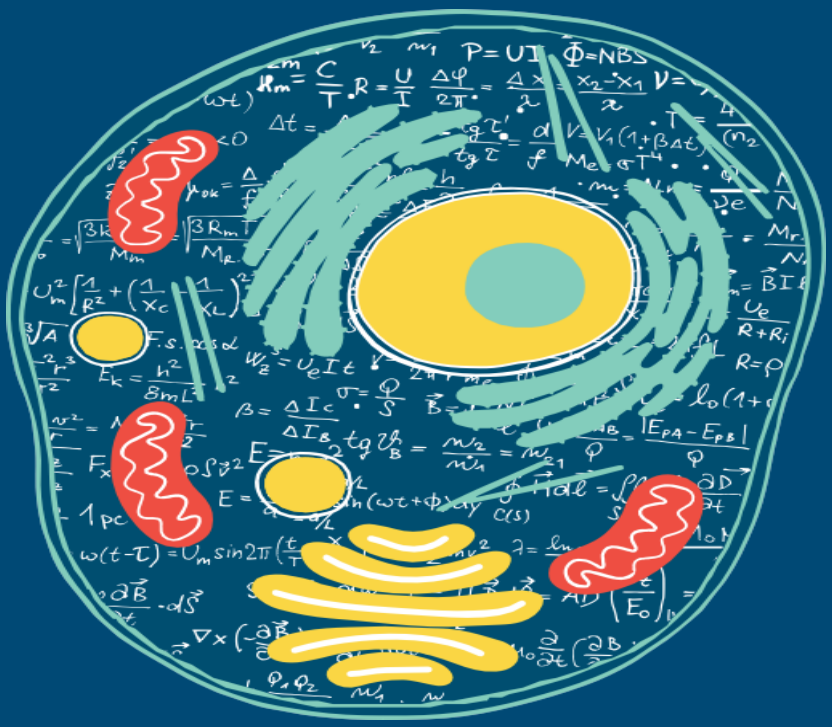


Finding Your Inner Modeler-3

University Of Alabama at Birmingham

June 13 - 14, 2019

A collaborative workshop for quantitative Cell/Molecular Biologists and Computational Modelers to learn how to work together

Funded by NSF MCB award #1901738

WELCOME

Welcome to the 2019 Finding Your Inner Modeler III Meeting. We are excited that you are here and we hope the meeting offers fresh and exciting ideas; which will aid and advance your research.

Elizabeth Sztul, Ph.D.
Professor
Department of Cell, Developmental and Integrative Biology
University of Alabama at Birmingham
esztul@uab.edu

Ryoichi Kawai, Ph.D.
Associate Professor
Department of Physics
University of Alabama at Birmingham
kawai@uab.edu

Carlos Lopez, Ph.D.
Assistant Professor
Biochemistry & Biomedical Informatics
Vanderbilt University
c.lopez@vanderbilt.edu

David Stone, Ph.D.
Professor
Department of Biological Sciences
University of Illinois at Chicago
dstone@uic.edu

AGENDA

Thursday, June 13, 2019

8:30 - 9:00 AM	**Registration** **Bevill Biomedical Research Building (BBRB) 170**
9:00 - 9:10 AM	**Welcome and Meeting Overview** **David Stone** University of Illinois at Chicago **Elizabeth Sztul** University of Alabama at Birmingham
9:50 - 9:10 AM	Project Presentation 1 Daniel Szymanski and Joe Turner Purdue University ***"Mechanical Modeling of Epidermal Morphogenesis with Integrated Experimental Verification"***
9:50 - 10:30 AM	Project Presentation 2 Gregor Neuert Vanderbilt University ***"Predictive Understanding of Cell Biological Systems Through Kinetic Analysis"***
10:30 - 11:10 AM	Project Presentation 3 Matthias Falk and Wonpil Im Lehigh University **"Modeling Gap Junctions and Direct Cell-to-Cell Communication: A Few Examples"**
11:10 - 11:30 AM	**Coffee Break**
11:30 - 12:30 PM	Keynote Talk 1 Julien Berro Yale University **"Essentially All Models Are Wrong, But Some Are Useful"**

AGENDA

12:30 - 1:10 PM	NSF MCB Program Officer Rita Miller National Science Foundation ***"Funding Opportunities at NSF-MCB for Biological Modeling"***
1:10 - 2:30 PM	**CATERED LUNCH**
2:30 - 3:10 PM	Community Modeling Anne Cowan University of Connecticut ***"VCell and the Potential of Community Modeling"***
3:10 - 4:00 PM	Project Presentation 4 Shahid Mukhtar University of Alabama at Birmingham ***"Getting to the Edge of Diverse Biological Networks"***
4:00 - 5:30 PM	Poster Session BBRB Lobby
5:30 PM	Happy Hour Mixer BBRB Lobby

AGENDA

Friday, June 14, 2019

9:00 - 9:40 AM	Project Presentation 5 Belinda Akba and Marcela Rojas-Pierce North Carolina State University ***"Qualitative-to-Quantitative Modeling Directs Experimental Inquiry: Uncovering Mechanisms of Membrane Fusion in Plants"***
9:40 - 10:20 AM	Project Presentation 6 Wouter-Jan Rappel University of California San Diego ***"Computational Modeling of Eukaryotic Cell Migration Inspired by Experiments"***
10:20 - 11:00 AM	Project Presentation 7 Lynn Dobrunz University of Alabama at Birmingham ***"Use of Mathematical Modeling to Investigate Mechanisms Regulating Neurotransmitter Release"***
11:00 - 11:20 AM	**Coffee Break**
11:20 - 12:20 PM	Keynote Talk 2 Wonpil Im Lehigh University ***"Biomolecular Modeling and Simulation Using CHARMM-GUI"***

AGENDA

12:20 - 12:50 PM	Match Website David Stone University of Illinois at Chicago Michael Blinov University of Connecticut Health **"Matching Computational and Modeling Expertise among Biologists"**
12:50 - 2:00 PM	**CATERED LUNCH**
2:00 - 2:40 PM	Tools for Modeling Carlos Lopez Vanderbilt University ***"Modeling with Programs Using PySB in Python"***
2:40 - 3:40 PM	Panel Discussion/Town Hall Szymansky, Neuert, Im, Lopez, Rappel, Berro, Blinov, Mukhtar, Stone, Turner, Cowan, Akpa
3:40 - 5:40 PM	*"Meet a Modeler" 30 Minute Consultation Sessions:* Szymansky, Neuert, Im, Lopez, Rappel, Berro, Blinov, Mukhtar (Assigned locations)
5:40 PM	*Closing Remarks* David Stone and Elizabeth Sztul Depart for airport or a vacation in the "Heart of Dixie"

ABSTRACTS

#1 Hong Qin
"Insights on longevity from gene networks"
University of Tennessee at Chattanooga
Department of Computer Science and Engineering; Department of Biology, Geology, and Environmental Sciences, and SimCenter

Biological aging is a complex phenotype with many genes involved, and is characterized by an exponential increase of mortality rate. Dietary restriction is a lifespan extension method that is conserved amon many species. We have developed a probabilistic gene network model for cellular aging that can capture the emergent aspect of cellular aging. We applied our network model to study the lifespan extension effect of calorie restriction, including lifespan data sets measured in yeast strains with deletion of SIR2 and TOR1, and in different glucose concentrations. Our results suggest that gene network robustness plays a major in the effect of dietary restriction. Our results show that network model for aging can offer new insights on molecular mechanism of cellular aging.

