CANCER GRAND CHALLENGES

Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by:

Dr. Wenwei Hu

Leukemia Inhibiting Factor (LIF), its role in tumorigenesis and Cancer Cachexia

Abstract: Cancer cachexia, a multifactorial disease characterized by weight loss, muscle wasting, and fat browning, is challenging due to the complexity of its metabolic disorder and the lack of effective therapies, calling for a better understanding of its underlying mechanisms. Leukemia inhibitory factor (LIF), a multi-functional cytokine, has been suggested to be a cachexia-inducing factor. We established a cachexia mouse model with conditionally inducible LIF expression that can help us to study cachexia and the contribution of LIF to it.

Date: Friday, December 16, 2022

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting:
https://rutgers.zoom.us/j/95437261171?pwd=V2pGam92TDRVVC9YQkJON29vdTBqUT09

Dr. Hu is a Professor in the Department of Radiation Oncology at the Rutgers Cancer Institute of New Jersey at Rutgers University. She received her PhD from Zhejiang University School of Medicine for research on mutagenesis induced by chemical carcinogen. Dr. Hu completed postdoctoral training in NYU Medical School focusing on DNA damage and repair before she moved to the University of Medicine and Dentistry of New Jersey continuing her postdoctoral training with Dr. Arnold Levine studying p53 and its signaling pathway. Since 2009, Dr. Hu has been a faculty member at Rutgers Cancer Institute of New Jersey. A major research interest of Dr. Hu's group is to study the function and regulation of tumor suppressor p53, which in turn impacts tumorigenesis. Her group's work also made important contributions toward understanding the mechanisms of mutant p53 accumulation and gain of oncogenic activity in tumors. In addition, Dr. Hu studies the function of LIF, a cytokine that is a p53 target in tumorigenesis.

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Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by:

Professor Sir Stephen O'Rahilly

Causes and consequences of obesity; some mechanistic insights from human genetics

Abstract: Two of the key questions we address in my lab are 1) Why are some people obese while others in similar environments remain lean and 2) Why does obesity lead to adverse health outcomes? In this talk, I will share some of our studies which I hope to have provided some novel insights into these questions. Our work has often started with detection of causative genetic defects in humans with rare extreme phenotypes and established mechanism through studies in cells, animal models and larger human populations.

Date: Friday, January 13, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/98243545222?pwd=OTZoSnNjZHZCamtEQytXQTNYNFk4dz09

Professor Sir Stephen O'Rahilly graduated in Medicine from University College, Dublin in 1982. From 1982-1991, he undertook postgraduate training in general medicine and endocrinology and in diabetes research in London, Oxford and Harvard. In 1991, he established his own laboratory in the University of Cambridge at Addenbrooke's Hospital where he was a Welcome Trust Senior Fellow in Clinical Science. In 1996, he was appointed to a newly created Chair of Metabolic Medicine. In 2002, he was appointed to the Chair of Clinical Biochemistry and Medicine at the University of Cambridge. He has a long-standing interest in the etiology and pathophysiology of human metabolic and endocrine disease. His scientific focus has been to improve diagnosis, prognostication, therapy, and prevention of metabolic and endocrine disease. He has won many awards for his work including the Society for Endocrinology Medal, the European Journal of Endocrinology Prize, the Novartis International Award for Clinical Research in Diabetes, the Heinrich Wieland Prize, the Rolf Luft Award, the Feldberg Prize, the Society for Endocrinology Dale Medal, and the InBev Baillet-Latour Prize for Health. He was elected to the Academy of Medical Sciences in 2000, to the Royal Society in 2003, and to the US National Academy of Sciences as a Foreign Associate in 2011. O'Rahilly was knighted in the 2013 Birthday Honors for services to medical research. In 2017, he became a member of the Royal Irish Academy and in 2019, he was elected as an Honorary Fellow of the Learned Society of Wales.

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The Cancer Cachexia Action Network presents a seminar by:

Dr. Thales Papagiannakopoulos

Investigating cancer-associated cachexia in genetic subtypes of lung cancer

Abstract: Treating KRAS-mutant lung adenocarcinoma remains a major challenge for clinical oncology because patients are refractory to standard-of-care and a large number of patients display symptoms of cancer-associated cachexia. Our group is investigating the development of cachexia in common genetic subtypes of KRAS-mutant lung adenocarcinoma and assessing how well-defined dietary regimens can promote or suppress cachexia.

Date: Friday, January 20th, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/94871160574?pwd=T0s3VXdNaytjelVtVDlPTUhCOFQ2Zz09

Dr. Thales Papagiannakopoulos is an associate professor and principal investigator at New York University in the Department of Pathology.Dr. Papagiannakopoulos received his Ph.D in 2010 at UC Santa Barabara. Shortly after, he worked as a Postdoctoral Fellow with the Tyler Jacks Laboratory at The Koch Integrative Insitute for Cancer Research in Cambridge, Massachusetts. In 2015 Thales moved on to New York University where he currently resides as head of the Thales Papagiannakopoulos Laboratory. Dr. Papagiannakopoulos has authored and co-authored multiple peer-reviewed scientific papers and presented works at many national and international conferences. Dr. Thales Papagiannakopoulos contributions have acclaimed recognition from honorable subject experts around the world. Dr. Thales Papagiannakopoulos academic career is decorated with several reputed awards and funding. A major focus of his laboratory is CRISPR/Cas9-based in vivo and in vitro approaches to study KRAS-driven lung cancer (the major subtype of lung cancer and one of the most aggressive and lethal solid tumors). Since the establishment of his laboratory in October 2015, his team has made significant progress in applying new approaches to characterize a major genetic subset of lung adenocarcinoma with NRF2/KEAP1 mutations (Ashouri et al., Nat. Comm, 2017; In Press: Romero R et al., Nature Medicine; Sayin VI et al., eLife).

