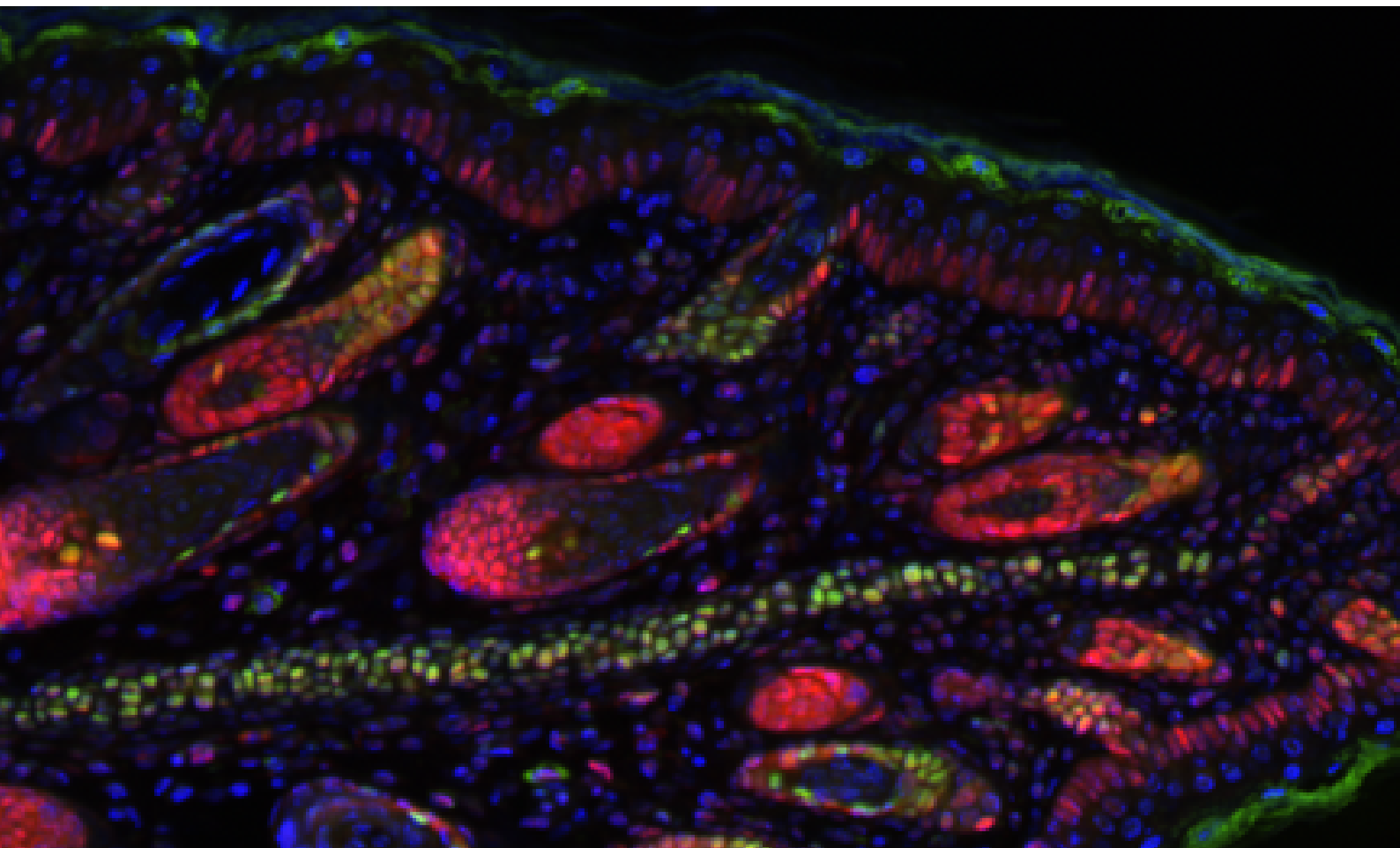
UC IRVINE

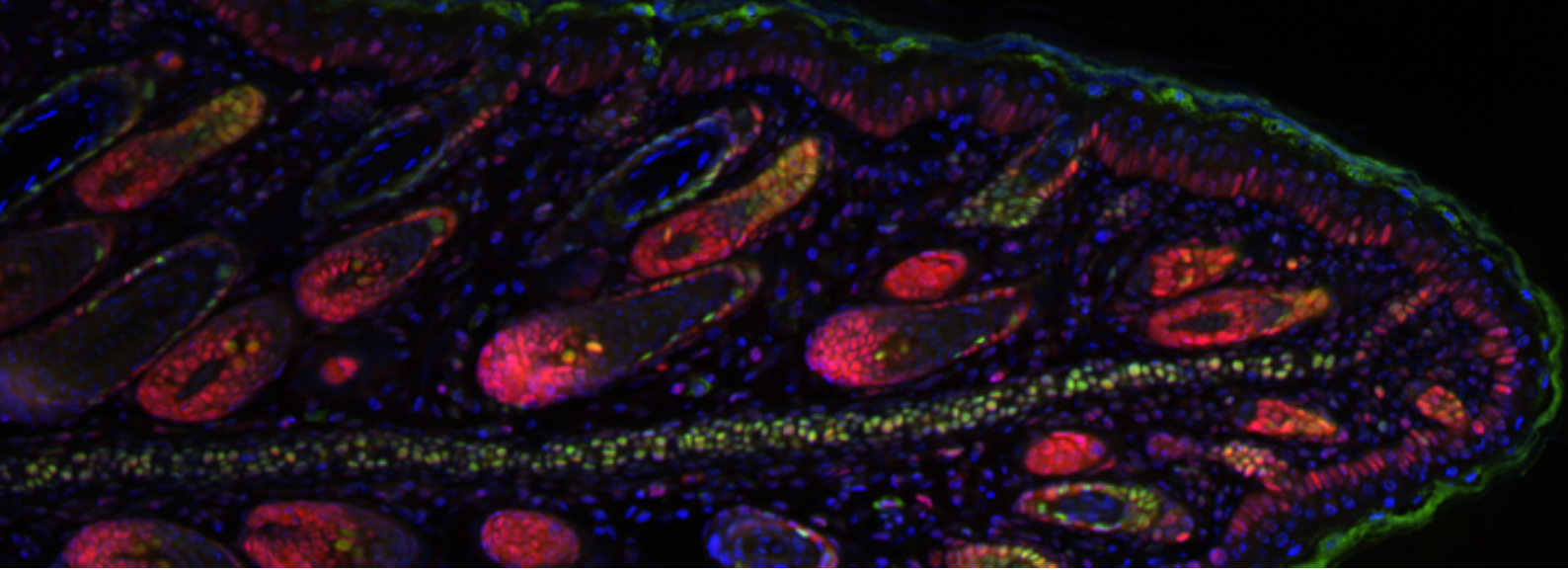
NSF-SIMONS CENTER FOR
MULTISCALE CELL FATE RESEARCH

INAUGURAL SYMPOSIUM ON MULTISCALE CELL FATE

OCTOBER • 1-2 • 2018

BECKMAN CENTER OF THE NATIONAL
ACADEMIES OF SCIENCES AND ENGINEERING





SPEAKERS

MARK ALBER
University of California, Riverside

RUTH BAKER
Oxford University

XING DAI
University of California, Irvine

HANA EL-SAMAD
University of California, San Francisco

STEVEN HENIKOFF
HHMI & Fred Hutchinson Cancer Research Center

ALEX HOFFMANN
University of California, Los Angeles

SUI HUANG
Institute for Systems Biology

MANOLIS KELLIS
Broad Institute & MIT

ANDRE LEVCHENKO
Yale University

ALEX MOGILNER
New York University

PEGGY MYUNG
Yale University

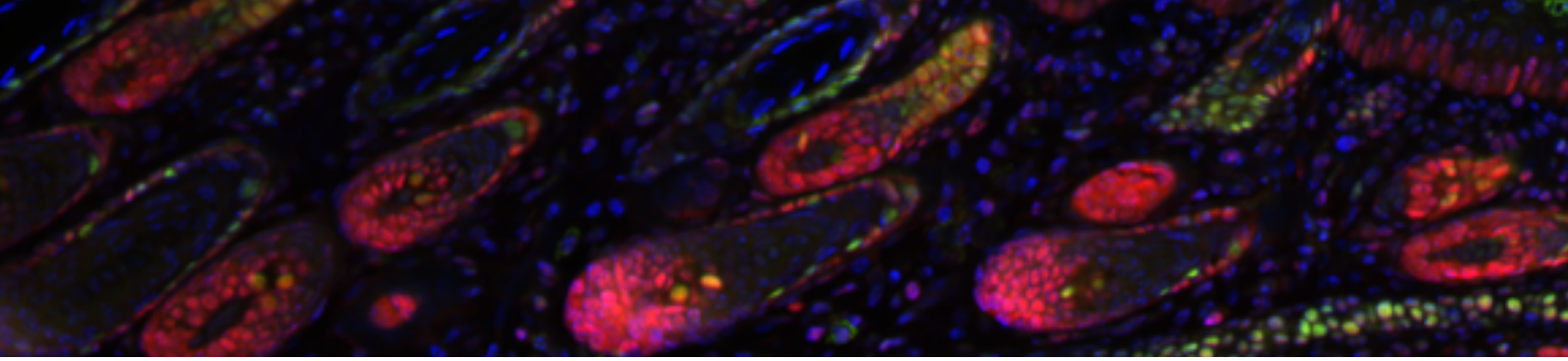
BING REN
University of California, San Diego

TATJANA SAUKA-SPENGLER
Oxford University

MICHAEL SHELLEY
Center for Computational Biology, Flatiron Institute
