

Speaker Bios

Workshop on Understanding Plasticity and its Role Across the Cancer Continuum

Jason Mills, M.D., Ph.D.

Professor | PennState College of Medicine



I am a human pathologist and scientist focusing on cell and developmental biology, as well as bioinformatics. My research focuses primarily on the GI tract, and how stem cells are recruited after injury. In particular we are interested in plasticity: how cells in tissue adapt to various stressors that induce a change in functional identity. For example, my group has been fascinated by how mature cells can rearrange to become proliferative, regenerating cells. We have shown that cells have a universal program, like those for division and death, to do this. This program, paligenosis, helps fuel tissue repair but also allows old cells to proliferate, which increases risk for mutation accumulation and development of chronic, pre-cancerous conditions (like metaplasia) and, eventually, tumorigenesis. I have also led several translational efforts, including clinical trials, to use my lab's study of paligenosis to better prevent or treat tumors.

Cory Abate-Shen, Ph.D.

Professor | Columbia University



Dr. Cory Abate-Shen began her independent career in 1991 at the Center for Advanced Biotechnology and Medicine at UMDNJ-Robert Wood Johnson Medical School (now Rutgers Medical School), where she rose rapidly through the ranks to Professor and founding Chief of the Division of Research in the Department of Medicine. In 1999, she founded the Prostate Program at The Cancer Institute of New Jersey. In 2007, she was recruited to Columbia University Vagelos College of Physicians and Surgeons as the Michael and Stella Chernow Professor of Urological Oncology at the Herbert Irving Comprehensive Cancer Center, with appointments across multiple departments. She served as Associate Director and twice as Interim Director of the cancer center, and from 2019-2024 was Chair of Molecular Pharmacology and Therapeutics, becoming the first woman to chair a basic science department at VP&S. She is currently the Robert Sonneborn Professor of Medicine. An internationally recognized leader in genitourinary malignancies, Dr. Abate-Shen pioneered genetically engineered mouse models of prostate and bladder cancer, leading to advances in cancer biomarkers, prevention, and therapy. Her honors include the AACR Charlotte Friend Award, the Break Through Cancer David M. Livingston Collaboration Award, election to the National Academy of Sciences, and fellowship in both AAAS and the AACR Academy.

Ben Stanger, M.D., Ph.D.

Professor | University of Pennsylvania



After receiving his S.B. degree from MIT and a combined MD and PhD from Harvard Medical School, Dr. Stanger completed a residency in Internal Medicine at UCSF, a clinical fellowship in Gastroenterology at Massachusetts General Hospital, and a research fellowship in Molecular Biology at Harvard. In 2006, he joined the Penn faculty as a member of the Gastroenterology Division. Dr. Stanger's laboratory investigates multiple aspects of pancreatic cancer biology, including metastasis, tumor immunology, metabolism, and therapy resistance. His research integrates mouse models and human data to identify new vulnerabilities that can be assessed clinically. With over 200 research publications, Dr. Stanger is also the author of *From One Cell*, a popular science book about embryonic development. He is currently the Hanna Wise Professor in Cancer Research at the University of Pennsylvania, where he serves as Director of the Pancreatic Cancer Research Center (PCRC).

Nabeel Bardeesy, Ph.D.

Professor | Massachusetts General Hospital, Harvard Medical School



Nabeel Bardeesy, PhD, is the Gallagher Chair in Gastrointestinal Cancer Research at Massachusetts General Hospital and Professor at Harvard Medical School. His research focuses on the molecular mechanisms that drive pancreatic and hepatobiliary cancers, with particular emphasis on how oncogenic signaling and metabolic reprogramming shape tumor cell state, therapeutic response, and resistance. His laboratory has studied cancers driven by FGFR2 rearrangements, mutant IDH1, STK11 loss, and GNAS/PKA pathway activation, defining how these alterations remodel differentiation, metabolism, stress-response programs, and interactions with the tumor microenvironment. A major goal is to identify the tumor dependencies created by these altered states and determine how they can be exploited therapeutically. This work integrates genetically engineered and patient-derived models with analyses of human tumor samples and specimens from clinical trials.