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Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by:

Dr. Ayelet Erez

Targeting the tumor MACROenvironment to improve Cancer Cachexia (CAC) diagnosis and therapy

Abstract: Cancer induces metabolic reprogramming within the tumor, its microenvironment, and its host, reaching the extreme with cancer-associated cachexia (CAC) at the end stage. While significant efforts invested in translating metabolic changes in the tumor and its microenvironment led to improved cancer patient care, it did not yield efficient biomarkers or treatment modalities for CAC.

Date: Friday, February 10, 2023

Time: 9:00 a.m.-11:00 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/99173844540?pwd=aC92eHpuYWlrQ2lublhyWlFvdWtaQT09

Dr.Erez earned a BSc and MD, cum laude, at the Technion-Israel Institute of Technology (1991 and 1994), followed by a year of rotating internship at the HaEmek Hospital, in Afula. She served as a pediatric resident at the Safra Children's Hospital in the Sheba Medical Center in Tel Aviv between 1995 and 2000 and earned a PhD in cancer genetics from the Tel Aviv University in 2005. She completed an American Board of Medical Genetics Clinical Genetics residency program together with a postdoctoral fellowship at Baylor College of Medicine in Houston, Texas in 2008. She then worked as an assistant professor of Molecular and Human Genetics at Baylor College of Medicine and as a medical geneticist at Texas Children's Hospital. She joined the Weizmann Institute's Department of Biological Regulation in 2012. The focus of her research is to understand the metabolic mechanism of the disease and to identify the differences between the healthy and diseased cell. Understanding these mechanisms can offer a means to improve the ability to diagnose and treat these diseases. Ayelet's laboratory has identified changes in the body fluids of cancer patients which may be utilized to detect and monitor disease progression. Her basic research is now delving into the complex metabolic pathways that integrate amino acid/nitrogen metabolism and glucose/oxidative stress. This includes searching for novel genes involved in cancer metabolism. She is in the process of establishing a pediatric cancer genetic clinic in Israel to nurture the bridge from scientific discoveries to new treatments, and to use clinical experience to guide scientists.

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Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by:

Matthew Vander Heiden

Relationship between muscle wasting and pancreatic cancer growth

Abstract: Peripheral tissue wasting is an early event in pancreatic cancer. We have found that pancreatic exocrine insufficiency induces a starvation response that contributes to tissue wasting. Furthermore, tissue wasting can provide amino acids to support pancreatic cancer growth.

Date: Friday, February 17, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/99285156224?pwd=SmloNWpJR3R2R3pBcHNURGllaFlCZz09

Dr. Matthew Vander Heiden is a practicing oncologist and instructor in medicine at Dana-Farber Cancer Institute/Harvard Medical School. He earned his doctoral and medical degrees from the University of Chicago, where he worked in the laboratory of Craig Thompson. Dr. Vander Heiden then completed a residency in internal medicine at Boston's Brigham & Women's Hospital and a hematology-oncology fellowship at Dana-Farber Cancer Institute/Massachusetts General Hospital. In 2010, Dr. Vander Heiden joined the MIT faculty. His work has been recognized by many awards including the Burroughs Wellcome Fund Career Award for Medical Sciences, the AACR Gertrude B. Elion Award, the HHMI Faculty Scholar Award, and an NCI Outstanding Investigator Award. The long-term goal of the Vander Heiden Laboratory is to understand how mammalian cell metabolism is adapted to support cancer initiation and progression. Current research interests of his laboratory include: 1) identifying which metabolic processes create bottlenecks for cell proliferation; 2) determining how metabolism is different in different cancers, examining in detail the influence of tissue type, tumor genetics, and tumor microenvironment; and 3) understanding how diet and whole-body metabolism influence cell metabolism in tissues to modify cancer and other disease phenotypes. Together, these studies will broaden the understanding of cancer cell metabolism and identify approaches to target metabolism for cancer therapy.

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The Cancer Cachexia Action Network presents a seminar by:

Dr. Daniel Marks

HYPOTHALAMIC MECHANISMS OF CACHEXIA

Abstract: ***Cachexia is common in cancer patients and has a devastating impact upon physical function, quality of life, and survival. We, along with others, demonstrated that signaling between tumor and the central nervous system (CNS) is critical for the metabolic, behavioral, and neuroendocrine dysfunction observed during tumor growth. We determined that the mediobasal hypothalamus (MBH) is uniquely equipped as both a sensor and amplifier of peripheral inflammatory signaling. This region has an attenuated, dynamic blood brain barrier and contains specialized cells that regulate appetite, endocrine function, and the autonomic nervous system. This talk will describe the role of persistent activation of MBH neurons regulating autonomic tone and neuroendocrine stress responses in the evolution of cancer cachexia.***

Date: March 24, 2023

Time: 8:00 a.m.-10:00 a.m.(ET)

Join Zoom :

https://rutgers.zoom.us/j/97572861182?

pwd=aTdSL3gyZTdkUVZRckU3alVlbzhiZz09&from=addon

Daniel L. Marks M.D., Ph.D. received medical and graduate training at the University of Washington, then completed his pediatric residency at the University of Utah, and a fellowship in pediatric endocrinology at OHSU. He is currently Senior Associate Dean for Research, Professor in Pediatric Endocrinology, Ray Hickey Chair for Pediatric Research, Director of the Papé Family Pediatric Research Institute, and Associate Director of the OHSU MD PhD program. His work is focused on the neuroendocrine control of body weight. He has a particular interest in pediatric weight regulation, including obesity, failure to thrive, and disease-associated cachexia. Dr. Marks also served as a Senior Scientific Advisor for the Bill & Melinda Gates Foundation.