#2 Gregory Reeves
"Dorsal/NF-κB exhibits a dorsal-to-ventral mobility gradient in the Drosophila embryo"
Hadel Al Asafena,[1], Natalie M. Clarkb,[1], Thomas Jacobsena, Rosangela Sozzanib, and Gregory T. Reevesa,[2]
a. Department of Chemical and Biomolecular Engineering, North Carolina State University, Raleigh, NC
b. Department of Plant and Microbial Biology, North Carolina State University, Raleigh, NC

Morphogen-mediated patterning is a highly dynamic developmental process. To obtain an accurate understanding of morphogen gradients, systems-biology-type models must be constructed, which in turn implies that biophysical parameters such as protein diffusivities must be quantified in vivo. The dorsal-ventral (DV) patterning of early Drosophila embryos by the NF-κB homolog Dorsal (Dl) is an excellent system for understanding morphogen gradient formation. Dl gradient formation is controlled by the inhibitor Cactus/IκB (Cact), which regulates the nuclear import and diffusion of Dl protein. However, quantitative measurements of spatiotemporal Dl movement are currently lacking. Here, we use scanning fluorescence correlation spectroscopy to quantify the mobility of Dl. We find that the diffusivity of Dl varies along the DV axis, with lowest diffusivities on the ventral side, and the DV asymmetry in diffusivity is exclusive to the nuclei. Moreover, we also observe that nuclear export rates are lower in the ventral and lateral regions of the embryo. Both cross correlation spectroscopy measurements and a computational model of Dl/DNA binding suggest that DNA binding of Dl, which is more prevalent on the ventral side of the embryo, is correlated to a lower diffusivity and nuclear export rate. We propose that the variation in Dl/DNA binding along the DV axis is dependent on Cact binding Dl, which prevents Dl from binding DNA in dorsal and lateral regions of the embryo. Thus, our results highlight the complexity of morphogen gradient dynamics and the need for quantitative measurements of biophysical interactions in such systems.

ABSTRACTS

#3 Liqun Zhang

"Molecular Dynamic Simulations on Human Beta Defensin Type 3 Embedded in Different Lipid Bilayers "

Rabeta Yeasmin, Mouhmad Elayya, George Rucker, Yilun Lee, Liqun Zhang
Chemical Engineering Department, Tennessee Technological University, Cookeville, TN, 3850

Human β defensin type 3 (hBD-3) belongs to the human innate immune system. It is a natural antibacterial peptide mainly secreted from the human epithelial cells, and has a charge density of +11. It can interact with bacterial cell membranes and translocate through the cell membrane. In order to understand the interaction and dynamics of hBD-3 with different lipid membranes during the translocation process, the hBD-3 in both monomer and dimer forms embedded inside different lipid bilayers were investigated using CHARMM and NAMD molecular dynamics simulations. The Gram- positive bacteria membrane was represented by POPG to POPE lipids with a molar ratio of 3:1; and Gram-negative bacteria membrane by POPG to POPE and POPG to POPC at a molar ratio of 1:3. Besides that, POPC mixed with 10% of PIP2 lipid bilayer was also investigated. In order to consider the concentration of salt to the structure and dynamics of hBD-3 and water dynamics, both systems having NaCl solvent concentration of 0.15 M and 0.3 M were worked on. It was found that water molecules can cross the lipid bilayers through hBD-3, with the flow rate dependent on the lipid chemical composition and oligomerization form of hBD-3. Reorientation of negatively charged lipid head toward hBD-3 was observed in all the systems. Aggregation of negatively charged lipids around hBD-3 was observed based on micro-second long all-atom simulations, although such kind of lipid segregation was not observed in lipid only systems. The result can help to understand the antibacterial functional mechanism of hBD-3.

#4 Nick Panchy

"Circadian regulation tempers ribosomal phosphorylation in response to changing day-night cycles"

Nicholas Panchy1, Tian Hong[1,2]
1. National Institute for Mathematical Biological Synthesis, University of Tennessee Knoxville, Knoxville;
2. Biochemistry & Cellular and Molecular Biology, University of Tennessee Knoxville, Knoxville

Time keeping at the molecular level occurs through the integration of both genetic and environmental signals, particularly the circadian clock and light sensing molecules. These interlinked systems regulate gene expression at multiple levels, including both transcription and translation. The ribosomal protein RPS6 is known to be cyclically phosphorylated in a light and clock dependent manner, but how RPS6 cycling is regulated and the function of cycling remain unexplored. To study RPS6 cycling, we have developed a model of RPS6 phosphorylation using a system of ordinary differential equations that incorporates both the circadian clock and light sensing. Our model suggests that phosphorylation of RPS6 is simulated by light while the circadian clock regulates dephosphorylation both during and in anticipation of periods of light exposure. Using this approach, we can not only correctly predict the phosphorylation of RPS6 under wild type, clock deficient, and light deficient conditions, but also infer the state of RPS6 phosphorylation under novel conditions including changes in the phase or period of light exposure. Our results suggest that, while light sensing is the primary driver of RPS6 phosphorylation, regulation by the circadian lock allows the system to anticipate and the light cycle adept to variations in it.

ABSTRACTS

#5 Tomasz Nawara
"STAR – A new approach for measuring the dynamics of clathrin mediated endocytosis"
Tomasz Nawara[1], Tejeshwar Rao1, Alexa Mattheyses[1]
1 Department of Cell, Developmental and Integrative Biology,
University of Alabama at Birmingham, Birmingham, AL

Clathrin mediated endocytosis is a dynamic process whereby vesicles are created at the plasma membrane to internalize a wide range of cargos. The formation of clathrin coated vesicles is driven by changes in membrane architecture and composition. Many of the molecular components involved in clathrin mediated endocytosis are known, and their dynamics relative to one another are defined. Similarly, the morphological stages of vesicle formation have been defined by static electron microscopy. However, the connection between the fluorescence based measurements of protein dynamics and the fixed cell electron microscopy data elucidating membrane shape remains unclear. Because methods that report membrane shape rely on fixed samples and protein dynamics on live fluorescence microscopy, we needed a new technique to unify protein dynamics with membrane shape throughout this highly dynamic process. Here we present Simultaneous Two- wavelength Axial Ratiometry (STAR), as an advanced microscopy technique for resolving the correlation between protein and membrane shape dynamics during vesicle formation, maturation and scission in real-time. We address two models of clathrin association at the plasma membrane during endocytosis: clathrin accumulating simultaneously with membrane deformation or accumulating as a flat lattice that then deforms to a pit. To do so, we imaged clathrin tagged with the STAR probe along with dynamin-2 transfected into Cos-7 cells. Whereas clathrin is a marker for vesicle formation, dynamin-2 is required for scission and allows us to discern completed endocytic events from abortive pits. We show that the membrane deforms as clathrin is accumulating, and pits do not appear out of flat clathrin lattices. We further distinguished three groups of endocytic events, suggesting there is not one unified pathway. Herein we present a powerful approach appropriate for the integration of protein dynamics with membrane shape changes during highly dynamic endocytic events in living cells with nanometer axial resolution. With future examination of other endocytic proteins, we hope to unify the physical process of vesicle formation in clathrin mediated endocytosis with its dynamics

#6 Tejeshwar Rao
"EGFR activation attenuates the mechanical threshold for integrin tension and focal adhesion formation"
Tejeshwar C. Rao[1], Victor Pui-Yan Ma[2], Aaron Blanchard[3], Tara M. Urner[1], Shreya Grandhi[1], Khalid Salaita[2,3], and Alexa L. Mattheyses[1]*
1. Department of Cell, Developmental, and Integrative Biology, University of Alabama at Birmingham, Birmingham AL
2. Department of Chemistry, Emory University, Atlanta, Georgia
3. Wallace H. Coulter Department of Biomedical Engineering, Georgia Institute of Technology and Emory University, Atlanta, Georgia

Mechanical forces, growth factors, and the extracellular matrix, all play critical roles in cell adhesion. To understand how epidermal growth factor receptor (EGFR) impacts the mechanics of adhesion we used tension gauge tether (TGT) probes displaying cRGDfK, an integrin ligand, and quantified integrin tension during cell adhesion. EGF exposure increased spread area, cell circularity, integrated integrin tension, tension occupancy, radial organization and size of focal adhesions (FAs) significantly in Cos7 cells on TGT surfaces. These findings suggest EGFR acts as a mechano-organizer, regulating integrin tension and FA spatial organization. Additionally, the mechanical force threshold for outside-in integrin activation is tunable by EGFR which promotes cell adhesion and spreading on lesser compliant substrates. Parallel genetic and pharmacologic strategies demonstrated that these phenotypes are driven by ligand-dependent EGFR signaling. Our results establish a novel mechanism where EGFR allosterically regulates integrin activation and cell adhesion, providing control over cellular responses to the environment.