Courant Institute, New York University

LEOR WEINBERGER
University of California, San Francisco

DAVID WILKINSON
The Francis Crick Institute

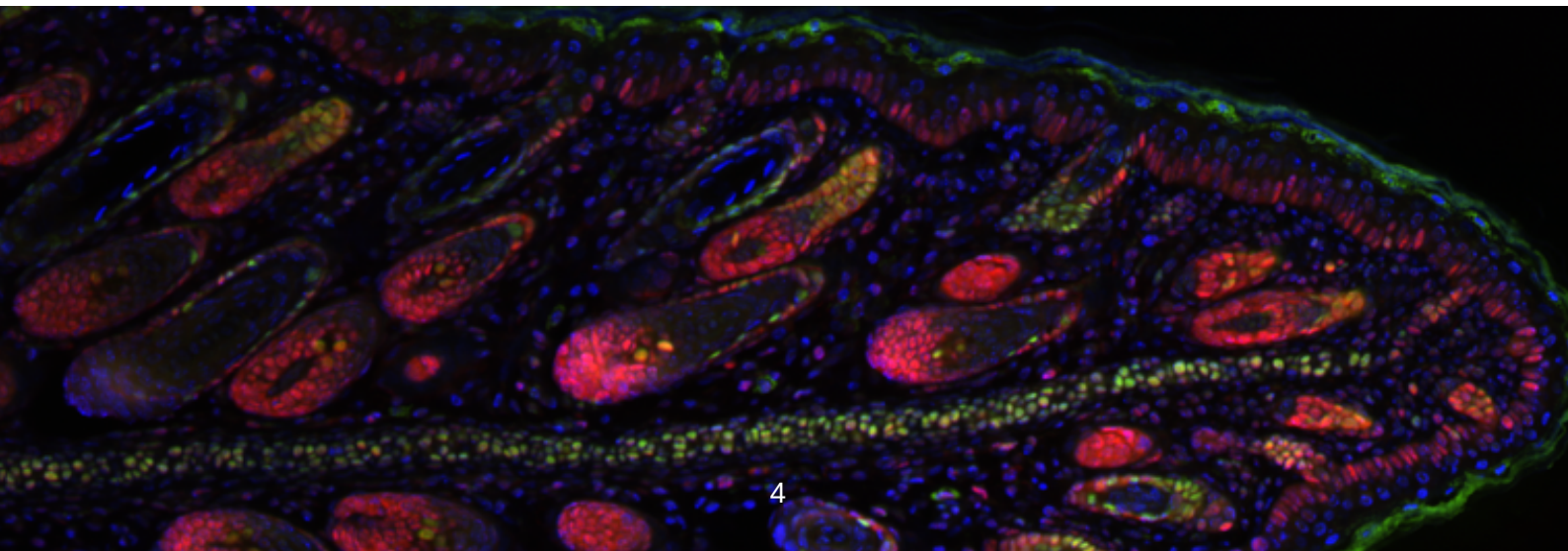


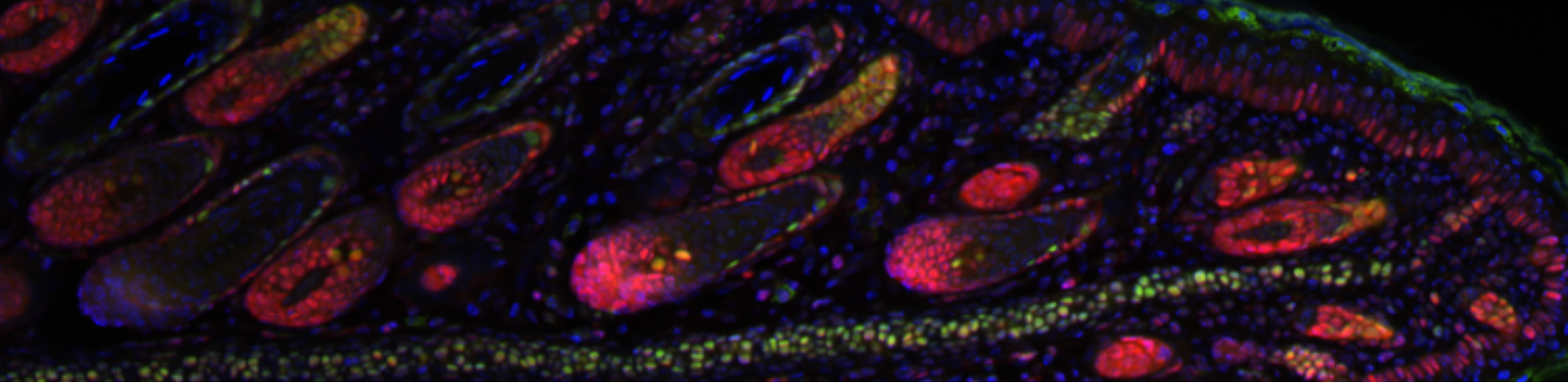
EVENT PROGRAM

MONDAY, OCTOBER 1

7:30 AM – 8:10 AM	**FULL BREAKFAST** Dining Room
8:10 AM - 8:15 AM	**OPENING ADDRESS** Pramod Khargonekar, UCI Vice Chancellor for Research
8:15 AM – 8:55 AM	**STRUCTURAL EPIGENOMICS: PRECISION MAPPING OF THE DYNAMIC NUCLEOSOME LANDSCAPE** Steven Henikoff
8:55 AM - 9:35 AM	**FUNCTIONAL ORGANIZATION OF THE HUMAN GENOME** Bing Ren
9:35 AM - 10:15 AM	**FROM GENOMICS TO THERAPEUTICS: UNCOVERING AND MANIPULATING THE GENOMIC CIRCUITRY OF HUMAN DISEASE** Manolis Kellis
10:15 AM - 10:20 AM	**INTERDISCIPLINARY OPPORTUNITY AWARD (IOA) ANNOUNCEMENT**
10:20 AM - 10:40 AM	**COFFEE BREAK** Atrium
10:40 AM - 11:20 AM	**SYNTHETIC BIOLOGY FOR SYSTEMS BIOLOGY** Hana El-Samad