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The Cancer Cachexia Action Network presents a seminar by:

Associate Professor Barry Laird

Cancer Cachexia – from a nutritional to an inflammatory condition

Abstract: ***The systemic inflammatory response (SIR) resulting from the host-tumor interface is recognized as a central tenet of cancer, following its established role in non-malignant disease. Using the Eastern Cooperative Oncology Group – Performance Status (ECOG-PS) and modified Glasgow Prognostic Score (mGPS) as a framework, we have systematically examined key aspects of cachexia including frailty, lean mass, measures of appetite and weight, to confirm that in cachexia, function and inflammation are the central pillars. Our work to date supports the increasing recognition and change of cachexia from a nutritional to an inflammatory condition.***

Date: Friday, April 21, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/98151704115?pwd=bEpKTENnMTVGZ0pqY2lyNjBTa3JUUT09

Barry is currently an academic clinican that specializes in palliative medicine. He is passionate about improving symptoms in people with life limiting illness. His focus in research is understanding how the tumor-host interaction in cancer is implicated in the genesis of symptoms. He is part of the Edinburgh Palliative and Supportive Care Group (EPaS – Group Lead Professor Marie Fallon) at the Institute of Genetics and Molecular Medicine. Barry holds consultant positions in Palliative Medicine at the Edinburgh Cancer Centre and St Columba's Hospice. His work has demonstrated that this inflammatory response influences survival and also quality of life in patients with advanced cancer. This understanding provides valuable insight into the genesis of cachexia, fatigue, pain and reduced physical function which in turn can inform potential novel therapeutic targets to address these. The overarching aim is to improve the care of patients with cancer through improved prognostication, stratification and efficacy of therapies through an evidence based translational research program from basic science to clinical trials. Professor Laird has close collaborations with local clinicians, multidisciplinary academic research groups and industry partners.

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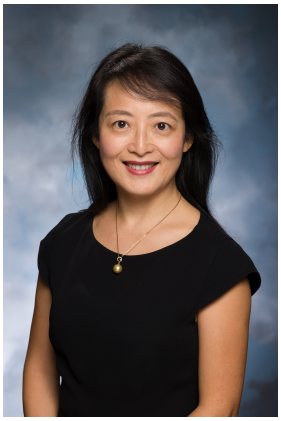
The Cancer Cachexia Action Network presents a seminar by:

Dr. Jessie Guo

G6pd is Essential to Maintain Redox Homeostasis and Biosynthesis for Lkb1-Deficient Kras-Driven Lung Tumor Growth

Abstract: ***Co-mutations of TP 53 and LKB1 represent two different subgroups of KRAS-driven NSCLC, with distinct biological properties, metabolic vulnerabilities, and responses to standard therapies. We found G6PD Glucose-6-Phosphate Dehydrogenase (G6PD), the rate-limiting enzyme of oxidative pentose phosphate pathway, is not essential for KrasG12D/+;p53-/- (KP) KP lung tumorigenesis, but is indispensable for KrasG12D/+;Lkb1-/- (KL) lung tumor growth. G6PD supports KL lung tumorigenesis via maintaining cytosolic NADPH production, which is essential to maintain redox homeostasis and de novo lipogenesis for KL tumor cell proliferation. At late stage of KL lung tumor progression, G6PD loss reprograms NADPH generating metabolic pathway by increasing serine uptake to sustain one carbon metabolism-mediated cytosolic NADPH generation.***



Date: Friday, May 12, 2023

Time: 8:00 a.m.-10:00 a.m.(ET)

Join Zoom Meeting

Dr. Guo is a tenured Associate Professor at Rutgers Cancer Institute of New Jersey. Her research is focused on the field of cancer metabolism in Kras-driven lung tumorigenesis. She has made tremendous contributions to understanding the role and mechanism of autophagy in supporting Kras-driven NSCLC. She demonstrated that both cell autonomous autophagy and host autophagy are essential for Kras-driven lung tumorigenesis. These findings suggest that targeting cancer metabolism by inhibiting autophagy is a valuable strategy to treat lung cancer. Additionally, she is interested to identify other metabolic vulnerabilities that can potentially be targeted for the treatment of KRAS-driven NSCLC.

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The Cancer Cachexia Action Network presents a seminar by:

Dr. Christopher M. Adams

Investigating Mechanisms and Treatment of Skeletal Muscle Atrophy

Abstract: My talk will discuss the research in my laboratory to discover and characterize novel stress-induced signaling mechanisms within skeletal muscle fibers that promote muscle atrophy and weakness during conditions such as aging, starvation, and immobilization. The talk will also discuss the work we are doing to discover and translate novel small molecule approaches for muscle atrophy and weakness.

Date: Friday, June 2, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/92226264544?pwd=dURIODRSZFY4cm5rQkFCOU9mekIzUT09

Dr. Adams completed his MD/Ph.D. in Physiology & Biophysics from the University of Iowa in 1999. Shortly after, Dr. Adams went on to the University of Texas Southwestern Medical Center where he completed his residency and post-doctoral fellowship in Endocrinology and metabolism in 2005. Dr. Adams then moved on to the University of Iowa from 2006-2021, where after 16 years, he became a tenured Professor of Medicine. Currently, Dr. Adams is a Professor of Medicine and Research Chair of the Division of Endocrinology, Diabetes, Metabolism and Nutrition at Mayo Clinic. Dr. Adams is also the founder and CEO of *Emmyon, Inc.*; a biotechnology company focused on small molecules for the prevention and treatment of skeletal muscle atrophy and related metabolic disorders. His clinical practice focuses on the care of patients with diabetes and other endocrine disorders. His research focuses on molecular mechanisms and the treatment of skeletal muscle atrophy and diabetes. Dr. Adams is the lead inventor on over 30 patents concerning small molecules that help maintain the structure and function of skeletal muscle. He leads a biotechnology company that is translating this research in collaboration with global companies and his laboratory at Mayo Clinic.