ABSTRACTS

#7 Bharat Mishra
"Dynamic transcriptional event modeling in Arabidopsis leaf senescence"
Bharat Mishra, Yali Sun, TC Howton, Nilesh Kumar & M. Shahid Mukhtar
Department of Biology, University of Alabama at Birmingham

Age-dependent senescence is a multifaceted and highly coordinated developmental phase in the life of plants that is manifested with genetic, biochemical and phenotypic continuum. Thus, elucidating the dynamic network modeling and simulation of molecular events, in particular gene regulatory network during the onset of senescence is essential. Here, we constructed a computational pipeline that integrates senescence-related co-expression networks with transcription factor (TF)-promoter relationships and microRNA (miR)-target interactions. Network structural and functional analyses revealed important nodes within each module of these co-expression networks. Subsequently, we inferred significant dynamic transcriptional regulatory models in leaf senescence using time-course gene expression datasets. Dynamic simulations and predictive network perturbation analyses followed by experimental dataset illustrated the kinetic relationships among TFs and their downstream targets. In conclusion, our network science framework discovers cohorts of TFs and their paths with previously unrecognized roles in leaf senescence and provides a comprehensive landscape of dynamic transcriptional circuitry.

#8 Amanda Johnson
"Pathway stimulation threshold rates regulate kinase activation"
Amanda N. Johnson[1], Guoliang Li[1], Hossein Jashnsaz[1], Alexander Thiemicke[1], Benjamin K. Kesler[1], Dustin C. Rogers[1] and Gregor Neuert[1]
1. Department of Molecular Physiology and Biophysics, School of Medicine, Vanderbilt University, Nashville, TN

All living cells experience changes in their external environment. Signaling networks detect, process, and regulate cell behavior in response to these changes. Although it is well established that stimulus concentration impacts cell response strength, no one has systematically investigated how stimulation rates affect signaling in any eukaryotic system. To address this limitation, we examined budding yeast osmosensing mitogen activated protein kinase (MAPK) Hog1 activation patterns during hyperosmotic stress treatments delivered at varying rates in single cells in real time. We discovered that rates below a threshold condition failed to induce Hog1 activation, correlating with dramatically reduced resistance to a second acute stress treatment. We further found that the protein tyrosine phosphatase Ptp2 serves as the major threshold rate condition regulator, and its expression levels can modulate the osmotic stress stimulation rate required for Hog1 activation. We have thus uncovered two new signaling principles: (1) a threshold rate condition regulating signaling activation and cell phenotype and (2) phosphatase regulation of the threshold rate condition. These rate-dependent mechanisms of signaling control, revealed here only through kinetic stimulation, may be generalizable to other rate sensitive signaling systems.

ABSTRACTS

#9 Jordan Fauser
"Allosteric Regulation of Engineered Shp2 Phosphatase"
Jordan Fauser, Vincent Huyot, Jennifer Klomp, Andrei Karginov
Department of Pharmacology, University of Illinois- Chicago

Shp2 is a ubiquitously expressed protein tyrosine phosphatase. Aberrant Shp2 signaling is associated with several pathologies; however, the exact role of Shp2 in various signaling pathways is less well defined. Current tools to study Shp2, such as expression of constitutively active or dominant negative Shp2 or small molecule inhibitors, evaluate the effect of long-term activation or depletion of Shp2. However, these studies often come to conflicting conclusions. Here we report the development of two alternative approaches that overcome existing limitations and provide highly specific temporal and spatial regulation of Shp2 signaling. The insertion of a domain, with regulatable dynamics through induced dimerization, into the catalytic domain of Shp2 enables specific control of Shp2 activity. This regulation occurs through allosteric disruption of the catalytic domain in the un-dimerized "off" state which is reversed in the dimerized "on" state. We have engineered allosterically regulated Shp2 proteins through the insertion of a Light Regulated (LightR) or Rapamycin Regulated (RapR) domain. Our RapR-Shp2 has iFKBP inserted into the catalytic domain of Shp2, disrupting the catalytic activity. Upon addition of rapamycin and FRB the iFKBP insert is stabilized and Shp2 catalytic activity is restored. Similarly, LightR-Shp2 has two VVD proteins inserted in the catalytic domain. Upon stimulation with blue light, VVD dimerizes and Shp2 catalytic activity is restored. These tools provide temporal control of Shp2 activity allowing the elucidation of its role in various pathways. LightR-Shp2 has the added advantage of deactivation; allowing for the investigation of transient Shp2 signaling and identification of secondary signaling events. We have shown that LightR and RapR-Shp2 activate the MAPK pathway leading to ERK activation and dephosphorylate EGFR, PLCγ, and FAK in living cells. To further understand the effect of the insertion on catalytic activity, we performed molecular dynamics simulations to probe the conformational dynamics of LightR-Shp2 in the dark and light state. Furthermore, we evaluated the effect of varied length of the amino acid sequence linking the light regulated domain to the catalytic domain of Shp2. This analysis revealed a destabilization of the substrate binding domain and disruption of the WPD loop in the dark state. We also observed, in silico, continued disruption of the catalytic domain in the light state of the construct containing a sub-optimal linker length. Our data demonstrate that allosterically regulated tools can be used to probe physiologically relevant pathways to elucidate the various roles of Shp2 phosphatase as well as providing insight into the design of other regulated enzymes.

ABSTRACTS

#10 Nilesh Kumar

"Systems Biology and Machine Learning in Plant–Pathogen Interactions"

Nilesh Kumar[1], Bharat Mishra[1] & M. Shahid Mukhtar[1,2,3]*

1. Department of Biology, University of Alabama at Birmingham, 1300 University Blvd., Birmingham, AL
2. Nutrition Obesity Research Center, University of Alabama at Birmingham, University Blvd., Birmingham, AL
3. Department of Surgery, UAB School of Medicine, Birmingham, AL

Systems biology is an inclusive approach to study the static and dynamic emergent properties on a global scale by integrating multi-omics datasets to establish qualitative and quantitative associations among multiple biological components. With an abundance of improved high throughput -omics datasets, network-based analyses and machine learning technologies are playing a pivotal role in comprehensive understanding of biological systems. Network topological features reveal most important nodes within a network as well as prioritize significant molecular components for diverse biological networks, including co-expression, protein-protein interaction, and gene regulatory networks. Machine learning techniques provide enormous predictive power through specific feature extraction from biological data. Deep learning, a subtype of machine learning, has plausible future applications because a domain expert for feature extraction is not needed in this algorithm. Inspired by diverse domains of biology, we here review classic systems biology techniques applied in plant immunity thus far. We also discuss additional advanced approaches in both graph theory and machine learning, which may provide new insights for understanding plant-microbe interactions. Finally, we propose a hybrid approach in plant immune systems that harnesses the power of both network biology and machine learning, with a potential to be applicable to both model systems and agronomically important crop plants.