11:20 AM - 12:00 PM	**SCALING AND POSITIONING OF MULTIPLE NUCLEI IN MUSCLE CELL** Alex Mogilner
12:00 PM – 1:30 PM	**LUNCH** Dining Room
1:30 PM - 2:10 PM	**CELL DIFFERENTIATION IN ANGIOGENESIS: SIGNALS AND DECISIONS** Andre Levchenko
2:10 PM - 2:50 PM	**PREDICTING CELL FATE DECISIONS – KNOWLEDGE-BASED VS DATA-DRIVEN MODELS** Alex Hoffmann
2:50 PM - 3:10 PM	**COFFEE BREAK & ISDR SIGNUPS** Students, postdocs, and all junior researchers invited
3:10 PM - 3:50 PM	**COLLECTIVE CELL INVASION: MATHEMATICAL MODELS AND BIOLOGICAL INSIGHTS** Ruth Baker
3:50 PM - 4:30 PM	**LIGHTNING TALKS BY JUNIOR RESEARCHERS**
4:30 PM - 5:15 PM	**INTERDISCIPLINARY SPEED-DATING FOR RESEARCH (ISDR)** Lawn
4:30 PM - 6:00 PM	**POSTER SESSION AND RECEPTION** Atrium and Courtyard
6:00 PM - 8:00 PM	**DINNER** Dining Room





EVENT PROGRAM

TUESDAY, OCTOBER 2

7:30 AM - 8:15 AM	**FULL BREAKFAST** Dining Room
8:15 AM - 8:55 AM	**MODELING AND SIMULATING ACTIVE MECHANICS IN THE CELL** Michael Shelley
8:55 AM - 9:35 AM	**MULTI-SCALE MODELS OF DEFORMATION AND EMBOLIZATION OF BLOOD CLOTS UNDER VARIABLE SHEAR FLOW** Mark Alber
9:35 AM - 10:35 AM	**COFFEE AND POSTER SESSION** Atrium
10:35 AM - 11:15 AM	**CELL FATE DECISIONS, MULTI-STABILITY, INSTABILITIES AND “WHY CANCER TREATMENT CAN BACKFIRE"** Sui Huang
11:15 AM - 11:55 AM	**HARNESSING TRANSCRIPTIONAL FLUCTUATIONS FOR CELL FATE CONTROL AND THERAPY** Leor Weinberger
12:00 PM - 1:30 PM	**LUNCH** Dining Room
1:30 PM - 2:10 PM	**BORDERS AND COMMUNITIES IN HINDBRAIN SEGMENTATION** David Wilkinson

2:10 PM - 2:50 PM

DECIPHERING GLOBAL NEURAL CREST GENE REGULATORY NETWORKS USING EPIGENOMIC, TRANSCRIPTIONAL AND CIS-REGULATORY PROFILING IN SPECIFIC CELL POPOLUATIONS IN VIVO