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The Cancer Cachexia Action Network presents a seminar by:

Assistant *Professor Marcus Seldin*

Leveraging genetic variation to identify modes of inter-organ communication

Abstract: *The research in our laboratory focuses on combining population genetics analyses with targeted experimental models in cells and mice, where we investigate mechanisms of interorgan communication. Specifically, we use genetic variation of "omics" measures (ex. RNA-Sequencing, metabolomics etc.) in mouse and human populations to generate hypotheses for patterns of signaling between metabolic organs. Initially, we observed that both known and new signals between tissues could be identified by surveying simple correlation structure across populations. Since then, we have expanded these models and focused efforts on physiologic dissection of endocrinology using gain- and/or loss-of function approaches in cultured cells and mouse models. This generalized approach is also sufficient to pinpoint context-specific interactions between tissues, such as sex-specific signals from muscle to other tissues. In addition, these analyses have guided discovery of new modes of tissue signaling, such as crosstalk between liver and heart.*

Date: Friday, August 18, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

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The approaches used by our lab to decipher inter-tissue communication have been applied to understand interactions between tumor and surrounding tissue and coordination between pathways which associate with cancer severity. These primarily rely on analysis if inter-individual differences in TCGA.

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The Cancer Cachexia Action Network presents a seminar by:

Dr. Fabio Penna

NAD+ metabolism in cancer- and chemotherapy-induced cachexia

Abstract: ***We recently demonstrated that muscle NAD+ deficiency is a common feature of cancer cachexia (CC), resulting from impaired biosynthesis, as observed in both preclinical CC models and cancer patients (Beltrà et al., Nat Comms 2023). Beyond the peripheral muscle impairments of energy production, also the liver control of systemic energy metabolism may be affected by both tumor and chemotherapy, making the liver a potential target for anti-cachexia interventions. The presentation will summarize the available evidence on liver NAD+ metabolism alterations and impact of NAD+ targeting in CC, and will show a deeper molecular characterization of the impact of NAD+ repletion in preclinical CC models.***

Date: Friday, September 15, 2023

Time: 8:00 a.m.-10:00 a.m. (ET)

Join Zoom Meeting

My scientific path started with the characterization of anabolic and catabolic circulating factors that associate with muscle wasting in several animal models of cancer cachexia, finding reduced IGF-1 and increased myostatin as common signs induced by tumor growth. My research provided the first evidence that tumor growth impairs the myogenic potential and that ERK inhibition rescues the myogenic capacity and partially prevents muscle depletion in tumor hosts. In parallel, I described the specific effects of tumor growth on muscle mitochondria and the ability of training exercise to revert such alterations in order to move the scientific interest from muscle mass to muscle quality, function, and metabolism. On the same line, the current focus of my research has moved from a muscle-centric perspective to a broad host metabolism approach, in order to understand cancer as a systemic host disease.

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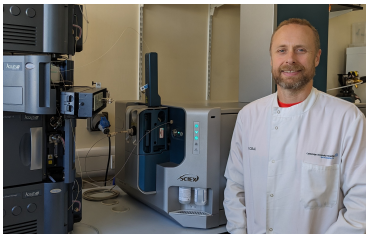
The Cancer Cachexia Action Network presents a seminar by:

Dr. Richard Kay

Plasma peptide LC-MS/MS analysis: how to find needles in haystacks

Abstract: *The analysis of endogenous peptides in plasma (using mass spectrometry) is very challenging due to their low concentration in the presence of a high abundant plasma protein background. Further compounding this issue is the inherent instability of bioactive peptides and their rapid clearance from circulation. We have developed strategies to extract and analyse peptides in plasma, and have applied this workflow to profile metabolically relevant gut peptides in a variety of diseases.*



Date: *Friday, October 20*

Time: *8:00 a.m.-10 a.m. ET*

Join Zoom Meeting

https://rutgers.zoom.us/j/97770361327?pwd=WXV5SDNNczFubFNpMFRZdnlhcllKUT09&from=addon

I have 20 years experience in developing LC-MS based methods for peptides and proteins in plasma, many of which were used to support pharmacokinetic experiments of biotherapeutics in preclinical and clinical trials. I joined the University of Cambridge in 2016 to set up the peptidomics and proteomics core facility in the Institute of Metabolic Science. Since 2016, we have helped researchers measure and characterise proteins and peptides in various matrices including plasma, tissues, organoids, secretion experiments, and sorted cells.