#11 Jack Linehan

"How to Beat the Heat: An Analysis of Homologous Proteins in Mesophile and Thermophile Prokaryotes"

Jack Linehan, Department of Physics, Department of Biology, DePaul University
Jesus Pando, Department of Physics, DePaul University

This study looked to identify features related to thermal stability and function in the amino acid chains of short globular proteins from mesophile and thermophile species, within the constraint that the protein fold to perform a specific function. To do so 540 homologous pairs of proteins were studied. The amino acid chains were converted to hydrophobicity signals by assigning a hydropathy score to each residue in the polypeptide. The hydrophobicity signals were passed through a wavelet packet transform and the resulting spectra analyzed. Bootstrapping was used to generate a control data set to determine if the true ordering of amino acids codes for a non-random fluctuation in hydropathy along the length of the polypeptide. A method to relate the spectral characteristics to the function of a protein making use of gene ontologies was developed as a proof of concept. As a group, mesophile and thermophile proteins have very similar total power. However, on a protein-to-protein basis the thermophile contains a greater total power in 489 of the 540 pairs (90.56%). The statistical measures skew and kurtosis were adapted so that a spectrum of skew and kurtosis values were generated for each protein. These values indicate that the fluctuation in hydropathy is non random and position dependent. Thermophile proteins have larger power at frequency bands 21 through 31 (average intervals of 100 to 77 amino acids), and 44 to 56 (on average 46 to 19 amino acids), which may contribute to their having greater total power in 90.56% of the pairs. Increases to the fluctuation in hydropathy within certain lengths throughout the total amino acid chain of a protein may be a means of raising the temperature at which a protein denatures.

ABSTRACTS

#12 Joseph Cleland
"Combining kinetic pathway stimulation with genetics to identify novel signaling mechanisms"
Joseph A. Cleland[1], Amanda N. Johnson[1] and Gregor Neuert[1]
1. Department of Molecular Physiology and Biophysics, School of Medicine, Vanderbilt University, Nashville, TN

To preserve health and survival, cells must accurately sense and respond to a variety of environmental stimuli. Currently, most research conducted to analyze cell signaling use instantaneous stimuli to perturb cells and mimic environmental change. However, this instantaneous environmental change may not be physiologically relevant, because it saturates the system masking subtle but important signaling mechanisms/dynamics. Therefore, we aim to determine how diverse patterns of stimuli that vary in regard to rate of change, intensity, duration, and time between sequential stimulations affect overall cell survival. To study how environmental change impacts cellular phenotypes, we use the budding yeast model organism Saccharomyces cerevisiae High Osmolarity Glycerol (HOG) pathway to discover novel signaling and gene expression mechanisms. Using budding yeast provides benefits such as short generation time, small cell size, and easily controllable genetics. In addition, the conserved Mitogen Activated Protein Kinase (MAPK) HOG pathway provides a simple and less redundant pathway to study. Furthermore, this pathway has easily quantifiable outputs such as cell volume change, osmosensing, and Hog1 nuclear localization. Using this model system, we have found that following instantaneous or step treatment of NaCl, the WT strain and ptp2Δ mutant respond equally; however, under varying rate treatments, their signaling profiles are significantly different. To further investigate how the HOG pathway regulates a response, we plan to study various tyrosine and threonine phosphatase deletion strains and examining their response to dynamic environmental change. Understanding the molecular function of phosphatases and kinases will provide insight into how signaling pathways in humans are affected by homologous proteins which are known to be associated with ailments such a cancer, neurological diseases, and autoimmune diseases.

#13 Bradly Alicea
"A New Kind of Developmental Biology"
OpenWorm Foundation and Orthogonal Research and Education Laboratory

Over the past decade, there has been an explosion of quantitative, high-throughput data generated by a host of new measurement technologies. Public access to these data provides information on a host of phenomena from behavioral to genomic, which allows for biological processes at multiple scales to be more fully characterized. Concurrently, new forms of collaboration and education have emerged which allow these data to be utilized by a broader community of scientists from many different fields and career stages. This can not only lead to new opportunities for scientific discovery, but also result in new forms of scientific practice: open-source computational biology, theory hackathons, the creation of open data repositories, and a wide range of published output. It is this combination of data recycling and collaborative flexibility which defines this new type of "open" model for scientific discovery. To demonstrate the I will draw from work being conducted in the DevoWorm group (http://devoworm.weebly.com) to demonstrate how this new kind of developmental neuroscience can proceed. By integrating multiple sources of data into a framework of computational modeling and analysis, we can demonstrate global processes in the developing nervous system as well as undiscovered phenomena related to developmental/newly-differentiated, emerging neural systems, and the nature of spatiotemporal complexity at multiple scales. Understanding the nervous system and developmental processes by using approaches such as networks/connectomics, the analysis of biological dynamics, and comparisons among a diversity of model organisms contributes to a richer view of developmental. Bringing together diverse sources of biological data also leads us to new insights into biological emergence and the complexity of cellular systems. We will conclude with considering how this work provides innovations in model-building, visualization, and perhaps even new biological laws.

ABSTRACTS

#14 Hossein Jashnsaz

"Combining theory with experiments to identify predictive models of signal transduction"

Hossein Jashnsaz[1], Zachary Fox[2], Jason J. Hughes[1], Guoliang Li[1], Brian Munsky[2], and Gregor Neuert[1]
1. Department of Molecular Physiology and Biophysics, School of Medicine, Vanderbilt University, Nashville, TN
2. Department of Chemical and Biological Engineering, Colorado State University, Fort Collins, CO

Predicting cell signaling response upon environmental changes over time or upon genetic mutations is currently an unsolved problem. The reason for this predicament is the fact that the mechanisms of how cells sense, process, and respond to extracellular stress are not fully understood. Here we present a combined theoretical and experimental framework to quantitatively compare predictive models of signal transduction networks. To demonstrate the feasibility of this approach, we used the High Osmolarity Glycerol (HOG) Stress Activated Protein Kinase (SAPK) signaling pathway in the yeast Saccharomyces cerevisiae model organism. We expose cells to different osmotic steps, pulses, linear or non-linear gradients to differentially regulate Hog1. Under these conditions, we quantify in single cells Hog1 nuclear localization as a measure of signal transduction using time lapse microscopy. We observe for these distinctive osmolyte stimulus profiles differentially modulate Hog1 intensity, duration and rate of activation. To qualitatively understand these data sets we established a network inference framework using simulated data as the ground truth. Our general model inference framework is based on an n-node circuit motif representing the Hog1 signal transduction pathway. In this framework, model complexity is changed by altering the number of the nodes, types and numbers of interactions. Several models are compared by their ability to make new predictions. We demonstrate how model predictions are impacted by altering the amount, type and order of data. By combining this approach with information theory criteria, we supplement intuition with rationally designed experiments to select biologically meaningful models. Our ultimate goal is to use this combined experimental and theoretical framework to develop data driven models that are maximally predictive of signaling responses. This approach is flexible and applicable to many different pathways, in different organisms and for a range of environmental conditions. In the future, such an approach maybe applicable to predict signaling responses upon genetic mutations, predict signaling response upon altered environmental exposure or predict signal responses upon drug treatments.

#15 Hao-Bo Guo

"A Suggestion of Converting Protein Intrinsic Disorder to Structural Entropy using Shannon's Information Theory"

Hao-Bo Guo[1],*, Yue Ma[2], Gerald A. Taskan[3], Hong Qin[1,4], Xiaohan Yang[2,3] and Hong Guo[2]
1. Department of Computer Science and Engineering, SimCenter, University of Tennessee, Chattanooga, TN
2. Department of Biochemistry and Cellular and Molecular Biology, University of Tennessee, Knoxville, TN
3. Biosciences Division, Oak Ridge National Laboratory, Oak Ridge, TN
4. Department of Biology, Geology, and Environmental Science, University of Tennessee Chattanooga, TN
*Correspondence: HBG (haobo-guo03@utc.edu)

We propose a framework to convert the protein intrinsic disorder content to structural entropy (H) using Shannon's information theory (IT). The structural capacity (C), which is the sum of H and structural information (I), equals to the amino acid sequence length of the protein. The structural entropy of the residues expands a continuous spectrum ranging from 0 (fully ordered) to 1 (fully disordered), consistent with Shannon's IT that scores the fully-determined state 0 and the fully-uncertain state 1. The IDP's in a living cell may participate in maintaining the high-energy-low-entropy state. In addition, under this framework, the biological functions performed by proteins and associated with the order or disorder of their 3D structures could be explained in views of information-gains or entropy-losses, or the reverse processes.