Tatjana Sauka-Spengler

2:50 PM - 3:10 PM

COFFEE BREAK

Atrium

3:10 PM - 3:50 PM

SINGLE-CELL ANALYSIS REVEALS A HAIR FOLLICLE DERMAL NICHE DIFFERENTIATION PATH THAT BEGINS BEFORE MORPHOGENESIS

Peggy Myung

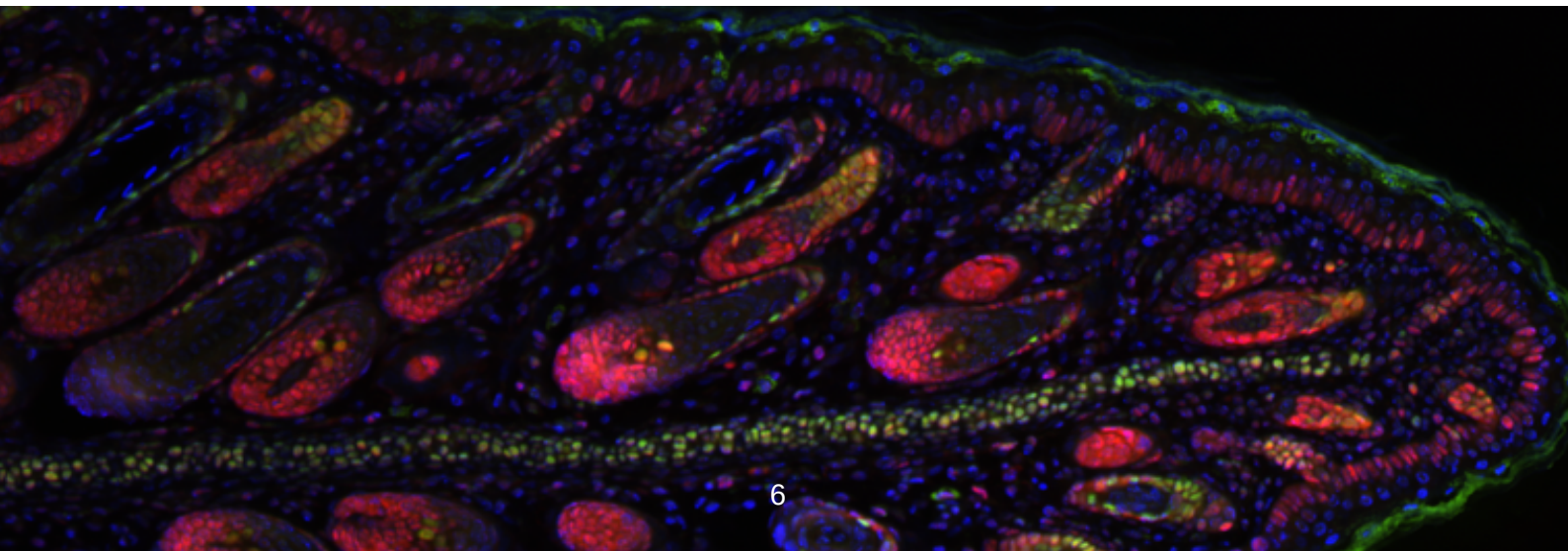
3:50 PM - 4:30 PM

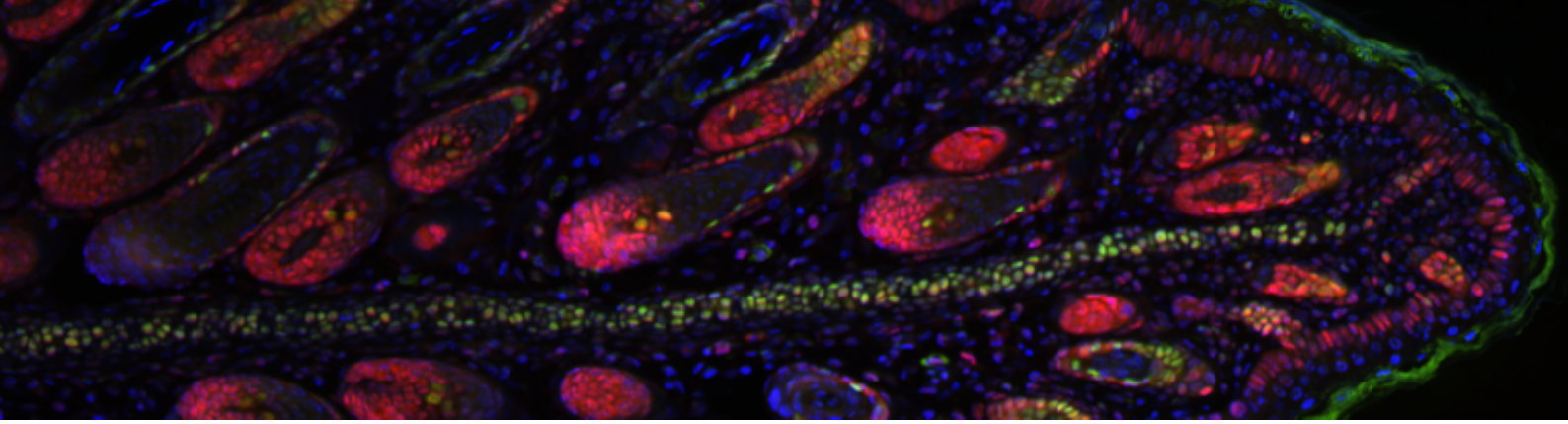
“EMT” CONTROL IN DEVELOPING AND REGENERATING EPITHELIA

Xing Dai

4:30 PM -

IOA ABSTRACT DUE, AND ADJOURN





ABSTRACTS

MONDAY, OCTOBER 1

Collective cell invasion: mathematical models and biological insights

RUTH BAKER

Cell invasion is fundamental to embryonic development, tissue regulation and regeneration, and the progression of many diseases, including cancer; yet the mechanisms that drive populations of cells to invade distinct targets are poorly understood. This talk will utilise the embryonic neural crest as an exemplar to study mechanisms of collective cell invasion. I will present results from an interdisciplinary collaboration that demonstrate the roles of population heterogeneity, and cell-cell and cell-microenvironment interactions, in driving invasion. I will also discuss future theoretical challenges in the field, and outline progress my group has made towards tackling them.

Synthetic Biology for Systems Biology

HANA EL-SAMAD

In this talk, we discus how precise tools for perturbation of cellular pathways should be used to extract precise answers, beyond statistical representations.

Structural epigenomics: Precision mapping of the dynamic nucleosome landscape

STEVEN HENIKOFF

We have developed a suite of genome-wide tools for high-resolution epigenomic mapping to provide mechanistic insights into nucleosome and transcription factor dynamics. Using a metabolic labeling approach, we have observed competition between nucleosomes and transcription factors behind the replication fork. We have identified asymmetrically unwrapped nucleosomes as structural features that result from transcription and remodeling. Our structural epigenomics strategy is general, including potential applications for non-invasive diagnosis of disease. Using a new method for probing the composition of chromatin complexes we have probed the mechanisms whereby promoters are dynamically maintained free of nucleosomes for initiation of transcription.

Predicting cell fate decisions – knowledge-based vs data-driven models

ALEX HOFFMAN

Predicting cell fate decisions is a key goal in systems biology and fundamental to clinical decision-making. Whereas the former typically involves kinetic models based on knowledge of molecular mechanism, the latter is typically based on measurements of "predictive biomarkers" and machine learning models. Examining the cellular decision to undergo apoptosis, we have related the two approaches to each other and thus gained some tantalizing insights about the determinants of the predictive power of clinical biomarkers.

From Genomics To Therapeutics: Uncovering And Manipulating The Genomic Circuitry of Human Disease

MANOLIS KELLIS

Perhaps the greatest surprise of human genome-wide association studies (GWAS) is that 90% of disease-associated regions do not affect proteins directly, but instead lie in non-coding regions with putative gene-regulatory roles. This has increased the urgency of understanding the non-coding genome, as a key component of understanding human disease. To address this challenge, we generated maps of genomic control elements across 127 primary human tissues and cell types, and tissue-specific regulatory networks linking these elements to their target genes and their regulators. We have used these maps and circuits to understand how human genetic variation contributes to disease and cancer, providing an unbiased view of disease genetics and sometimes re-shaping our understanding of common disorders. For example, we find evidence that genetic variants contributing to Alzheimer's disease act primarily through immune processes, rather than neuronal processes. We also find that the strongest genetic association with obesity acts via a master switch controlling energy storage vs. energy dissipation in our adipocytes, rather than through the control of appetite in the brain. We also combine genetic

information with regulatory annotations and epigenomic variation across patients and healthy controls to discover new disease genes and regions with roles in Alzheimer's disease, heart disease, prostate cancer, and to understand cellular diversity through single-cell RNA-seq and pleiotropic effects by integration with rich intermediate phenotypes and electronic health records. Lastly, we develop systematic technologies for systematically manipulating these circuits by high-throughput reporter assays, genome editing, and gene targeting in human cells and in mice, demonstrating tissue-autonomous therapeutic avenues in Alzheimer's disease, obesity, and cancer. These results provide a roadmap for translating genetic findings into mechanistic insights and ultimately therapeutic treatments for complex disease and cancer.

Cell differentiation in angiogenesis: signals and decisions

ANDRE LEVCHENKO

Angiogenesis is a key developmental, physiological and pathological process, critical for proper functioning of any animal tissue. The formation of a new blood vessel involves complex signaling inputs and multi-layer signal processing. As a result, cells adopt distinct transient fates leading to growth and maturation of a vascular tree. In particular, cells can adopt the 'tip' and 'stalk' fates, associated with migration and proliferation. In spite of decades of intense research, we still know little about how these fates are chosen by the cells, and how this decision is biased by chemical inputs and coordinated within cell arrays. In this talk, I will review the recent results from our lab, providing the basis for understanding of this crucial decision making process, specifically how it might be controlled and pathologically altered by complex environmental inputs. The results indicate intricate encoding and decoding events, unfolding in time and modulated by homotypic and heterotypic cell interactions. We hypothesize that similar decision making principles may extend to other developmental and photogenic processes, including those associated with cancer progression.