Howard Crawford, Ph.D.

Professor | Henry Ford Health



The Crawford laboratory studies the cell plasticity in pancreatic ductal adenocarcinoma - from initiation to metastasis. We have explored the process of acinar cell transdifferentiation to form the neoplastic precursor, the metaplastic duct and the cellular heterogeneity within these lesions. With this heterogeneity being maintained in pancreatic intraepithelial neoplasia (PanINs), marked collectively by gastric cell identities (neck, pit, tuft and enteroendocrine cells), we now use mouse models to define the functions of each cell type, especially in the establishment of the immune suppressive microenvironment that is maintained throughout the transition to invasive carcinoma. Cellular plasticity remains a hallmark of the cancerous epithelia with the establishment of classical, basal and intermediate molecular subtypes capable of adapting to environmental challenges, including most therapies. We are actively identifying the functions and vulnerabilities of these pancreatic cancer subtypes.

Kathleen DelGiorno, Ph.D.

Assistant Professor | Vanderbilt University



The DelGiorno laboratory works to understand epithelial plasticity in the pancreas in response to chronic injury or oncogenic mutation and how this contributes to tissue damage, repair, and tumorigenesis. Pancreatic acinar cells harbor an amazing amount of plasticity and possess the ability to de- and trans-differentiate to manifold cell types characteristic of other gastrointestinal organs but not typically found in the pancreas. Among these de novo populations are chemosensory tuft cells, which we have found inhibit tissue injury and tumor formation, identifying an inherent mechanism by which epithelial plasticity inhibits cancer development. By combining mouse models and patient samples with histopathology, RNA-sequencing approaches, and ultra microscopy we have identified a concerted program of plasticity resulting in distinct populations with the ability to orchestrate stromal deposition, immune infiltration, and disease progression. Our major goals are to target those processes that drive disease or to co-opt beneficial pathways for patient benefit.

Andrew Ewald, Ph.D.

Professor | Johns Hopkins School of Medicine



Dr. Andrew Ewald seeks to identify the cellular programs and molecular toolkits used by cancer cells as they spread through the body during metastasis. To achieve this goal, he nucleated and leads teams of engineers, molecular and cell biologists, and physicians to identify the most clinically important steps in cancer progression and to develop new tools to identify targets for novel anti-cancer therapies. The key insight was to bring together live culture of tumors with cutting edge imaging and genetic techniques to study cancer invasion and metastasis in real-time. His lab is recognized internationally for having: discovered several conserved molecular programs used by breast cancer cells during metastasis, demonstrated that groups of breast cancer cells collectively metastasize, revealing molecular regulators of cancer cell plasticity, elucidating molecular mechanisms of cancer cell survival during metastasis, and developing new approaches to identify anti-metastatic therapeutic targets.

Karuna Ganesh, M.D., Ph.D.



Associate Member | Memorial Sloan Kettering Cancer Center

Dr. Karuna Ganesh's research focuses on understanding the cellular and molecular mechanisms that drive cancer metastasis, with a particular emphasis on metastatic stem cells (MetSCs), regenerative plasticity, and therapy resistance. Her laboratory integrates patient-derived organoid models, genetically engineered mouse models, transcriptomic, epigenomic, and single-cell approaches to define how tumor-intrinsic and microenvironmental factors regulate metastatic progression. The lab is particularly interested in elucidating how phenotypic plasticity enables metastatic dissemination, identifying the roles of stromal and immune cells in shaping metastatic stem cell behavior, and discovering signaling pathways that can be therapeutically targeted to prevent or treat metastatic disease. Through close collaboration with clinicians, the Ganesh Lab seeks to translate these discoveries into novel therapeutic strategies that improve outcomes for patients with advanced colorectal and other gastrointestinal cancers.