ABSTRACTS

#16 Rohit Venkat
"Understanding the regulation of gene transcription programs by kinetic signaling concentration gradients"
Rohit Venkat and Gregor Neuert
Department of Molecular Physiology & Biophysics, School of Medicine, Vanderbilt University, Nashville, TN

A fundamental question in biology is how a specific signaling protein precisely coordinates the transcription of hundreds of genes in a kinetic and dose-dependent manner. While many studies have shown that transcription factor affinity can set thresholds of transcriptional activation, it is currently unknown how kinetic signaling concentration gradients establish gene-specific thresholds of transcriptional activation within a single cell. Importantly, the dependence of transcriptional activation upon kinetic signaling concentration gradients may contribute to the regulation of gene transcription programs by generating dose-dependent transcriptional responses, ensuring gene activation specificity, and promoting the dynamic coordination of gene expression. To address this question, we will investigate signaling thresholds of transcriptional activation in response to osmotic stress in the budding yeast Saccharomyces cerevisiae. In preliminary experiments, we find that while a sudden increase in osmolyte concentration results in robust Hog1 activation and synchronous induction of a set of co-regulated genes, a linear increase in Hog1 activation over time, brought about by a quadratic osmolyte concentration gradient, enables the time-resolved separation of transcriptional activation thresholds for individual genes. To determine whether these gene-specific signaling thresholds are a genome-wide feature of transcriptional regulation, we will employ highly sensitive genome-wide sequencing methodologies to temporally resolve the transcriptional activation thresholds for individual genes in response to different Hog1 signaling dynamics. Because transcriptional activation depends on the coordinated action of chromatin remodelers to alter the local chromatin environment and stimulate transcription, mutant strains of specific chromatin regulators will be used to assess their impact on setting these thresholds. For pairs of identified gene loci with matching transcriptional activation thresholds, we will further interrogate the co-regulation of single-cell transcriptional activation thresholds using single-molecule RNA fluorescence in situ hybridization (smRNA FISH) microscopy. Combined with single-cell experiments in conditional mutant strains, this approach will provide the ability to distinguish between different mechanisms of transcriptional regulation that could not otherwise be achieved by population-level studies. By illuminating how critical signaling thresholds are set in individual cells and the impact of chromatin regulators in setting these thresholds, our work will lay the foundation for improved mechanistic understanding of the role of kinetic signaling concentration gradients and chromatin remodeling in regulating gene transcription programs in a threshold-dependent manner.

#17 Benjamin Kesler
"Dissecting long non-coding RNA regulation with single-cell image analysis"
Benjamin Kesler, Alexander Thiemicke, Rohit Venkat, Gregor Neuert
Department of Molecular Physiology and Biophysics, School of Medicine, Vanderbilt University, TN

Human diseases are often caused by abnormal biological processes within specific cell types or individual cells within a tissue, yet most studies have focused on cell populations and ignored possible differences between individual cells. One reason is a lack of easily implemented and broadly applicable experimental and computational approaches for microscopy image acquisition and analysis. We propose an approach in which we optimize the experimental data acquisition prior to analysis to generate high quality images that are easier to analyze computationally. The images are then processed and analyzed in parallel to determine cellular and nuclear boundaries, which allows for single-cell quantification of features of interest. We applied our approach to investigate mammalian antisense long noncoding RNAs (lncRNAs) Tsix and Xist. By utilizing two-color, single-molecule RNA-FISH and our image acquisition and analysis pipeline, we were able to quantify Tsix and Xist at single-cell and single-molecule resolution in more than 2000 cells and perform analyses that suggested transcription-based mechanisms of inhibition instead of the transcripts themselves. This data and generalizable pipeline set the stage for further quantitative investigation of this locus as well as other features across diverse species, cell types, and imaging modalities.

ABSTRACTS

#18 Garrett Sager
"Quantifying the Effects of Overexpression During Fluorescence Recovery After Photobleaching"
Garrett Sager[1,2], Ryoichi Kawai[2] and Elizabeth Sztul[1]
1. Department of Cell, Developmental and Integrative Biology
2. Department of Physics
University of Alabama at Birmingham

As techniques to understand intracellular activity advance, difficulties to understand the nuances of each experimental and theoretical approach ensue, potentially leading to misinterpreted data. One such experiment is fluorescence recovery after photobleaching (FRAP), which is done by overexpressing a fluorescent tagged protein, photobleaching the molecules in a given area, then measuring how quickly the surrounding, photoactive proteins are exchanged with the photobleached proteins. Using this information, theorists devised a simple formula to measure the diffusion constant using the time required to reach half the maximum recovery, t1/2, and the radius of the bleached area. Thus, this technique was originally developed to measure diffusion. As time progressed, FRAP began to also be used to measure the reactive properties of proteins. However, the law of mas action predicts overexpression should alter naturally occurring reaction rates. While this potential downside is well agreed upon conceptually, there is a lack of quantitative evidence showing this effect. Using a mathematical analysis and Monte Carlo simulation, we explore how overexpression during FRAP could alter the way we understand vesicles are initially formed at the cis-Golgi.

#19 Alexander Thiemicke
"Kinetic changes of hypertonic stress differentially regulate caspase activation dynamics and cell fate in human cells"
Alexander Thiemicke and Gregor Neuert
Department of Molecular Physiology and Biophysics, School of Medicine, Vanderbilt University, Nashville, TN

Cells are frequently exposed to changes in their environment. Such kinetic environmental changes may result in differential regulation of cell signaling, phenotype and cell fate. Yet studies on how kinetic changes of extracellular perturbations affect dynamics of signaling are in its infancy. To demonstrate the importance of kinetic changes in the environment, we chose hyperosmotic stress by NaCl that has been shown to be critically involved in physiological and pathophysiological processes in several human tissues. Resident immune cells can get activated by NaCl in such tissues and contribute to inflammation. We demonstrate that kinetic NaCl concentration gradients have a differentiating effect on cell signaling and cell fate. Upon instant addition of 300 mosmol/kg NaCl (step), viability decreases strongly (to below 10%) within 5h in human T cell and monocytic cell lines. When the same concentration is reached gradually over a period of ten hours (ramp), cell viability only decreases to 40%. To investigate molecular mechanisms, we use fluorescent cell barcoding for flow cytometry of intracellular processes to screen for dynamic changes in apoptosis, proliferation, DNA damage, inflammation, MAPK signaling and translation. This screen reveals a differential regulation of caspase and stress signaling between step and ramp NaCl application. Other processes, such as proliferation, inflammation and translation show a dependence on the cumulative exposure of NaCl, but are independent of the kinetic profile of NaCl application. When applying NaCl by ramp the activation of caspases is inhibited, which is sufficient for improving viability to the same level observed in pan-caspase inhibition by drug. These results suggest that tissues may depend on specific kinetic environmental changes to regulate signaling and cell fate and we identified signaling through caspase pathways as sensor of kinetic environments. Quantitative determination of the effect of kinetic environmental changes on human cells will help to better understand normal physiology and diseases associated with high NaCl such as cancer, cardiovascular and autoimmune diseases.

ABSTRACTS

#20 Michael Blinov
"The Center for Reproducible Biomedical Modeling"
Michael L Blinov[1], David P. Nickerson[2], John H. Gennari[3], Arthur P. Goldberg[4], Jonathan R. Karr[4], Ion I. Moraru[1], Herbert M. Sauro[3]
1. University of Connecticut School of Medicine
2. University of Auckland
3. University of Washington
4. Icahn School of Medicine at Mount Sinai

The Center for Reproducible Biomedical Modeling is an NIH-funded center which aims to enable comprehensive predictive models of biological systems, such as whole-cell models, that can guide medicine and bioengineering. Achieving this goal requires new tools, resources, and best practices for systematically, scalably, and collaboratively building, simulating and applying models, as well as new researchers trained in comprehensive modeling. To meet these needs, the center develops new technologies for comprehensive modeling, works with journals to provide authors, reviewers, and editors model annotation and validation services, and organizes courses and meetings to train researchers to model systematically, scalably, and collaboratively. More information about the center is available at http://reproduciblebiomodels.org.