Scaling and positioning of multiple nuclei in muscle cell

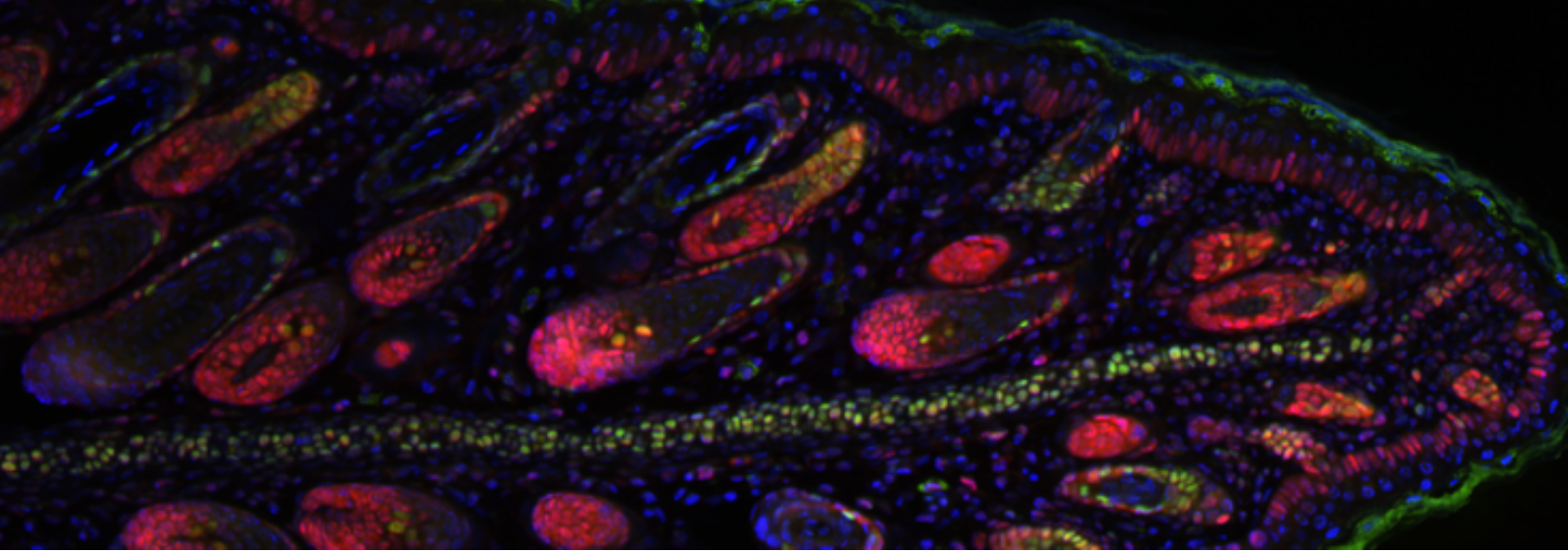
ALEX MOGILNER

Giant cells, including muscle cells, often have multiple nuclei. In muscle cells, the nuclei have to be distributed uniformly across the cell for reasons yet unknown; disruptions of such distribution lead to disease. We use microscopy and genetic perturbations in muscle cells in Drosophila embryo and larvae to, first, formulate a microtubule/motor force balance hypothesis of the nuclear positioning, and then use the data to constrain a computational screen of forces that explain stationary and dynamic distributions of the nuclei. Furthermore, we find that the nuclear sizes scale with the local cytoplasmic volume around the nuclei. We screen mechanical, transport and reaction-diffusion models of this scaling.

Functional Organization of the Human Genome

BING REN

The 3-dimentional architecture of chromosomes in eukaryotic cells enables long-range communication between enhancers and promoters, and contributes to spatiotemporal gene expression programs in multicellular species. Detailed knowledge of how chromatin architecture dynamically reorganizes during development and in different cell types is critical for studying the gene regulatory programs controlling cell fate specification and elucidating the molecular basis of human diseases. We have delineated the dynamic chromatin architecture at high resolution during key developmental stages of human cardiomyocyte differentiation from embryonic stem cells. We observed dramatic changes in chromatin compartments, topological domains and enhancer/promoter interactions, which was correlated with dynamic gene expression patterns. The chromatin loop interactions help us to predict target genes of non-coding genetic variants associated with cardiac-related traits/diseases. We also generate maps of long-range chromatin interactions centered on human promoters in a large panel of human cell/tissue types. We use this information to infer the target genes of candidate regulatory elements, and suggest potential regulatory function for non-coding sequence variants associated with a large number of physiological traits and diseases. Integrative analysis of these promoter-centered interactome maps reveals widespread enhancer-like promoters involved in gene regulation and common molecular pathways underlying distinct groups of human traits and diseases.



ABSTRACTS

TUESDAY, OCTOBER 2

Multi-scale models of deformation and embolization of blood clots under variable shear flow

MARK ALBER

Thromboembolism, one of the leading causes of morbidity and mortality worldwide, is characterized by formation of obstructive intravascular clots (thrombi) and their mechanical breakage (embolization). A novel two-dimensional multi-phase computational model will be described that simulates active interactions between the main components of the clot, including platelets and fibrin. It can be used for studying the impact of various physiologically relevant blood shear flow conditions on deformation and embolization of a partially obstructive clot with variable permeability. Simulations provide new insights into mechanisms underlying clot stability and embolization that cannot be studied experimentally at this time. In particular, model simulations, calibrated using experimental intravital imaging of an established arteriolar clot, show that flow-induced changes in size, shape and internal structure of the clot are largely determined by two shear-dependent mechanisms: reversible attachment of platelets to the exterior of the clot and removal of large clot pieces. Model simulations also predict that blood clots with higher permeability are more prone to embolization with enhanced disintegration under increasing shear rate. In contrast, less permeable clots are more resistant to rupture due to shear rate dependent clot stiffening originating from enhanced platelet adhesion and aggregation. These results can be used in future to predict risk of thromboembolism based on the data about composition, permeability and deformability of a clot under specific local haemodynamic conditions.

“EMT” control in developing and regenerating epithelia

XING DAI

Fates of epithelial cells are not fixed but instead can respond to myriad physiological, pathological and experimental stimuli. Epithelial-to-mesenchymal transition (EMT) is a recognized form of cell fate plasticity that converts epithelial cells (e.g. epiblast) into mesenchymal cell types (e.g., mesoderm) during embryogenesis. EMT-like processes are also strongly implicated in cancer metastasis and wound healing. Mild forms of epithelial plasticity exist in committed epithelial tissues, but how this plasticity is kept in check during development and regeneration is poorly understood. In our recent studies, we have identified transcription factors Ovol1/2 as key master inhibitors of EMT-like events in developing skin and mammary epithelia. We have shown EMT-promoting transcription factor Zeb1 as a functionally relevant direct target of Ovol2, and cell adhesion protein alpha-catenin as a novel target of Zeb1. Combining experiments with mathematic modeling, we have discovered a critical role of the Ovol2-Zeb1 circuit in generating and maintaining multiple intermediate states between terminal epithelial and mesenchymal-like epithelial states. Our ongoing work examines the importance of this circuit in adult skin and mammary epithelia, focusing particularly on its involvement during physiological and pathological regeneration.

Cell Fate Decisions, Multi-stability, Instabilities and “Why Cancer Treatment can Backfire"

SUI HUANG

Stable cell states, such as cell types, are attractor states in the gene regulatory network that controls the cell type-defining gene expression patterns and can be modeled as a non-linear dynamical system that exhibits multi-stability (multiple attractor states). We have proposed that a cell fate decision is a bifurcation event in this dynamical system: the stable progenitor state is destabilized, thereby forcing the multipotent cells in it to move to nearby attractors that represent the predestined lineage options to which the former can differentiate. This model can be formally linked to Waddington's epigenetic landscape and offers a framework for interpreting single-cell transcriptome data. This approach differs from the current data-driven heuristic computational methods aimed at discovering patterns in the data pattern but is agnostic of the underlying governing principles. The model predicts qualitatively distinct counterintuitive cell behaviors. In the talk I will present single-cell resolution gene expression profile measurements in cell populations undergoing cell fate decisions, and show how the data is consistent with two predictions of the theory: (i) a high-dimensional "Early Warning Signal" that precedes critical transitions and (ii) the existence of "rebellious cells" which manifests a phenotype switch in the direction opposite to the intended one. They could explain why cancer therapy, which seeks to push tumor cells into the apoptotic state, can instead induce a stem-like state in the surviving cells. Experimental results in mouse models will be presented and practical implications discussed, such as a possible theoretical limit of "curability" of cancer because the therapeutic intervention, due to instabilities in cell state dynamics and cellular heterogeneity, inevitably generates cancer-stem cells while killing others: the treatment backfires.

