme complex. Structural analysis of CTX-M-9's X-ray crystallographic structure at sub-
angstrom resolution has enabled us to study enzyme catalysis as well as perform computational molecular
docking. A major current debate is the identity of the catalytic base involved in deprotonating the nucleophilic
Ser70 which attacks the beta lactam ring. In a recent X-ray crystallographic analysis of CTX-M-9 at 0.88 Å,
several key hydrogen atoms as well as hydrogen bonding states of residues involved in catalysis were identified,
suggesting the catalytic water and Glu166 as the general base. Using this structure as a template, we screened
the ZINC small molecule library with DOCK and identified novel inhibitors against CTX-M-9. Further structure-
based design and synthesis has improved the affinity of the best inhibitor from 20uM to 89nM. Recently, we
obtained a 0.89 Å crystal structure of CTX-M beta-lactamase in complex with the recently developed 89nM non-
covalent inhibitor. With the structure currently under refinement, nearly all hydrogen atoms in the active site,
including those on the ligand, polar protein side chains and catalytic water, can be identified in the unbiased
difference electron density map.

**Understand the Catalytic Mechanism of 3-Hydroxyanthranilate-3,4-Dioxygenase by Visualizing the Intermediates**

Fange (Katherine) Liu, Jiafeng Geng, Kederlin Dornevil, and Aimin Liu, Departments of Chemistry and Center for
Diagnostics and Therapeutics, Georgia State University, P.O. Box 4098, Atlanta, GA.

3-Hydroxyanthranilate-3,4-dioxygenase (HAAO), a representative type III extradiol dioxygenase, catalyzes the
non-heme Fe(II)-dependent oxidative ring opening reaction of hydroxyanthranilic acid (3-HAA) with a $k_{\text{cat}}$ at
around 22 s$^{-1}$. HAAO is a critical component in the kynurenine pathway that degrades L-tryptophan in mammals
for biosynthesis of NAD$^+$ and glutaryl CoA. This enzyme also plays a central role in the 2-nitrobenzoic acid
degradation in bacteria. The crystal structures of HAAO from a variety sources have been determined. By
performing the in crystallo reactions, we trapped a series of catalytic cycle intermediates and determined their 3-
dimensional structures. These results lead to a proposed mechanistic model which is in large part in agreement
with the major chemical steps of the dioxygenase mechanism defined in type I extradiol dioxygenase,
homoprotocatechuate 2,3-dioxyg (HPCD). However, distinct features are also present including at least one
unprecedented intermediate. The oxygen insertion is unambiguously demonstrated to proceeds in a step-wise
oxygen insertion manner as opposite to being concerted. The unstable intermediate product α-amino-β-
carboxymuconate-ε-semialdehyde (ACMS) is trapped in both metal-bound and tunnel-bound forms. The
intermediates structurally characterized in this study allow us to present a snapshot view of the rapid and efficient
catalytic reaction in this representative type III extradiol dioxygenase.
### SER-CAT MEMBERS