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Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by: Dr. Jan H.J Hoeijmakers

DNA damage: impact on Cancer and Aging: remarkable applications of Nutritional Interventions

Abstract: *Dr. Jan H.J Hoeijamakers and his research group have discovered that accumulating DNA damage next to causin replication stress, mutations and cancer, also causes transcription stress and aging but at the same time triggers a potent anti-cancer, anti-aging 'survival' response, that resembles calorie restriction (CR). Applying 30% CR dramatically delayed accelerated aging in mouse repair mutants by reducing DNA damage and its sequelae, explaining the anti-aging, anti-cancer mechanism of CR. Translation to the first progeroid DNA repair patients even surpassed the enormous benefits in mice, revising nutritional guidelines for these syndromes. The clinical implications extend to counteracting neurodegeneration, side effects of chemo/radiotherapy, and surgery-related ischemia reperfusion injury.*

Date: October 27,2023

Time: 8:00am-10am ET

Join Zoom Meeting

https://rutgers.zoom.us/j/94062656216?pwd=TElSRWRKNEt5cnVucUJmSitWanJIdz09&from=addon

Dr. Jan H.J. Hoeijmakers, is currently a professor of Molecular Genetics at the Erasmus University in Rotterdam. His research focuses on DNA repair and the impact of nutrition on cancer and aging. His team made major contributions to cloning human repair genes, elucidating underlying repair mechanisms, and generating numerous mouse mutants mimicking rare human repair syndromes. Currently, research on the underlying molecular mechanisms is combined with clinical trials on the protective effect of short-term fasting as nutritional preconditioning for (oncological) surgery and chemotherapy, improving the quality of life of (ex)cancer patients. Dr. Hoeijmakers heads research teams in the Erasmus Medical Center (Rotterdam), Princess Máxima Center for Pediatric Oncology (Utrecht, both in the Netherlands), and CECAD (Cologne, Germany). For his scientific achievements he has obtained numerous national and international awards and distinctions.

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The Cancer Cachexia Action Network presents a seminar by:
Professor Laure Bindels

Gut microbiota in cancer cachexia

Abstract: ***Cancer cachexia is a multifactorial syndrome characterized by muscle wasting and adipose tissue loss, leading to a significant weight loss that impacts patient's quality of life, tolerance to treatment, response to therapy, and survival. Despite the strong contribution of cachexia to cancer morbidity and mortality, muscle and adipose tissue wasting still lack approved therapeutic treatments. During this presentation, I will focus on the contribution and therapeutic interest of the gut microbiota in this context.***

Date: Friday, December 1, 2023

Time: 8:00 a.m.-10:00 a.m.(ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/94381252571?pwd=QmhyRTR4cFlaWU9PMWJZMTkyTm9mQT09&from=addon

Dr. Laure Bindels is a professor at the UCLouvain in Brussels, Belgium. She is a Welbio investigator whose research group has a focus on integrative physiology, metabolism, and nutrition, to investigate the role of the gut microbiota in the development of metabolic and behavioral disorders associated with obesity and cardiometabolic risk, alcohol dependence, cancer development, and cachexia. Dr. Bindel's work over the last ten years demonstrated that the composition and metabolic activity of the gut microbiota are altered in cancer cachexia and that microbiota-targeted approaches can dampen cachectic features.

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The Cancer Cachexia Action Network presents a seminar by: Dr. Mauricio Berriel Diaz

Genomic liver profiling identifies REV-ERB alpha-regulated hepatokines promoting tissue wasting in cancer cachexia

Abstract: *We conducted in-depth genomic profiling of the liver in multiple weight-stable cancer and cancer cachexia models. An integrative multilevel analysis approach identified a distinct gene signature that included hepatocyte-secreted factors and the circadian clock as key modulator of hepatic transcriptional reprogramming in cancer cachexia. Notably, hepatocyte-specific genetic re-constitution of the cachexia-repressed circadian regulator REV-ERBα ameliorated peripheral tissue wasting, revealing a novel mechanism by which the liver contributes to peripheral tissue wasting in cancer cachexia.*

Date: *Friday, April 12,2024*

Time: *8 a.m. to 10 a.m. (ET)*

Join Zoom Meeting:
https://rutgers.zoom.us/j/99783554763pwd=aVBLdE01aVBsQ3p2M3d4c1V6 bHh4UT09&from=addon

Mauricio earned his PhD in Animal Physiology in 2004 from the University of Marburg, Germany, focusing on energy metabolism regulation during torpor, a hibernation-like state of hypometabolism. In 2005, he joined the junior group lead by Stephan Herzig at the German Cancer Research Center (DKFZ) in Heidelberg. Together with Stephan, he developed a research program linking metabolic diseases and cancer. One research focus since then and upon his move to Helmholtz Munich in 2015 has been cancer cachexia, including the characterization of the roles of liver and adipose tissue metabolism as well as tumor-secreted factors. Further contributions addressed different aspects of liver metabolism in the development of metabolic diseases, and specific aspects of tumor metabolism contributing to cancer progression and related therapeutic strategies against cancer.

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The Cancer Cachexia Action Network presents a seminar

by: Dr.Asya Rolls, PhD*(Technion)*

Central Intelligence: Brain Regulation of Physiological and Immune Responses in Cancer

Abstract: ***Cancer is a systemic disease and, thus, requires adaptations of the entire organism's physiology. The brain, as the master regulator of the body, can orchestrate the organism's response to cancer through behavioral and physiological adaptations, including the anti-tumor immune response. This presentation will explore our findings on how the brain's reward system can influence the anti-tumor immune response as well as some new evidence on the brain's role in regulating liver function, which may have significant implications for cachexia management.***

Date: Friday, May 3 2024

Time: 8:00 a.m-9:30 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/98355337181?pwd=UmNFZ3V4aE8vZU0yOE5nUTN0aXM4UT09&from=addon

Asya Rolls studies the physiological mechanisms whereby emotions and thoughts affect physical health. Her laboratory uses chemogenetic, optical, and behavioral approaches to investigate how specific brain activity affects the immune response. By deciphering the neuronal pathways mediating brain-immune signals, her work aims to harness the brain's therapeutic potential. Rolls is a Prof. at the Rappaport Medical School, Technion, Israel Institute of Technology and was an International Howard Hughes Medical Institute (HHMI)-Wellcome investigator.