#21 Jason Hughes
"Interrogating the role of chromatin regulation in single-cell transcription"
Jason Hughes, Amanda Johnson, Gregor Neuert
Department of Molecular Physiology and Biophysics, School of Medicine, Vanderbilt University

Chromatin regulation through nucleosome positioning and histone post-translation modification is a major step in determining gene expression in cells. The two main types of chromatin regulators (CR), ATP-dependent chromatin remodelers and histone modifiers, are often multi-protein complexes that are multifunctional in the different modifications they can cause. Five evolutionary conserved chromatin regulators (SAGA, INO80, Rpd3, RSC, SWI/SNF) have previously been investigated at the population-level in stress response pathways. The transcriptional role of these regulators should also be studied at the single cell level as many different models of gene-regulation can be described by the same population-level transcriptional dynamics. When S. cerevisiae are exposed to changes in osmolarity, the High-osmolarity glycerol (HOG) MAPK pathway is activated and phosphorylated HOG1 is translocated to the nucleus. In the nucleus, Hog1 together with SAGA, INO80, Rpd3, RSC, SWI/SNF regulate the cells chromatin state resulting in changes in transcription that prepare cells for future stresses. Therefore, the HOG pathway is an ideal system to study how chromatin remodelers impact gene expression in single cells. To better understand these processes and to distinguish between direct and indirect effects of chromatin regulators, we generate conditional mutant strains of selected chromatin remodeling proteins. We will quantify gene expression changes in single cells to understand how chromatin remodeling proteins impact transcription and how these changes protect cells from osmotic stress. These quantitative single cell experiments will help us to better understand fundamental mechanisms of how chromatin regulating proteins impact transcription and contribute to phenotype in single cells.

ABSTRACTS

#22 Rashida Abdul-ganiyu

"Isolation and characterization of a biosensor for monitoring phosphorylated Gbg during yeast gradient sensing [in mating yeast]"

Rashida Abdul-Ganiyu, Leon A. Venegas, Brian Kay and David E Stone. University of Illinois at Chicago. Chicago, IL

The ability of cells to respond to chemical gradients is essential across eukaryotic species. Chemotropism (directed growth) is necessary for cellular processes like axonal guidance, angiogenesis, pollen tube guidance, and fungal life cycles. We use the mating response of the budding yeast, Saccharomyces cerevisiae, as a model-system to study how cells interpret chemical gradients. During mating, yeast cells secrete peptide pheromones that bind to cell surface receptors on the opposite mating type. Binding of pheromone triggers a signaling cascade that results in cell-cycle arrest, formation of mating projections, and cell fusion to form zygotes. We have devised a model that explains how cells sense pheromone gradients and establish a chemotropic growth site (CS) oriented toward their partners. In mating mixtures, this occurs in four phases; global internalization of the receptor and G protein, polarization of signaling and trafficking proteins (Gradient Tracking Machine, GTM) at the default polarity site (DS) (assembly), redistribution (tracking) of the GTM upgradient from the DS to the CS, and stabilization of the GTM at the CS shortly before the onset of chemotropic growth. The G protein bg subunit (Gbg) is a critical part of this process. We have made predictions about the relative distribution of Gbg and receptor species during tracking of the GTM, which are central to our working model. To test these predictions and to better understand the spatio-temporal dynamics of phosphorylated Gbg (GbPg) during gradient sensing, we have developed a GbPg biosensor. Using phage-display affinity-selection experiments, we identified a Forkhead Associated (FHA) variant, that bound with high apparent affinity to a GbP peptide in vitro. In vivo characterization experiments showed that this FHA variant (the biosensor) physically interacts with GbPg and not to unphosphorylated Gbg in yeast cells. Further analysis to assess the utility of the biosensor revealed that, just like GFP-Gb (a Gbg reporter), the biosensor redistributes from the DS to the CS during gradient sensing.