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Florida State University, Tallahassee, FL 32306  March 15, 2013
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<th>Address</th>
<th>Title</th>
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<tr>
<td>Artikis, Efrosini</td>
<td><a href="mailto:ea09d@my.fsu.edu">ea09d@my.fsu.edu</a></td>
<td>(727) 331-1080</td>
<td>Tallahassee, FL 32304</td>
<td>Student</td>
</tr>
<tr>
<td>Askenasy, Isabel</td>
<td><a href="mailto:iaskenasy@fsu.edu">iaskenasy@fsu.edu</a></td>
<td>(850) 567-8788</td>
<td>Tallahassee, FL 32310</td>
<td>Student</td>
</tr>
<tr>
<td>Bienkiewicz, Ewa</td>
<td><a href="mailto:ewa.bienkiewicz@med.fsu.edu">ewa.bienkiewicz@med.fsu.edu</a></td>
<td>(850) 645-7326</td>
<td>Tallahassee, FL 32312</td>
<td>Faculty</td>
</tr>
<tr>
<td>Bingol, Kerem</td>
<td><a href="mailto:akb09@my.fsu.edu">akb09@my.fsu.edu</a></td>
<td>(850) 339-4248</td>
<td>Tallahassee, FL 32310</td>
<td>Student</td>
</tr>
<tr>
<td>Buchanan, Susan</td>
<td><a href="mailto:skbuchan@helix.nih.gov">skbuchan@helix.nih.gov</a></td>
<td>(301) 594-9222</td>
<td>Bethesda, MD 20892</td>
<td>Faculty</td>
</tr>
<tr>
<td>Chen, Yan</td>
<td><a href="mailto:ychen54@student.gsu.edu">ychen54@student.gsu.edu</a></td>
<td>(678) 447-4153</td>
<td>Atlanta, GA 30303</td>
<td>Student</td>
</tr>
<tr>
<td>Chen, Yu</td>
<td><a href="mailto:ychen1@health.usf.edu">ychen1@health.usf.edu</a></td>
<td>(813) 974-7809</td>
<td>Tampa, FL 33612</td>
<td>Faculty</td>
</tr>
<tr>
<td>Chen, Tong</td>
<td><a href="mailto:tc09@my.fsu.edu">tc09@my.fsu.edu</a></td>
<td>(850) 644-2048</td>
<td>Tallahassee, FL 32306</td>
<td>Student</td>
</tr>
<tr>
<td>Chrzas, John</td>
<td><a href="mailto:chrzas@anl.gov">chrzas@anl.gov</a></td>
<td>(630) 252-0648</td>
<td>Lemont, IL 60439</td>
<td>Faculty</td>
</tr>
<tr>
<td>Cocozaki, Alexis</td>
<td><a href="mailto:aic08@fsu.edu">aic08@fsu.edu</a></td>
<td>(850) 645-4909</td>
<td>Tallahassee, FL 32306</td>
<td>Faculty</td>
</tr>
<tr>
<td>Das, Nabanita</td>
<td><a href="mailto:ndas@magnet.fsu.edu">ndas@magnet.fsu.edu</a></td>
<td>(850) 459-1017</td>
<td>Tallahassee, FL 32310</td>
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<tr>
<td>Davies, Christopher</td>
<td><a href="mailto:davies@musc.edu">davies@musc.edu</a></td>
<td>(843) 792-1468</td>
<td>Charleston, SC 29425</td>
<td>Faculty</td>
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<tr>
<td>Davis, Ian</td>
<td><a href="mailto:ian.davis60@gmail.com">ian.davis60@gmail.com</a></td>
<td>(404) 413-5562</td>
<td>Cumming, GA 30028</td>
<td>Student</td>
</tr>
<tr>
<td>Delucas, Lawrence</td>
<td><a href="mailto:Duke2@uab.edu">Duke2@uab.edu</a></td>
<td>(205) 934-5329</td>
<td>Birmingham, AL 35294</td>
<td>Faculty</td>
</tr>
<tr>
<td>Dornevil, Kednerlin</td>
<td><a href="mailto:krikkrac@gmail.com">krikkrac@gmail.com</a></td>
<td>(770) 715-4003</td>
<td>Dacula, GA 30019</td>
<td>Student</td>
</tr>
<tr>
<td>Dunham, Christine</td>
<td><a href="mailto:christine.m.dunham@emory.edu">christine.m.dunham@emory.edu</a></td>
<td>(404) 712-1756</td>
<td>Atlanta, GA 30322</td>
<td>Faculty</td>
</tr>
<tr>
<td>Dyda, Fred</td>
<td><a href="mailto:Fred.Dyda@nih.gov">Fred.Dyda@nih.gov</a></td>
<td>(301) 402-4496</td>
<td>Bethesda, MD 20892</td>
<td>Faculty</td>
</tr>
<tr>
<td>Edison, Art</td>
<td><a href="mailto:aedison@ufl.edu">aedison@ufl.edu</a></td>
<td>(352) 392-4535</td>
<td>Gainesville, FL 32610</td>
<td>Faculty</td>
</tr>
<tr>
<td>Ellington, W.R</td>
<td><a href="mailto:wellington@fsu.edu">wellington@fsu.edu</a></td>
<td>(850) 645-6900</td>
<td>Tallahassee, FL 32306</td>
<td>Faculty</td>
</tr>
<tr>
<td>Esaki, Shingo</td>
<td><a href="mailto:shingoe@yahoo.com">shingoe@yahoo.com</a></td>
<td>(678) 613-7474</td>
<td>Atlanta, GA 30312</td>
<td>Student</td>
</tr>
<tr>
<td>Fajer, Piotr</td>
<td><a href="mailto:pfojer@fsu.edu">pfojer@fsu.edu</a></td>
<td>(850) 645-1337</td>
<td>Tallahassee, FL 32306</td>
<td>Faculty</td>
</tr>
<tr>
<td>Fu, Zheng-Qing</td>
<td><a href="mailto:fuzq@anl.gov">fuzq@anl.gov</a></td>
<td>(630) 252-0648</td>
<td>Lemont, GA 60439</td>
<td>Faculty</td>
</tr>
<tr>
<td>Geng, Jiafeng</td>
<td><a href="mailto:jeng1@student.gsu.edu">jeng1@student.gsu.edu</a></td>
<td>(678) 620-8488</td>
<td>Atlanta, GA 30329</td>
<td>Student</td>
</tr>
<tr>
<td>Gu, Yina</td>
<td><a href="mailto:ygu@chem.fsu.edu">ygu@chem.fsu.edu</a></td>
<td>(850) 570-5072</td>
<td>Tallahassee, FL 32310</td>
<td>Student</td>
</tr>
<tr>
<td>Hu, Zhongjun</td>
<td><a href="mailto:zh12b@my.fsu.edu">zh12b@my.fsu.edu</a></td>
<td>(850) 617-7816</td>
<td>Tallahassee, FL 32304</td>
<td>Student</td>
</tr>
<tr>
<td>Huo, Lu</td>
<td><a href="mailto:huo1@student.gsu.edu">huo1@student.gsu.edu</a></td>
<td>(860) 310-7506</td>
<td>Riverview, FL 33579</td>
<td>Student</td>
</tr>
<tr>
<td>Lewandowski, Eric</td>
<td><a href="mailto:elewandowski@health.usf.edu">elewandowski@health.usf.edu</a></td>
<td>(813) 310-7506</td>
<td>Riverview, FL 33579</td>
<td>Student</td>
</tr>
</tbody>
</table>
Li, Hong
hong.li@fsu.edu
(850) 644-6785
Tallahassee, FL 32306
Faculty