Single-cell analysis reveals a hair follicle dermal niche differentiation path that begins before morphogenesis

PEGGY MYUNG

How complex tissue structures emerge from a seemingly homogenous population of cells is a universal question in development. This phenomenon has been examined across numerous appendages, including teeth and hair follicles (HFs). However, the molecular signals that orchestrate these initial events in appendage formation remain poorly delineated. Specifically, it is unknown if molecular specification occurs prior to morphogenesis. The HF is one of the most tractable models to study organogenesis as its constituent populations, including its dermal niche (dermal condensate, DC) population are molecularly and morphologically defined. The HF-inductive properties of the DC are well-established, yet tracing steps that precede its morphologic emergence has been challenging, as conventional tools lack the ability to discriminate quantitative molecular differences between apparently uniform cells at a single-cell level. Combining unbiased single-cell RNA sequencing techniques with in vivo lineage analysis, we delineate key molecular and cellular events that direct DC cell fate prior to and during formation of a morphologic DC. We show that DC cells specify a differentiation trajectory, a path by which dermal cells differentiate into DC cells, that begins prior to HF initiation and originates from Wnt-activated dermal progenitors. Our integrated analysis shows that Wnt/β-catenin activation is required for dermal cell competency to progress into a dynamic phase of maturation and proliferation that leads to DC cell differentiation. Further, our combined analysis allowed us to revisit a decades-old theory, which proposed that quiescent differentiated DC cells are progeny of pre-DC cells that just divided. We localize this proliferating pre-DC population to the peri-DC region. Together, these findings uncover key stages in DC cell fate specification that were previously limited by morphological criteria while bringing new insight into mechanisms of niche establishment during organogenesis.

Borders and communities in hindbrain segmentation

DAVID WILKINSON

The formation of sharp borders between tissue subdivisions is important for establishing precise patterns of cell types. However, borders are fuzzy at early stages, likely due to imprecision in the spatial regulation of cell identity, and the intermingling of cells during morphogenesis. Studies of the vertebrate hindbrain have suggested that Eph/ephrin-mediated cell segregation and dynamic regulation of cell identity contribute to border sharpening. A further mechanism was suggested by studies in the early 2000s which showed that cells transplanted between segments switch identity to match their new neighbors. I will discuss our findings which reveal that cell intermingling and identity switching occurs during normal hindbrain development in zebrafish. We have uncovered a retinoid-mediated community effect which switches the identity of cells that intermingle into an adjacent segment. These findings reveal a mechanism that acts at early stages, prior to upregulation of Eph-ephrin-mediated cell segregation, to ensure that sharp and homogeneous segments are formed.

Deciphering global neural crest gene regulatory networks using epigenomic, transcriptional and cis-regulatory profiling in specific cell popoluations in vivo

TATJANA SAUKA-SPENGLER

TPrecise control of developmental processes is embedded in the genome in the form of gene regulatory networks (GRNs). Our current understanding of vertebrate GRNs is fragmentary and incomplete, because of the difficulties in performing the needed global analyses in vertebrate embryos. We have defined an integrated approach that embraces the complexity of vertebrates, and have tested it by conducting a comprehensive in vivo study of the GRN underlying the neural crest (NC). The NC is an emblematic vertebrate cell type, as it is a multipotent stem cell-like population characterised by its migratory ability and diverse derivatives. Genome-wide analyses of NC chromatin and transcriptional dynamics revealed the full complement of NC enhancers and their transcription factor inputs. We found 'super-enhancer'-like clusters and tested their operation using both in vivo reporters and epigenome engineering. Our pipeline integrates cis-regulatory data with single-cell transcriptomes, which enabled construction and functional validation of a comprehensive NC-GRN. The unprecedented resolution of our integrated analysis of NC development has broad implications for NC-associated diseases. The ability of our approach to define and test the GRN underlying a system as complex as the neural crest demonstrates its unique power to dissect gene regulatory circuits in vivo, with broad implications for vertebrate GRN discovery and study.

Modeling and simulating active mechanics in the cell

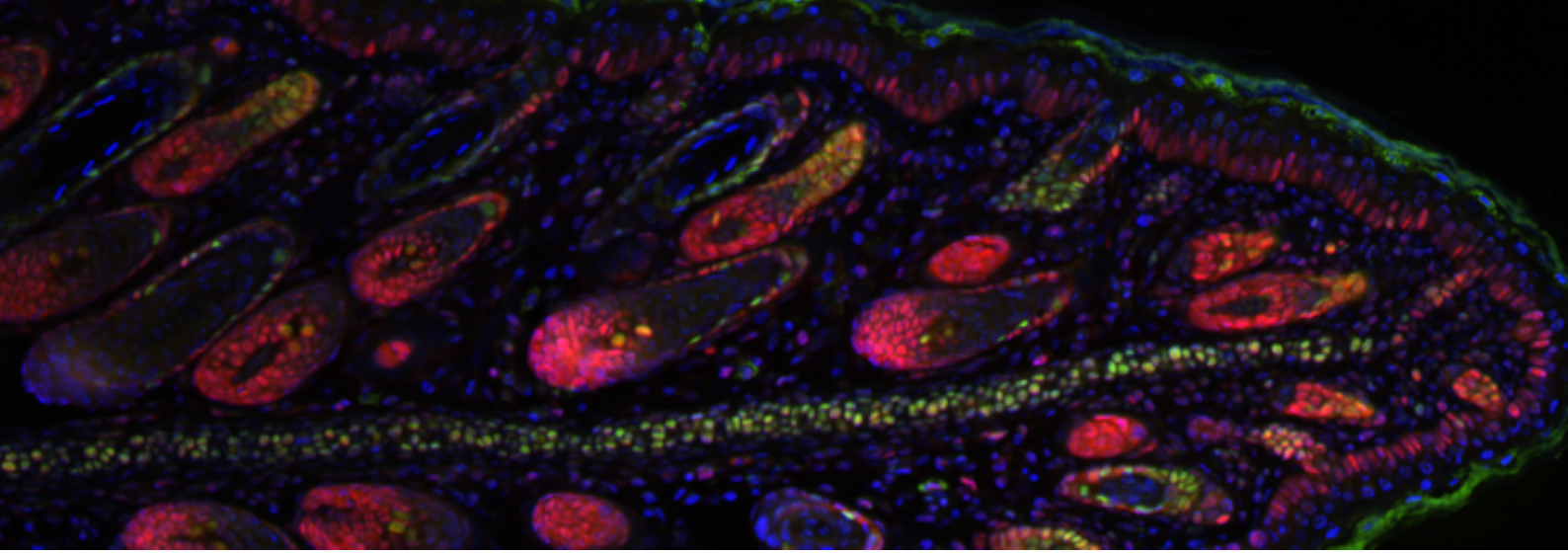
MICHAEL SHELLEY

Many fundamental phenomena in eukaryotic cells — nuclear migration, spindle positioning, chromosome segregation — involve the interaction of (often transitory) cytoskeletal elements with boundaries and fluids. Understanding the consequences of these interactions require specialized numerical methods for their large-scale simulation, as well as mathematical modeling and analysis. In this context, I will discuss the recent interactions of mathematical modeling and large-scale, detailed simulations with experimental measurements and perturbations of activity-driven biomechanical processes within the cell.