List of Participants

Rashida Abdul-Ganiyu
University of Alabama
At Birmingham
rabdul9@uic.edu

Taiaba Afrin
University of Alabama
at Birmingham
taiaba@uab.edu

Nasim Ahmed
University of Alabama
at Birmingham
anasim@uab.edu

Belinda Akpa
North Carolina State University
bsakpa@ncsu.edu

Bradly Alicea
Orthogonal Research and
Education Lab
Bradly.alicea@outlook.com

Allyson Angermeier
University of Alabama
at Birmingham
arranger@uab.edu

Mounica Bandela
University of Illinois
at Chicago

Nicholas Barbera
University of Illlinois
at Chicago

Aundrea Bartley
University of Alabama
At Birmingham
abartley@uab.edu

Soniya Bastola
University of Alabama
At Birmingham
sbastola@uab.edu

Kevin Battles
University of Alabama
At Birmingham
danielkb@uab.edu

Abby Beatty
Auburn University
aeb0084@auburn.edu

Jazmine Benjamin
University of Alabama
at Birmingham
jibenj@uab.edu

Julien Berro
Yale University
julien.berro@yale.edu

Michael Blinov
University of Connecticut Health
blinov@uchc.edu

Yves Chiswili Chabu
University of Missouri
chabuc@missouri.edu

Chenbei Chang
University of Alabama
at Birmingham
cchang@uab.edu

Amanda Clark
Auburn University
adc0032@tigermail.auburn.edu

Joseph Cleland
Vanderbilt University
joseph.a.cleland@vanderbilt.edu

Renee Dale
Louisiana State University
rdale1@lsu.edu

Yasaman Dasteh Goli
University of Maryland
yas.ds@Hotmail.com

Kimia Dasteh Goli
University of Illinois
at Chicago
kdaste2@uic.edu

Thomas Detchemendy
University of Alabama
at Birmingham
tdutch@uab.edu

Danish Diwan
University of Alabama
at Birmingham
danishd@uab.edu

Lynn Dobrunz
University of Alabama at Birmingham
dobrunz@uab.edu

Jordan Fauser
University of Illinois
at Chicago
jfause2@uic.edu

Aleksandra Foksinska
University of Alabama
at Birmingham
afoksin@uab.edu

Khalid Freij
University of Alabama
at Birmingham
kfreij95@uab.edu

Mehran Ghafari
University of Tennessee
Chattanooga

Karen Gonzalez
University of Georgia
Karen. Gonzalez@uga.edu

Christopher Graham
University of Alabama
at Birmingham
cdg2t@uab.edu

Morgan Greene
University of Alabama
at Birmingham
meg3@uab.edu

Haobo Guo
University of Tennessee
Chattanooga

Priyanka Gupta
University of Alabama
at Birmingham
prigupta@uab.edu

Alison Ha
University of Illinois
at Chicago
aha6@uic.edu

Quamarul Hassan
University of Alabama
at Birmingham
hassank@uab.edu

List of Participants

Yecheng He
University of Alabama
at Birmingham
ianjuc@uab.edu

Timothy Howton
University of Alabama
At Birmingham
tchowton@uab.edu

Jason Hughes
Vanderbilt University

Wonpil Im
Lehigh University
wonpil@lehigh.edu

Hossein Jashnsaz
Vanderbilt University
hjashnsaz@gmail.com

Muhammad Zaki Jawaid
University of California Davis
zjawaid@ucdavis.edu

Amanda Johnson
Vanderbilt University
Amanda.meyer@vanderbilt.edu

Shreya Kashyap
University of Alabama
At Birmingham
skashyap@uab.edu

Ryoichi Kawai
University of Alabama
At Birmingham
kawai@uab.edu

Benjamin Kesler
Vanderbilt University
Benjamin.k.kesler@vanderbilt.edu

Jasim Khan
University of Alabama
At Birmingham
jasimk@uab.edu

Nilesh Kumar
University of Alabama
At Birmingham
Nileshkr@uab.edu

Damian Kuna
University of Alabama
at Birmingham
dkuna@uab.edu

Stephen Lenzini
University of Chicago
at Illinois

Ding Li
University of Illinois
at Chicago
rabdul9@uab.edu

John Lineham
DePaul University

Carlos Lopez
Vanderbilt University
c.lopez@Vanderbilt.Edu

Jarrad Marcell
University of Illinois
at Chicago
jarrad.marcel@gmail.com

Justyna Meissner
University of Alabama
at Birmingham
Meissner@uab.edu

Rita Miller
National Science Foundation
rimiller@nsf.gov

Bharat Mishra
University of Alabama
at Birmingham
bharat26@uab.edu

Kasturi Mitra
University of Alabama
at Birmingham
kasturi@uab.edu

Diego Moncada
University of Georgia
Diego.MoncadaGiraldo@uga.edu

William Monroe
University of Alabama
at Birmingham
wsmonroe@uab.edu

Shahid Mukhtar
University of Alabama
at Birmingham
smukhtar@uab.edu

Tomasz Nawara
University of Alabama
at Birmingham
tnawara@uab.edu

Gregor Neuert
Vanderbilt University
gregor.neurt@vanderbilt.edu

Jordan Newson
University of Illinois
at Chicago

Thi Nguyen
University of Alabama
at Birmingham
kimthi@uab.edu

Nicholas Panchy
University of Tennessee
Knoxville
panchy@nimbios.org

Hannah Pennington
University of Illinois
at Chicago
hpenni2@uic.edu

Zachary Petty
University of Georgia
Zachary.petty25@uga.edu

Hong Qin
University of Tennessee
at Chattanooga
Hong-Qin@utc.edu

Mohammad Mijanur Rahman
University of Alabama
at Birmingham
mijanur@uab.edu

List of Participants

Tejeshwar Rao
University of Alabama
At Birmingham
tjrao@uab.edu

Wouter-Jan Rappel
University of California
at San Diego
rappel@physics.ucsd.edu

Heather Ray
University of Alabama
At Birmingham
hjray@uab.edu

Gregory Reeves
North Carolina State University
gtreeves@ncsu.edu

Mohamned Rigi
University of Alabama
At Birmingham
rigi@uab.edu

Marcela Rojas-Pierce
North Carolina State University
mrojasp@ncsu.edu

Garrett Sager
University of Alabama at Birmingham
gsager56@uab.edu

Katrina Sahawneh
University of Alabama at Birmingham
ksahawne@uab.edu

John Schoelz
University of Alabama at
Birmingham
schoelzj@uab.edu

Hwaseon Seo
University of Alabama at
Birmingham
shs0626@uab.edu

Manvi Sharma
University of Alabama
at Birmingham
manvi@uab.edu

Rosalie Sinclair
University of California Davis
rmsinclair@ucdavis.edu

Sean Snowden
Morehouse College
sean.snowden@morehouse.edu

Piotr Stasiak
University of Alabama
at Birmingham
stasiak@uab.edu

Bethanie Michelle Statler
University of Illinois
at Chicago
estatl2@uic.edu

David Stone
University of Illinois
at Chicago
dstone@uic.edu

Tara Sudhadevi
University of Illinois
at Chicago
taras@uic.edu

Yali Sun
University of Alabama
at Birmingham
yalisun@uab.edu

Elizabeth Sztul
University of Alabama
at Birmingham
esztul@uab.edu

Daniel Szymanski
Purdue University
szymandb@purdue.edu

Alexander Thiemicke
Vanderbilt University
alexander.thiemicke@vanderbilt.edu

Kaushlendra Tripathi
University of Alabama
at Birmingham
tripathi@uab.edu

Joseph Turner
University of Nebraska-
Lincoln
jaturner@unl.edu

Peyton Vanwinkle
University of Alabama

at Birmingham
pev18@uab.edu

Rohit Venkat
Vanderbilt University
rohit.venkat@vanderbilt.edu

Becky Wakefield
University of Alabama
at Birmingham
beckyw@uab.edu

Jeff Walker
University of Alabama
at Birmingham

Melissa Walker
University of Alabama
at Birmingham
sfidemw@uab.edu

Shuzhen Wang
University of Alabama
at Birmingham

Tayleur White
University of Alabama
at Birmingham
twhite1@uab.edu

William Willoughby
University of Alabama
at Birmingham
wrwillou@uab.edu

Sin Wan Wong
University of Illinois
at Chicago
swong30@uic.edu

List of Participants

Shaolong Yang
University of Alabama
At Birmingham
shaolongyang@uabmc.edu

Liqun Zhang
Tennessee Tech
lzhang@tntech.edu

DINING

Dining Around Birmingham:

Lucy's Coffee & Tea
Gourmet coffees and teas accompanied by breakfast pastries, sandwiches, and fresh salads
Open for breakfast and lunch
Dress code: Casual
Phone: +1 205-328-2007
Address: 2007 University Blvd, Birmingham, AL 35233

Wings Around the Clock
Casual spot for hot wings with a variety of sauces
Open for Lunch and Dinner
Dress code: Casual
Phone: +1 205-581-8088
Address: 801 20th St S, Birmingham, AL 35205

Makarios Kabobs & Grill
Greek, Mediterranean, and American Cuisine
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-731-7414
Address: 940 20th St S, Birmingham, AL 35205

Sitar Indian Cuisine
Serving authentic Indian cuisine
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-323-6500
Address: 1801 4th Avenue South, Birmingham, AL 35233

Highland's Bar and Grill $$
Celebrity Chef Frank Stitt's signature restaurant blends French Classics with Southern Favorites.
Nominated for 2011 James Beard Foundation "Restaurant of the Year".
Reservations required**
Open for dinner
Dress code: Shirt/Slacks Req.
Phone: +1 205-939-1400
Address: 2011 11th Ave S, Birmingham, AL 35205

Chez Fonfon $$
Located in Five Points South next door to big brother, Highlands Bar and Grill, Chez Fonfon is a bustling French café focusing on provincial food and wine.
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-939-3221
Address: 2007 11th Ave S, Birmingham, AL 35205

DINING

Surin West
Traditional Thai favorites in a hip Five Points South location.
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-324-1928
Address: 1918 11th Ave S, Birmingham, AL 35205

Jim 'N Nick's Bar-B-Q
Barbeques cooked "low and slow" using a 25 year old recipe. Everything is made from scratch
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-320-1060
Address: 1908 11th Ave S, Birmingham, AL 35205

The Original Pancake House
The Original Pancake House was founded in Portland, Oregon in 1953 by Les Highet and Erma Hueneke. Drawing upon their many years of experience in the culinary field, and their extensive working knowledge of authentic national and ethnic pancake recipes.
Open for breakfast and lunch
Dress code: Casual
Phone: +1 205-933-8837
Address: 1931 11th Ave S, Birmingham, AL 35205

The Fish Market
Experience Greek influenced fresh seafood at a Birmingham landmark
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-322-3330
Address: 612 22nd St S, Birmingham, AL 35233

Mellow Mushroom
Gourmet pizza and a great selection of craft beers offered in an eclectic Five Points South location
Open for lunch and dinner
Dress code: Casual
Phone: +1 205-933-5061
Address: 1200 20th St S Ste 100, Birmingham, AL 35205

Bottega Restaurant & Café $$
Splendid Italian and Mediterranean cuisine await in this elegant historical building. Chef Frank Stitt is nominated for the 2011 James Beard Foundation "Chef of the Year".
Reservations required**
Open for lunch and dinner
Dress code: Shirt/Slacks Req.
Phone: +1 205-939-1000
Address: 2240 Highland Ave, Birmingham, AL 35205

DINING

The J. Clyde
Best place in Birmingham to drink beer with a beer menu that exceeds 8 pages. Great pub food as well.
Open for dinner
Dress code: Casual
Phone: +1 205-939-1312
Address: 1312 Cobb Ln, Birmingham, AL 35205

Ocean $
Opened by George Reis in 2002, and located in the trendy Five Points district of Birmingham's Southside. Enjoy a casual night out with friends at the inviting and contemporary patio bar, or venture inside for some of the best seafood in town.
Reservations required**
Open for dinner
Dress code: Shirt/Slacks Req.
Phone: +1 205-933-0999
Address: 1218 20th St S, Birmingham, AL 35205