David Goodrich, Ph.D.



Professor | Roswell Park Comprehensive Cancer Center

The long-term goal of the Goodrich lab is to understand how alterations in key tumor suppressor genes like RB1, TP53, and PTEN drive cancer progression. These tumor suppressors are among the most frequently altered genes in human cancer, particularly in advanced metastatic and therapeutically resistant cancer. Understanding how they function at the molecular level will both refine our fundamental understanding of cancer biology and inspire novel therapeutic approaches to treat it. A major focus of current work is understanding how these genes impact prostate and lung cancer phenotypic plasticity. As we understand cancer phenotypic plasticity better, we explore possible therapeutic approaches that could be used to suppress it and thereby improve therapeutic outcomes for cancer patients.

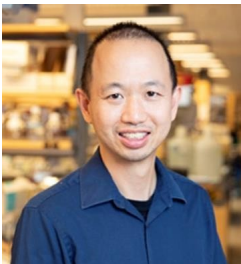
Ophir Klein, M.D., Ph.D.



Professor | Cedars-Sinai Health Sciences University

Ophir Klein serves as the inaugural Executive Director of Cedars-Sinai Guerin Children's and Executive Vice Dean for Children's Services, and he is the David and Meredith Kaplan Distinguished Chair in Children's Health. He is a global leader whose team has made seminal contributions to understanding the development of normal and diseased organs, as well as elucidating mechanisms underlying the pathogenesis of complex inherited disorders, many of which manifest during early neonatal and childhood life. He has led the establishment of Guerin Children's as a respected leader in pediatric scholarly research, training, and specialty clinical services. Dr. Klein has received multiple honors, including election to the National Academy of Medicine and the American Association of Physicians. He has had multiple advisory roles to the NIH and led professional societies. Dr. Klein was educated at the University of California, Berkeley, where he earned a B.A. in Spanish Literature. He subsequently attended Yale University School of Medicine, where he received a Ph.D. in Genetics and an M.D. He then completed residencies at Yale-New Haven Hospital in Pediatrics and at UCSF in Clinical Genetics. Prior to his Cedars-Sinai appointment, Dr. Klein served as Chief of Medical Genetics and Craniofacial Anomalies and Director of the Institute for Human Genetics at UCSF.

Ken Lau, Ph.D.



Professor | Vanderbilt University

Dr. Lau is internationally recognized in field of single-cell systems biology. His research focuses on gut biology, specifically the molecular events that occur at the intersection of mucosal damage, inflammation, and tumor initiation. His laboratory applies systems biology approaches to investigating interactions between cells and their microenvironment in modulating cellular identity, development, and the origins of cancer. Serving as co-chair of the NCI Human Tumor Atlas Network, he has led efforts to map human cancers at single-cell resolution, setting new standards for integrative cancer research. He is currently a full professor in the Department of Cell and Developmental Biology at Vanderbilt University, and the director of the Center for Computational Systems Biology.

Anirban Maitra, M.D.

Professor | NYU Langone Health



The overarching goal of the Maitra laboratory at Perlmutter Cancer Center is to improve the survival of patients with pancreatic cancer through earlier detection, cancer interception and rational therapies. For nearly 25 years, the Maitra laboratory has been singularly dedicated to the cause of researching pancreatic cancer, one of the most lethal cancers known to mankind, with a clear line of sight to translational advances that can improve patient outcomes in the clinic and favorably impact those at high risk of the disease. Their research has been consistently supported by the National Cancer Institute (National Institutes of Health), as well as foundations such as the Pancreatic Cancer Action Network. Through this journey, the Maitra laboratory has collaborated with many national and international colleagues, emblematic of the team science approach that we are convinced is required for success against pancreatic cancer, and further, had the great fortune of mentoring numerous exceptional trainees, augmenting the next generation of cancer researchers.

Katerina Politi, Ph.D.