Harnessing transcriptional fluctuations for cell fate control and therapy

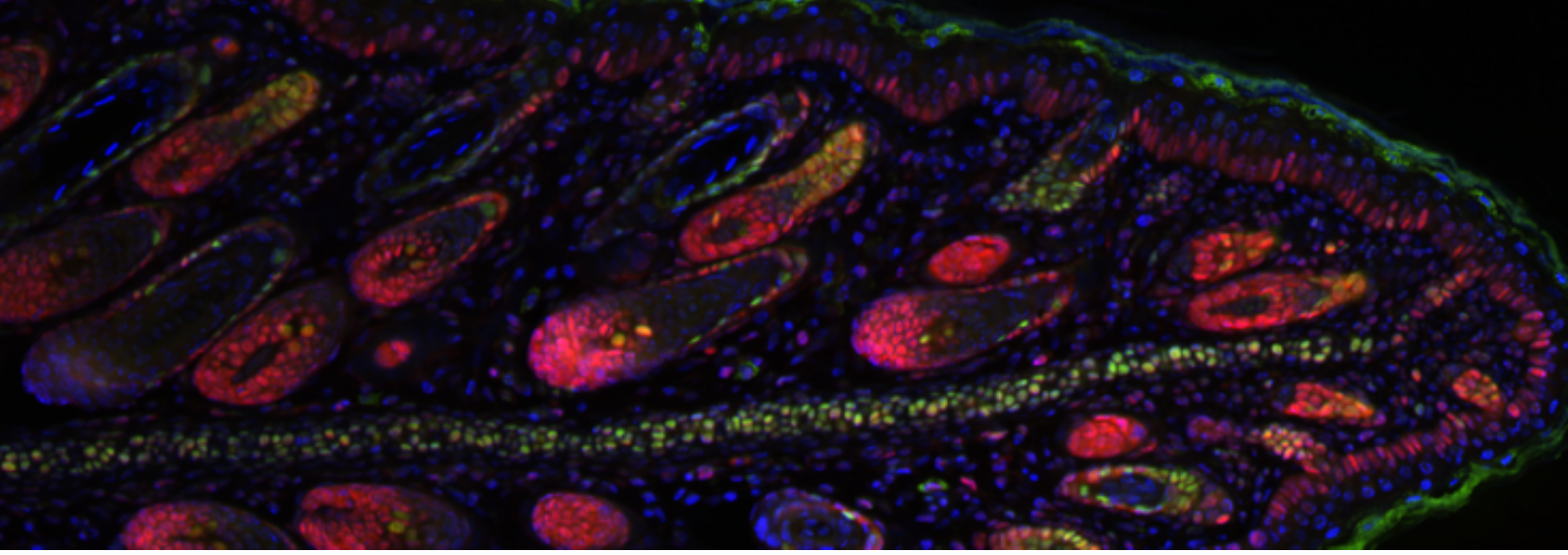
LEOR WEINBERGER

TBD



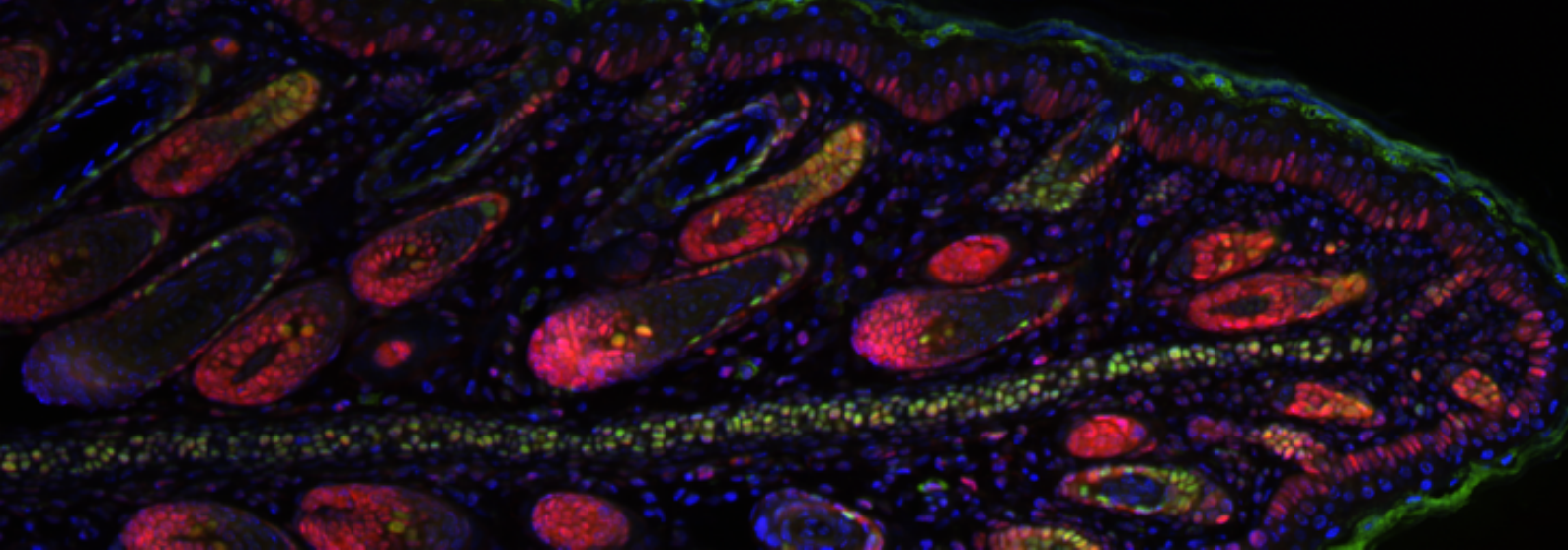
LIGHTNING TALK PRESENTERS

Mikahl Banwarth-Kuhn, University of California, Riverside
Heyrim Cho, University of Maryland
Siddharth Dey, University of California, Santa Barbara
Alvaro Fletcher, University of California, Irvine
Ben Fogelson, University of Utah
Maike Hansen, Gladstone Institutes, University of California, San Fransisco
Núria Folguera-Blasco, Centre de Recerca Matemàtica/Francis Crick
Wei Li, University of California, Los Angeles
Theresa Loveless, University of California, Irvine
Evan Maltz, University of California, Los Angeles
Hamid Mirzaei, UT Southwestern
Simon Mitchell, University of California, Los Angeles
Kitt Paraiso,University of California, Irvine
Melanie Worley, University of California, Berkeley
Jingyu Zhang, University of Pittsburgh