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Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by: Dr. Denis C. Guttridge, PhD

NF-kB and the muscle microenvironment in regulating cancer cachexia

Abstract: The goal of our laboratory is to understand whether NF-kB regulation of differentiation is relevant in tumorigenesis. To study this property of NF-kB, we utilize skeletal muscle as a model system of differentiation. In vitro and in vivo genetic approaches have provided insight on the function of NF-kB in myogenesis, which led us to understanding the contribution of this signaling pathway in various skeletal muscle disorders, including the cancer cachexia syndrome. We continue to pursue the mechanisms regulated by NF-kB that promote muscle atrophy in cancer with the goal of identifying targets suitable for advanced therapies.

Date: Friday, May 17 2024
Time: 8:00-9:30 (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/96377024139?pwd=YU5HTTZ4ZnhmVWZZMkdnRzBja05lQT09&from=addon

Dr. Guttridge initiated studies on NF-kB and cancer cachexia as a postdoctoral fellow at University of North Carolina, Chapel Hill. He continued this work in his laboratory as an Assistant Professor to Professor at Ohio State University and the James Comprehensive Cancer Center. During that time, Dr. Guttridge led efforts to grow the cancer cachexia community through the organization of international conferences starting in Boston in 2012 and then took part in establishing the current cancer cachexia society.

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The Cancer Cachexia Action Network presents a seminar

by: Dr. Ed Reznik, PhD

Multimodal Metabolic Analysis and Cachexia At-Scale

Abstract: *I will describe our research group's efforts to use heterogeneous, multimodal readouts of metabolism to understand the regulation of metabolism, the etiology of metabolic phenotypes in bulk primary tumors, and the effects of systemic physiology on tumor phenotypes.*

Date: Friday, May 31 2024
Time: 8:00-9:30 (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/98132087864?pwd=WHd2aE9OS1pWYlI4QnExUzhQSklGUT09&from=addon

I am an Assistant Member in the Computational Oncology Service at Memorial Sloan Kettering Cancer Center (MSKCC). I completed my undergraduate studies in Biological and Biomedical engineering at Cornell University and a Ph.D. with Daniel Segre at Boston University, studying genome-scale models of bacterial metabolism. I have worked at MSKCC since 2013, first as a postdoctoral fellow with Chris Sander and subsequently since 2017 as a faculty member. Our laboratory uses computational and quantitative approaches to study tumor metabolism, mitochondrial genetics, and systemic physiology.

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CANCER GRAND CHALLENGES

Team: CANCAN

The Cancer Cachexia Action Network presents a seminar

by: Dr. Puneeth Iyengar M.D., Ph.D.

Identification of novel cancer genetics driving biology associated with unintentional weight loss

Abstract: *In this talk, we will describe our initial approaches to identify novel tumor intrinsic genetics that influence cancer cachexia development. We will then describe more recent efforts using big data approaches that may provide additional insight into unintentional weight loss mechanisms.*

Date: Friday, June 14 2024
Time: 8:00-9:30 a.m. (ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/98206120827?pwd=OStQeThwMk5BS1QvdWx3U1pjaTNYQT09&from=addon

Puneeth Iyengar, MD, PhD and his federally funded laboratory have focused on basic/translational/clinical research efforts primarily in two areas of systems biology that are fundamentally reliant on tumor-host interactions - metastatic development in cancer and mechanisms driving cancer cachexia biology. He received his BS in biology at MIT conducting research with Uttam Lal Rajbhandary and Nobel Laureate Har Gobind Khorana on assembly of protein translation machinery. He completed his MD, PhD at the Albert Einstein College of Medicine (NY), his thesis work under the supervision of Philipp Scherer, identifying how collagen VI secretion from adipose host tissue promotes breast cancer progression. He then completed his residency at MD Anderson Cancer Center before joining UT Southwestern Medical Center in 2010 as a junior faculty. He eventually became Vice Chair at UTSW in the Dept of Radiation Oncology and Co-Director of the Thoracic Oncology program. In 2023, he was recruited to Memorial Sloan Kettering Cancer Center to become Director of the Metastatic Service in Radiation Oncology. Puneeth Iyengar has led multiple phase 1-3 clinical trials focused on synergizing local therapies with immunotherapy-based systemic therapies in advanced disease. His group has also provided insight into how tumor intrinsic and extrinsic biology regulate host tissues in metastatic disease and cachexia.

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CANCER GRAND CHALLENGES

Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by:

Dr. Jason Pitarresi, PhD

Pancreatic cancer cachexia is mediated by tumor-derived PTHrP

Abstract: *We have used mouse models to determine that the pro-cachectic tumor-derived factor PTHrP is upregulated in pancreatic ductal adenocarcinoma. Genetic deletion or pharmacological inhibition of PTHrP reduces cachexia phenotypes in highly cachectic pancreatic cancer lines and overexpression of PTHrP induces cachexia in non-cachectic clones, demonstrating that PTHrP is both necessary and sufficient to drive cachexia. Additionally, the deletion of Pth1r, the cognate receptor for PTHrP, specifically in adipocytes blocked cachexia induction suggesting direct tumor cell to adipocyte crosstalk. Mechanistically, we find that PTHrP blocks fatty acid synthesis in adipose tissue by potently downregulating the de novo lipogenesis (DNL) pathway, and we are developing tools to understand more deeply the role of DNL in cachectic adipose tissue wasting.*