Professor | Yale School of Medicine



My laboratory is focused on studying mechanisms of lung tumorigenesis, understanding fundamental processes that underlie sensitivity and resistance to new and established anti-cancer therapies and identifying new therapeutic approaches in lung cancer. We also have long-standing experience using mouse and patient-derived models for mechanistic and therapeutic studies. One area of intense investigation is in understanding the biology and therapeutic vulnerabilities of oncogene-driven lung cancers like those harboring mutations in EGFR. In this regard, we have extensively studied the phenotype of mice that express lung-adenocarcinoma associated Epidermal Growth Factor Receptor (EGFR) mutants to address clinically relevant problems such as resistance to targeted therapies and tumor progression. Moreover, my lab studies the signaling pathways and genes that cooperate with mutant EGFR and other oncogenes to transduce oncogenic signals within the cell.

Julien Sage, Ph.D.

Professor | Stanford University



Dr. Sage's work has focused on the RB tumor suppressor pathway and how inactivation of RB promotes tumorigenesis in children and adults. Dr. Sage became initially interested in small cell lung cancer (SCLC) because of the nearly ubiquitous loss of RB in this cancer type and the intriguing relationship in mice and humans between loss of RB and the growth of neuroendocrine lesions. In the past few years, the Sage lab has developed pre-clinical models for SCLC (and other neuroendocrine tumors), including genetically engineered mouse models. His lab has used these models to investigate signaling pathways and cell-cell interactions driving the growth and metastatic ability of SCLC and to identify novel therapeutic targets in this recalcitrant cancer. A major focus of the lab has been on defining genetic and epigenetic heterogeneity in SCLC and investigating the mechanisms underlying plasticity.

Charles Sawyers, M.D.

Professor | Memorial Sloan Kettering Cancer Center



Charles L. Sawyers is a Howard Hughes Medical Institute Investigator and has served as Chair of the Human Oncology and Pathogenesis Program at Memorial Sloan Kettering Cancer Center since 2006, where he studies mechanisms of cancer drug resistance with an eye toward developing novel therapies. His group currently focuses on understanding how lineage plasticity develops as a mechanism of resistance to androgen receptor signaling inhibitors. Examples of recent work include: (i) defining a role for the tumor microenvironment (extracellular matrix), acting through the YAP/TEAD pathway, in restraining the transition of prostate adenocarcinoma (PRAD) to neuroendocrine prostate cancer (NEPC) (PMID: 41509338) and (ii) identifying MYC pathway reprogramming as a critical feature of the NEPC transition, partnering with TIP60 as a coactivator to drive NEPC lineage proliferation (PMID: 42146329).



Eric Snyder, M.D., Ph.D.

Professor | University of Utah Health Academic Medical Center

The molecular networks that specify cellular identity and suppress alternative cell fates are tightly regulated during normal tissue homeostasis. The genetic and epigenetic changes that accumulate during cancer progression can disrupt these networks, often with lethal consequences for cancer patients. In many types of cancer, cell identity dictates not only intrinsic malignant potential, but also response to therapy, even in tumors harboring the same targetable mutations. Although tissue of origin is a major determinant of cancer cell identity, cancer cells can also undergo lineage switching in the course of their natural history and in response to the selective pressure of targeted therapy. Our overall goal is to determine how the loss of cellular identity and acquisition of alternative differentiation states contributes to cancer progression and alters therapeutic response.



Sydney Shaffer, M.D., Ph.D.

Assistant Professor | University of Pennsylvania

My lab studies how cancer cells change state over time, and how those dynamics shape both the origins of cancer and its response to therapy. Tumor cells move between transcriptional states that are not encoded in their genomes, and a central question is how long such a state persists. States that last only moments behave very differently from states that a cell passes down to its descendants, because only the latter can be acted on by selection. We develop lineage tracing methods that follow single cells and their progeny over time, and read out cell state and clonal identity together, so that we can watch plasticity unfold rather than infer it from a snapshot. We apply this in precancerous tissue, asking how plastic cell states contribute to the earliest steps of transformation, and in treated tumors, asking how they allow rare cells to survive therapy.