Liu, Aimin
feradical@gsu.edu
(404) 413-5532
Atlanta, GA 30302
Faculty

Liu, Fange
Fliu7@student.gsu.edu
(404) 432-3382
Tucker, GA 30084
Student

Longo, Liam
liam.longo@gmail.com
(772) 361-9697
Tallahassee, FL 32304
Student

Marshall, Alan
marshall@magnet.fsu.edu
(850) 644-0529
Tallahassee, FL 32310
Faculty

Morris, Kathy
kmorris@BCL4.bmb.uga.edu
(706) 542-3384
Athens, GA 30602
Faculty

Nichols, Derek
d nichol1@health.usf.edu
(813) 777-4532
Tampa, FL 33609
Student

Noble, Alex
Ajin10d@fsu.edu
(850) 270-7088
Tallahassee, FL 32310
Student

Pemberton, Orville
opember1@health.usf.edu
(813) 988-6990
Tampa, FL 33617
Student

Peng, Yu
ypeng@fsu.edu
(850) 645-4909
Tallahassee, FL 32310
Post-Doc

Polson, Darcie
darcie.miller@stjude.org
(901) 595-6277
Memphis, TN 38112
Faculty

Ramia, Nancy
nfr07@my.fsu.edu
(850) 339-3285
Tallahassee, FL 32310
Student

Rogers, David
david.rogers@uky.edu
(850) 257-5205
Lexington, KY 40536
Student

Rose, John P.
rose@BCL4.bmb.uga.edu
(706) 542-1750
Athens, GA 30602
Faculty

Rydel, Timothy
timothy.j.rydel@monsanto.com
(636) 737-5642
Chesterfield, MO 63017
Faculty

Schellenberg, Matthew
matthew.schellenberg@nih.gov
(919) 541-5759
Research Triangle Park, N, NC 27709
Faculty

Sen, Ilker
isen@fsu.edu
(850) 645-1335
Tallahassee, FL 32303
Post-Doc

Shao, Yaming
yshao@fsu.edu
(850) 645-4909
Tallahassee, AL 32306
Post-Doc

Smith, Emmanuel
esmith3@health.usf.edu
(813) 396-9188
Tampa, FL 33602
Student

Spear, John
jms092@fsu.edu
(845) 656-1665
Tallahassee, FL 32306
Faculty

Spilman, Michael
mspilman@fsu.edu
(901) 218-1124
Tallahassee, FL 32308
Post-Doc

Stagg, Scott
staggq@asu.edu
(850) 645-7872
Tallahassee, FL 32306
Faculty

Strowpe, Beth
mestroupe@bio.fsu.edu
(850) 645-9318
Tallahassee, FL 32306
Faculty

Tang, Li
ltang@fsu.edu
(850) 645-4909
Tallahassee, AL 32306
Student

Tenorio, Connie
cat08d@my.fsu.edu
(954) 655-5151
Tallahassee, FL 32304
Student

Terrell, James
tterrell18@student.gsu.edu
(404) 433-8332
Marietta, GA 30066
Student

Tsui, Tsz Kin Martin
tt12c@my.fsu.edu
(218) 666-8784
Tallahassee, FL 32306
Student

Twigg, Pam
ptwigg@fsu.edu
(256) 656-4049
Tallahassee, FL 32308
Post-Doc
Tenth Annual SER-CAT Meeting

Wang, Bi-Cheng
wang@BCL1.bmb.uga.edu
(706) 541-1747
Athens, GA 30602
Faculty

Wei, Ling
lw11e@my.fsu.edu
(850) 559-6756
Tallahassee, FL 32310
Student

White, Stephen
stephen.white@stjude.org
(901) 595-3040
Memphis, TN 38205
Faculty

Wright, Anna
ask09d@my.fsu.edu
(253) 222-8564
Tallahassee, FL 32301
Student

Yu, Ge
gy11@my.fsu.edu
(850) 570-5760
Tallahassee, FL 32304
Student

Zhang, Yujie
yujie.zhang@med.fsu.edu
(850) 459-0128
Tallahassee, FL 32306
Student

Sponsors
FSU Office of the VP for, Research
opope@fsu.edu

(850) 644-8664
Tallahassee, FL 32306
Faculty

Bruker
jennifer.schulz@bruker-axs.com
(608) 276-3087
Madison, WI 53711
Faculty

GenScript USA Inc.,
nicole.niu@genscript.com
(732) 902-1603
Piscataway, NJ 8854
Faculty

The University of Georgia,
kmorris@BCL4.bmb.uga.edu
(706) 542-3384
Athens, GA 30602
Faculty

NOTES