Date: *September 13,2024*

Time: *8 a.m. ET to 9:30 a.m. ET*

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Dr. Jason R. Pitarresi received his PhD from Ohio State University in the laboratory of Dr. Michael C. Ostrowski, where he built new mouse models to study the role of the tumor microenvironment in pancreatic cancer initiation. In his graduate thesis work, he discovered mechanisms to explain the failure of stromal fibroblast targeted Smoothened inhibitors in pancreatic cancer patients. He joined the labs of Anil K. Rustgi and Ben Z. Stanger at the University of Pennsylvania for his postdoc, where he developed highly metastatic mouse models of pancreatic cancer and showed that the metastasis promoting gene PTHrP undergoes collateral amplification as part of the KRAS amplicon. Mechanistically, he demonstrated that PTHrP drives metastasis by initiating epithelial-to-mesenchymal transition (EMT) and subsequently used mouse models to demonstrate that a partial EMT program occurs in pancreatic tumor cells to facilitate the metastatic process. He joined the faculty at the University of Massachusetts Chan Medical School in 2022, where his lab studies how tumor cell plasticity alters tumor-host interactions, focusing on adipose tissue wasting in cancer cachexia and mechanisms of immunosuppression in pancreatic cancer.

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Team: CANCAN

The Cancer Cachexia Action Network presents a seminar by: Dr. Nada Kalaany, PhD.

Spatiotemporal metabolic networks in pancreatic cancer and associated cachexia"

Abstract: *We use an inducible mouse model of pancreatic cancer to conduct a longitudinal analysis of systemic metabolic network alterations that occur during cancer progression and its associated cachexia. The extracted data are leveraged to delineate potential cross-tissue networks and to identify promising strategies for the prevention and treatment of pancreatic cancer cachexia.*

Date: Friday, December 13, 2024

Time: 8:00 a.m.-9:30 a.m.(ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/95260417743?pwd=c3NUR21reExNWlBjdUtJdXA5Tm5adz09&from=addon

Nada Kalaany received her PhD from UT Southwestern Medical Center (at Dallas, Texas), where she studied the role of nuclear hormone receptors in lipid metabolism. She completed her post-doctoral training in Cancer Metabolism at the Whitehead Institute at MIT where she identified a mechanism regulating the sensitivity of tumors to diet restriction. She is currently Associate Professor at Harvard Medical School, Boston Children's Hospital where her lab focuses on identifying in vivo metabolic dependencies in cancers and understanding the metabolic crosstalks between tumors and their hosts.

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The Cancer Cachexia Action Network presens a seminar by:

Dr. Dafna Bar-Sagi PhD

Exercise-immune axis in pancreatic tumorigenesis

Abstract: Pancreatic ductal adenocarcinoma (PDA), which accounts for more than 90% of cases of pancreatic cancer, is one of the most lethal human malignancies. It is currently ranked third among cancer-related deaths in the United States, and, due to a rise in disease incidence, is projected to become the second cause of cancer-associated mortalities by 2030. Effective therapeutic interventions for PDA are limited underscoring the urgent need to identify new treatment options that will improve outcomes for PDA patients. Within this framework, work in our laboratory focuses on assessing the impact of exercise on pancreatic cancer development and progression. We have found that exercise has protective effects on both primary (pancreas) and metastatic (liver) tumor growth. These anti-tumor effects are mediated by exercise-dependent engagement of immune activation pathways that are unique to the host organ and are shaped by the interplay between peripheral and local immunity. The identity and function of these pathways and their translational relevance will be discussed.

Date: Friday,February 28,2025
Time: 8:00 a.m.-9:30 a.m.(ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/97785412655?pwd=Ysty67NNQ7m7ggAuz1YQradIHiW5au.1&from=addon

Dr. Bar-Sagi is a cancer biologist widely recognized for her work on pathophysiological processes that drive the initiation and progression of mutant Ras tumors. She has made fundamental contributions to the understanding of the mechanisms that couples extracellular signals to Ras activation of effector pathways that control cell proliferation and survival. Dr. Bar-Sagi is also credited with the discovery of Ras oncogene-dependent mechanisms that enhance tumor cell fitness via immune evasion and metabolic adaptation. Dr. Bar-Sagi is the Saul Farber Professor of Biochemistry and Molecular Pharmacology. Aside of leading her research program, Dr. Bar-Sagi is Executive Vice President and Vice Dean for Science, Chief Scientific Officer of NYU Langone Health.

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The Cancer Cachexia Action Network presens a seminar by:

Dr.Christine Chio PhD

The role of methionine oxidation in cancer associated cachexia

Abstract: Cancer cachexia, a severe metabolic disorder characterized by progressive fat and muscle loss, is highly prevalent in pancreatic ductal adenocarcinoma (PDA) patients and contributes to their poor prognosis. Recent evidence suggests that adipose tissue loss, driven by increased browning of white adipose tissue (WAT), precedes muscle wasting and may play a critical role in cachexia development. Our research identifies MSRA as a key regulator of WAT browning in cachectic conditions, and genetic deletion of MSRA in mouse models of PDA effectively alleviates cachexia and extends survival, offering potential therapeutic insights.