Galley and Garden $
Traditional Southern Cuisine is fused with flavors of New Orleans and the Caribbean
Reservations required**
Open for dinner
Dress code: Shirt/Slacks Req.
Phone: +1 205-939-5551
Address: 2220 Highland Ave, Birmingham, AL 35205

Taco Mama
Authentic Mexican restaurant
Dress code: Casual
Open for lunch and dinner
Phone: +1 205-644-8355
707 Richard Arrington Blvd South

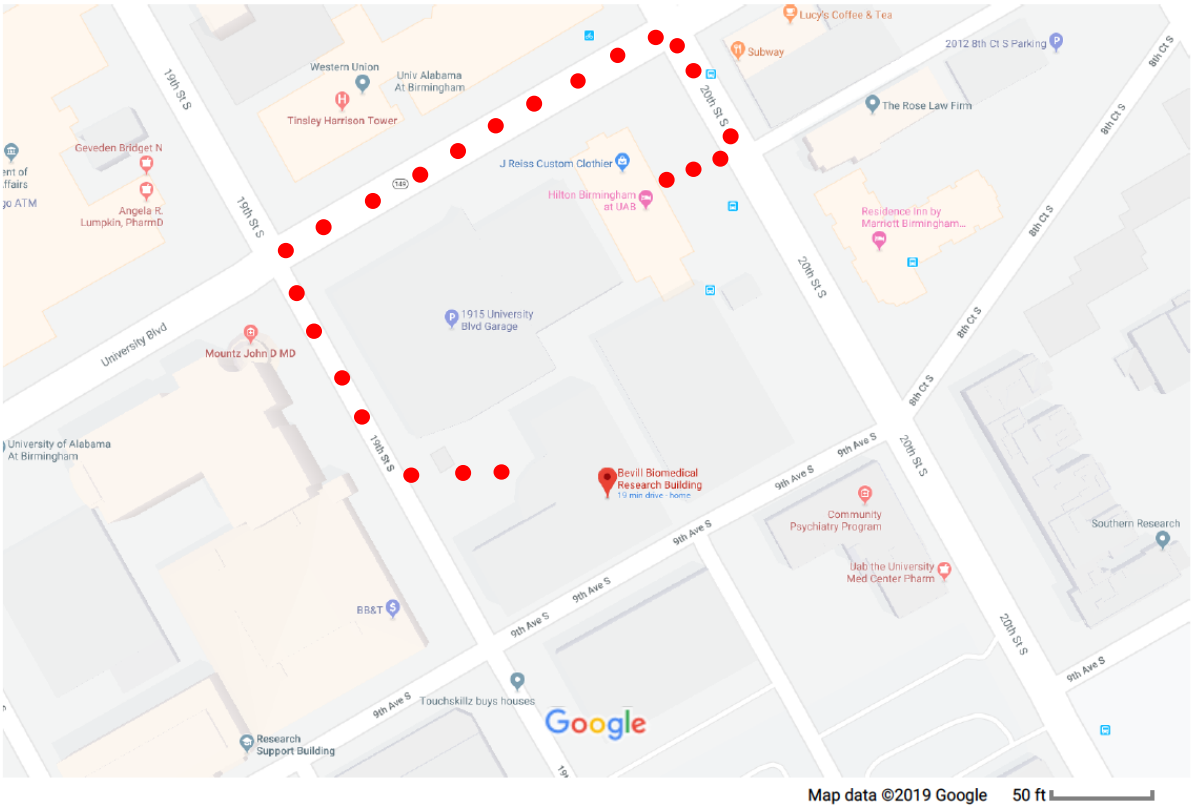
NOTES

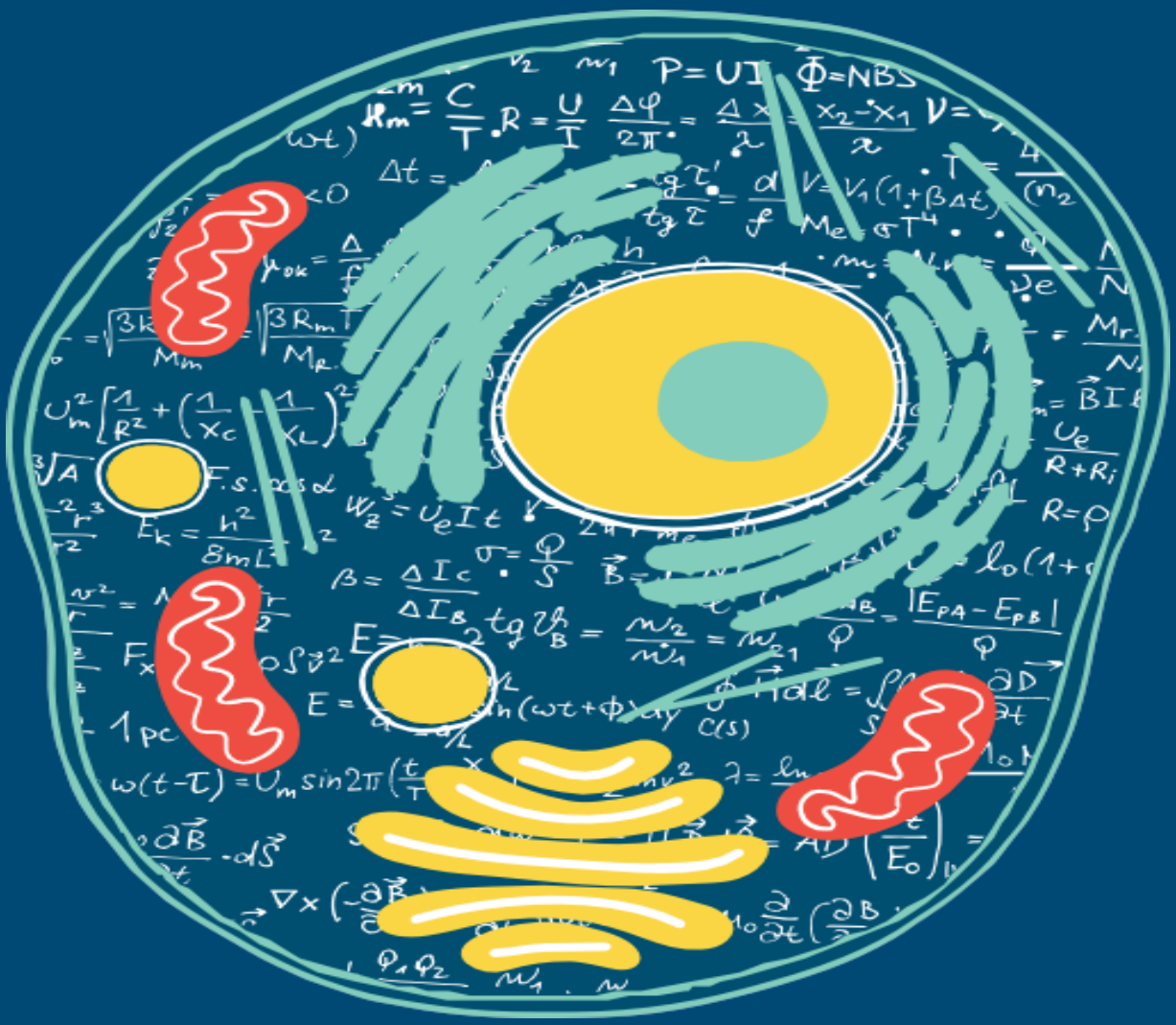
NOTES

NOTES

NOTES

Google Maps Bevill Biomedical Research Building





Finding Your Inner Modeler-3

On behalf of the Finding Your Inner Modeler III organizers, we would like to say thank you for your support of our 2019 Meeting.

Directors: Drs. Elizabeth Sztul, Ryoichi Kawai, Carlos Lopez, and David Stone

Administrative Team: Sarita Womack, Rene Eubank, and Megan Rollins

This work was sponsored by NSF MCB award #1901738