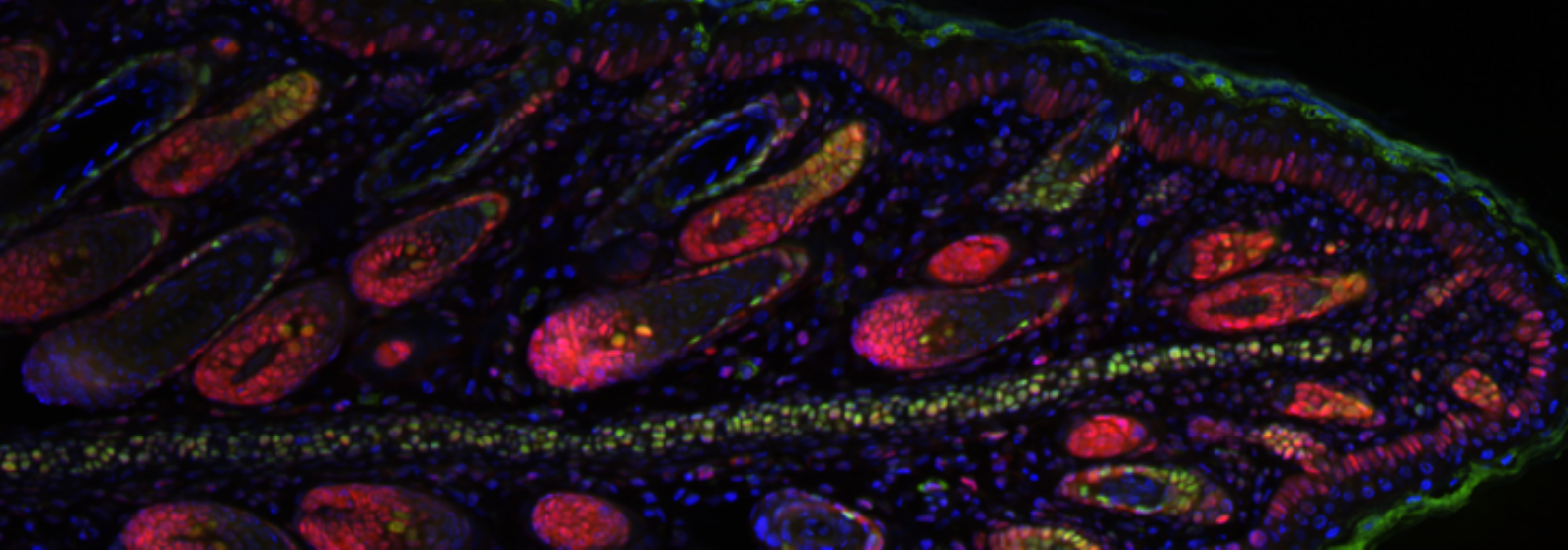
POSTERS PRESENTERS

Emerson Arehart, University of Utah
Emmanuel Asante-Asamani, Hunter College
Mikahl Banwarth- Kuhn, University of California, Riverside
Matt Bovyn, University of California, Irvine
Courtney Carlson, University of California, Irvine
Stephenson Chea, University of California, Irvine
Anush Chiappino-Pepe, École Polytechnique Fédérale de Lausanne (EPFL)
Heyrim Cho, University of Maryland
Karen Chung, Northwood High School
Siddharth Dey, University of California, Santa Barbara
Lucy Dolmadjian, University of California, Irvine
Alvaro Fletcher, University of California, Irvine
Ben Fogelson, University of Utah
Núria Folguera-Blasco, Centre de Recerca Matemàtica/Francis Crick Institute
Lianna Fung, University of California, Irvine
Cameron Gallivan, University of California, Irvine
Daniel Haensel, University of California, Irvine
Lifeng Han, Arizona State University
Maike Hansen ,Gladstone Institutes (UCSF)
Zachary Hemminger, University of California, Los Angeles
Shan Mandy Jiang, University of California, Irvine
Kevin Johnston, University of California, Irvine
Jinsu Kim, University of California, Irvine



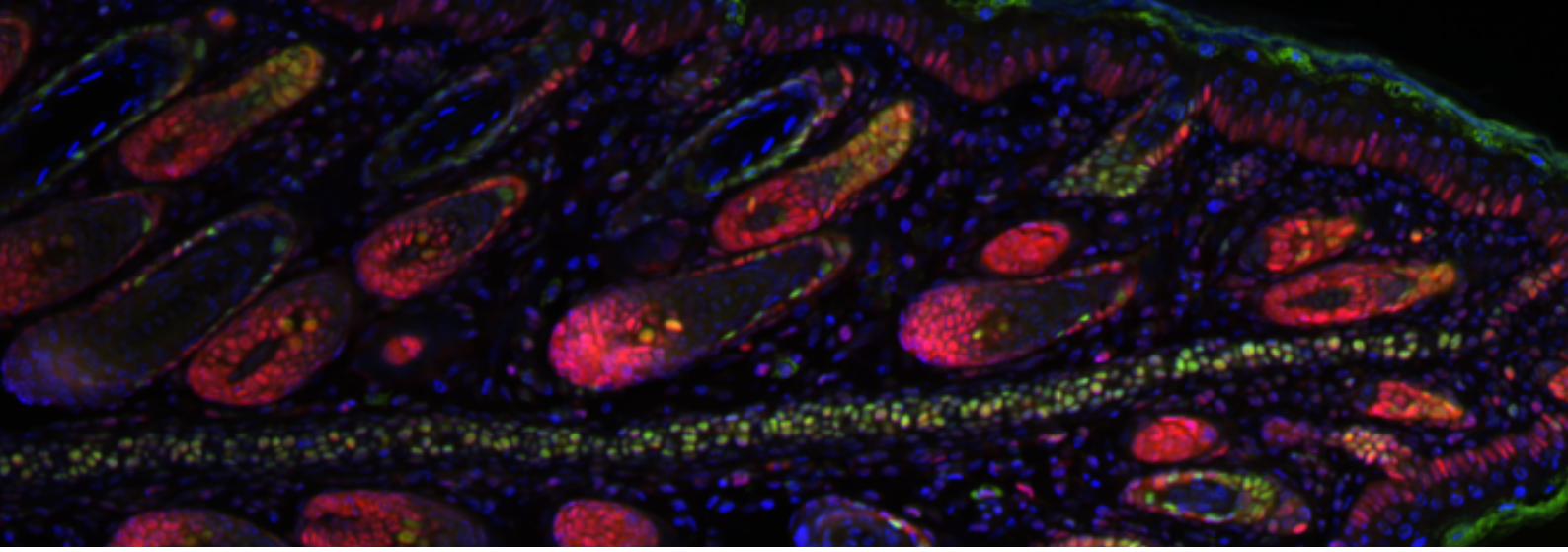
POSTERS PRESENTERS

Marek Kimmel, Rice University
Ryan Lannan, University of California, Los Angeles
Wei Li, University of California, Los Angeles
Lily Li, University of California, Irvine
Guohao Liang, University of California, Irvine
Pei Liu, Pennsylvania State University
Shuang Liu, Interdisciplinary Mathematics Institute
Theresa Loveless, University of California, Irvine
Anna Luzzi, Los Angeles Biomedical Research Institute
Evan Maltz, University of California, Los Angeles
Hamid Mirzaei, UT Southwestern
Simon Mitchell, University of California, Los Angeles
Julien Morival, University of California, Irvine
Maeve Nagle, University of California, Los Angeles
Kitt Paraiso, University of California, Irvine
Elizabeth Read, University of California, Irvine
Mason Schechter, University of California, Irvine
Steven Sera, University of California, Riverside
Gessner Soto, University of Colorado
Yidan Sun, University of California, Los Angeles
Ying Tang, University of California, Los Angeles
David Tatarakis, University of California, Irvine
Robert Taylor, University of California, Irvine



POSTERS PRESENTERS

Xiaojun Tian, Arizona State University
Noam Vardi, University of California, San Francisco
Xiaojie Wang, University of California, Los Angeles
Shuxiong Wang, University of California, Irvine
Killian Wood, California State University, Fullerton
Melanie Worley, University of California, Berkeley
Bin Xu, University of Notre Dame
Zi Ye, University of Minnesota
Jin Yu, Beijing Computational Science Research Center
Lihua Zhang, University of California, Los Angeles
Jingyu Zhang, University of Pittsburgh
Thanutra Zhang ,University of California, Los Angeles
Yangyang Wang, University of California, Irvine
Zixuan Cang, University of California, Irvine



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