Date: Friday, March 14
Time: 8:00 a.m.-9:30 a.m.(ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/92348248184?pwd=2hqNTXtbMcPcNCB9Zo4Js1hVe1q8pb.1&from=addon

I am an Assistant Professor of Genetics & Development at Columbia University, with joint appointments at the Institute for Cancer Genetics and the Herbert Irving Comprehensive Cancer Center. My research focuses on the role of reactive oxygen species (ROS) and redox metabolism in cancer progression. During my postdoctoral training at Cold Spring Harbor Laboratory, I helped develop advanced organoid models to study pancreatic ductal adenocarcinoma (PDA) and identified ROS-driven oxidative modifications as key regulators of tumor progression. My lab investigates how redox signaling, such as the reversible oxidation of cysteine and methionine residues, functions as a regulatory mechanism in cancer. Using chemical proteomics, we systematically map these oxidation events across the proteome to determine their impact on PDA progression and therapeutic response. Our ongoing work seeks to define how redox metabolism influences the tumor microenvironment and drives systemic metabolic changes in PDA, such as in the context of cancer-induced cachexia.

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The Cancer Cachexia Action Network presents a seminar

by: Dr. Salvador Aznar Benitah

SYSTEMIC IMPACT OF METASTASIS OVER HOST METABOLISM

Abstract: ***Among the many factors influencing metastasis aggressiveness, special emphasis has been put on the metabolic changes within the cancer cells adapting to the new tumour microenvironments (TME). For this project, we aim to investigate how oral squamous cell carcionoma, pancreatic ductal carcinoma, and metastatic triple-negative breast cancer (mTNBC) regulate the host's metabolism to ensure a contin-uous energy supply for metastatic growth. By unravelling the mechanisms by which metastatic tumours manipulate the host's metabolism, we are gaining insights into novel therapeutic targets for disrupting the energy supply to metastatic cells. This holds the potential to significantly improve the treatment of aggressive metastatic cancers, ultimately leading to improved patient outcomes.***

Date: Friday November 8
Time: 8:00 am-9:30 am ET

Join Zoom Meeting:

https://rutgers.zoom.us/j/92144249025?pwd=3V0BGI6aL0BGrKOiNqakSnhseqTsiF.1&from=addon

Dr.Salvador Aznar Benitah obtained his Honours BSc in Biochemistry and Molecular Biology at McGill University in 1998. In 2007, after a postdoctoral work in the laboratory of Fiona Watt at the London Research Institute (Cancer Research UK), he established his own lab at the Center for Genomic Regulation (CRG) as a Junior ICREA researcher. Dr.Aznar Benitah's lab aims at understanding the molecular mechanisms underlying adult stem cell function during homeostasis, ageing and cancer, with special interests in epigenetics, spatiotemporal regulation of stem cells (circadian rhythms), and the link between metastatic-initiating-cells and their epigenetic and metabolic mechanisms. Based on his work on metastasis, Salvador funded ONA Therapeutics in 2019 as a spin-off of IRB Barcelona and ICREA to develop new therapies targeted against metastasis. Dr. Anzar Benitah is the recipient of several prestigious awards that include the Banc Sabadell Award in Biomedicine (2015), Doctor Diz Pintado Award in Biomedicine (2016), and the Beug Foundation Metastasis Award (2015).

CANCER GRAND CHALLENGES

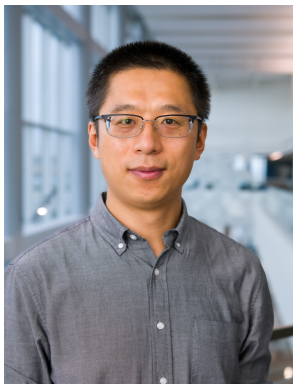
Team: CANCAN

The Cancer Cachexia Action Network presens a seminar by:

Dr. Jinhai Yu, Ph.D.

Cancer Cachexia in STK11/LKB1-mutated NSCLC is Dependent on Tumorsecreted GDF15

Abstract: *Tumor loss-of-function mutations in STK11/LKB1, a regulator of the energy sensor AMPactivatedprotein kinase, induce cancer cachexia (CC) in preclinical models and are associated with cancer-related weight loss in NSCLC patients. Here we characterized the relevance of the integrated stress response (ISR) cytokine growth differentiation factor 15 (GDF15) in regulating cachexia using several patient-derived and genetically engineered STK11/LKB1-mutant NSCLC tumor lines. Antibody neutralization or genetic silencing of GDF15 in multiple human and mouse STK11/LKB1-mutant NSCLC lines were sufficient to eliminate in vivo circulating GDF15 levels and abrogate cachexia induction, suggesting tumor-secreted GDF15 as a conduit and a therapeutic target through which NSCLCs with STK11/LKB1 loss-of-function mutations promote cachexia-associated wasting.*



Date: Friday, May 30th
Time: 8:00 a.m.-9:30 a.m.(ET)

Join Zoom Meeting

https://rutgers.zoom.us/j/99524226870?pwd=YCQEjjmEjLraisFTiigxw0eYJI30aJ.1&from=addon

Dr. Jinhai Yu earned his Ph.D. from the University of Chinese Academy of Sciences and completed postdoctoral training at Tsinghua University. In 2018, he joined Rodney Infante's lab as a postdoctoral researcher and is now an Assistant Professor in the Cachexia Group at the Center for Human Nutrition, UT Southwestern Medical Center. Using patient-derived and genetic-engineered cancer cachexia models, their group is trying to unravel the tumor-intrinsic drivers of cancer cachexia. They demonstrated that tumor STK11/LKB1 loss-of-function mutations are not only a biomarker but also causal in precipitating host wasting. Building on this insight, they recently identified tumor-secreted GDF15 as both necessary and sufficient to induce cachexia in the context of STK11/LKB1 deficient NSCLC, positioning GDF15 inhibition as a therapeutic strategy for this high-risk patient subset. Through close collaboration with clinical teams, they are now advancing GDF15-targeted approaches toward clinical evaluation, with the goal of improving quality of life and survival for STK11/LKB1 loss-of-function mutated NSCLC patients.

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