Michael Shen, Ph.D.

Professor | Columbia University

For the past thirty-two years, my laboratory has investigated fundamental mechanisms of mammalian development and cancer using in vivo analyses of genetically-engineered mice as well as human organoid and xenograft models. In recent years, my lab has been focused on analyses of lineage fidelity in development and lineage infidelity in cancer, focusing on the prostate and bladder. In our studies of genitourinary cancers, we have been particularly interested in epigenomic mechanisms that regulate lineage plasticity in the context of tumor initiation, tumor evolution, progression to advanced disease, and emergence of drug resistance. Ongoing projects include analyses of the epigenetic regulation of lineage plasticity, as well as of molecular pathways governing prostate regression and cell death following androgen-deprivation, and the role of the tumor microenvironment in modulating treatment response.



Andrea Viale, M.D.

Associate Professor | UT MD Anderson Cancer Center

Dr. Viale earned his medical degree from the University of Turin and trained as a clinical oncologist before pursuing a full-time career in cancer research. Following postdoctoral training at the European Institute of Oncology and Dana-Farber Cancer Institute, he established his independent laboratory at The University of Texas MD Anderson Cancer Center, where he is an Associate Professor in the Department of Genomic Medicine and a faculty member of the Graduate School of Biomedical Sciences. Dr. Viale leads a multidisciplinary research program centered on pancreatic cancer, with major interests in tumor functional heterogeneity, clonal evolution, and mechanisms of early tumor progression. By integrating genome editing and engineering, molecular barcoding and lineage tracing, high-content and spatial imaging, and next-generation sequencing with clinically relevant models of human disease, his group tackles ambitious biological questions to uncover new mechanisms of cancer initiation, evolution, and therapeutic response.

Kwok-Kin Wong, M.D., Ph.D.



Professor | NYU Langone Health

Dr. Kwok-Kin Wong, professor of medicine at New York University Langone Health, has authored more than 400 peer reviewed publications and is a member of The American Society for Clinical Investigation/The American Association of Physicians (ASCI/AAP). Dr. Wong's research, teaching and clinical activities focus on understanding the genetic alterations driving lung tumor formation and the testing of new targeted and immune lung cancer therapies. His laboratory integrates human cancer genomic studies with generation of novel preclinical cancer models to investigate the efficacy of new targeted therapeutics and immunotherapeutics. These studies will help uncover the molecular mechanisms and genetic underpinnings of sensitivity and resistance to targeted therapies and immunotherapy. Insights into these processes gained by Dr. Wong and his team continue to lead us closer to curative treatments for patients with advanced lung cancer.

Jing Yang, Ph.D.



Professor | University of California San Diego

Dr. Jing Yang's research focuses on understanding the molecular basis of tumor metastasis. Her pioneering work demonstrated a critical role for the Epithelial-Mesenchymal Transition (EMT) program in metastasis, helping bring EMT to the forefront of cancer research. Her lab currently investigates two major directions: 1. Regulation of Epithelial-Mesenchymal Plasticity in Metastasis. Her group aims to uncover novel extracellular and intracellular mechanisms that control EMT dynamics during tumor invasion and metastasis. They identified extracellular matrix stiffening and loss of epithelial polarity as critical drivers of EMT, and they continue to dissect the underlying molecular mechanisms. 2. Regulation of Metastasis Dormancy. Her group generated inducible EMT mouse models to demonstrate that reversible EMT (epithelial-mesenchymal plasticity) is required for successful tumor progression and metastasis. Currently, her lab utilizes these mouse models, along with human breast cancer cell lines and patient-derived xenograft (PDX) models, to explore how EMT plasticity regulates metastatic dormancy and chemoresistance.