



Conference Co-Chairpersons

Frank McCormick, UCSF Helen Diller Family Comprehensive Cancer Center, San Francisco, CA

Dafna Bar-Sagi, New York University Langone Medical Center, New York, NY

Channing J. Der, UNC Lineberger Comprehensive Cancer Center, Chapel Hill, NC

Remarkable discoveries regarding how RAS proteins function as key regulators of signal transduction and drivers of oncogenesis have emerged over the past three decades. Despite intensive efforts by the pharmaceutical industry, these findings have not been translated into clinically effective anti-RAS therapies, and RAS has been widely perceived as “undruggable.” However, recent genome-wide sequencing studies performed in search of new targets revealed that RAS and its key effectors (BRAF and PIK3CA) are the only oncogenes mutated beyond single digit frequencies in the cancers that comprise three of the top four causes of death in the U.S. (lung, colon, and pancreatic cancer). This has prompted renewed interest in targeting RAS, including the recent US National Cancer Institute announcement of a RAS “megaproject” to support such efforts. The primary focus of this AACR Special Conference was to discuss both the exciting progress and the many still-unresolved issues that will affect how these discoveries can be leveraged. This conference brought together over 300 researchers from academia and industry to assess the opportunities, challenges, and prospects of achieving the holy grail of cancer research, a therapeutic approach for the ~30 percent of human cancers that are RAS-mutant.

The AACR would like to thank the following organizations for their generous support of this conference.

Professional Educational Grants



The AACR thanks the following organizations for their generous support of the travel awards provided at this conference.

Award Supporters



Center to
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Conference Program

MONDAY, FEBRUARY 24

7:00 p.m.-8:30 p.m. OPENING SESSION /WELCOME/KEYNOTE TALK
Grand Harbor Ballroom South

Ras membrane localization, ciliary trafficking, and anti-RAS drug candidates
Alfred Wittinghofer, Max Planck Institute of Molecular Physiology, Dortmund, Germany

8:30 p.m.-10:00 p.m. OPENING RECEPTION
Salons 5-8

TUESDAY, FEBRUARY 25

7:00 a.m.-8:00 a.m. BREAKFAST
Asbury Hall Foyer

8:00 a.m.-10:00 a.m. PLENARY SESSION 1: RAS REGULATION
Grand Harbor Ballroom South

Membrane targeting of RAS
Mark R. Philips, New York University, New York, NY

Developing therapies for RAS driven cancers
Karen Cichowski, Brigham & Women's Hospital, Boston, MA

Regulation of K-RAS function by lysine acetylation
Kevin M. Haigis, Massachusetts General Hospital, Charlestown, MA

Yap1 activation enables bypass of oncogenic K-RAS addiction in pancreatic cancer*
Haoqiang Ying, University of Texas MD Anderson Cancer Center, Houston, TX

Myc-Ras cooperation can overwhelm tumor suppressive mechanisms within lung adenocarcinomas*
Catherine H. Wilson, University of Cambridge, Cambridge, United Kingdom

10:00 a.m.-10:30 a.m. BREAK
Asbury Hall Foyer

*Short talks from proffered papers

10:30 a.m.-12:30 p.m. PLENARY SESSION 2: RAS EFFECTORS 1 – BASIC BIOLOGY

Grand Harbor Ballroom South

Mechanisms regulating RAF signaling in normal and disease states

Deborah K. Morrison, National Cancer Institute, Frederick, MD

From RAS to ERK: Unraveling the importance of isoform differences

Adrienne D. Cox, University of North Carolina at Chapel Hill, Chapel Hill, NC

Targeting K-RAS signaling in lung and pancreatic adenocarcinomas

Mariano Barbacid, Spanish National Cancer Research Center (CNIO), Madrid, Spain

Global quantitative proteomic and phosphoproteomic analysis of oncogenic K-RAS-driven mouse pancreatic cancer*

Ayumu Taguchi, The University of Texas MD Anderson Cancer Center, Houston, TX

Cooperation between RAS network mutations in cancer*

Edward C. Stites, Washington University School of Medicine, St. Louis, MO

12:30 p.m.-2:30 p.m. LUNCH ON OWN

2:30 p.m.-4:30 p.m. PLENARY SESSION 3: RAS EFFECTORS 2 – THERAPEUTICS

Grand Harbor Ballroom South

Next-generation strategies to target RAF

Gideon E. Bollag, Plexxikon Inc., Berkeley, CA

Title to be announced

Neal Rosen, Memorial Sloan-Kettering Cancer Center, New York, NY

Discovery of a first-in-class ERK 1,2 inhibitor

Ahmed A. Samatar, West Windsor, NJ

Disruption of CRAF-mediated MEK activation is required for effective MEK inhibition in KRAS mutant tumors*

Piro Lito, Memorial Sloan Kettering Cancer Center, New York, NY

Impact of RAL signal transduction on genetic program and growth control in KRAS- and BRAF-mutated colorectal cells and prognostic potential of pathway-responsive genes in cancer patients*

Reinhold Schäfer, Charité Universitaetsmedizin, Berlin, Germany

4:30 p.m.-7:00 p.m. POSTER SESSION A with RECEPTION

Salons 5-8

*Short talks from proffered papers

WEDNESDAY, FEBRUARY 26

7:00 a.m.-8:00 a.m. BREAKFAST
Asbury Hall Foyer

8:00 a.m.-10:00 a.m. PLENARY SESSION 4: RAS BIOLOGY – SYSTEMS APPROACHES
Grand Harbor Ballroom South

Tissue- and tumor-specific functions of H- and K-RAS isoforms in mouse models of cancer
Allan Balmain, UCSF Helen Diller Family Comprehensive Cancer Center, San Francisco, CA

Systematic functional genomics and K-RAS
William C. Hahn, Dana-Farber Cancer Institute, Boston, MA

Identifying K-RAS synthetic lethal interactions using systems biology and functional genomics
Alejandro Sweet-Cordero, Stanford University, Stanford, CA

Copper is required for oncogenic BRAF signaling and tumorigenesis
Chris M. Counter, Duke University Medical Center, Durham, NC

10:00 a.m.-10:30 a.m. BREAK
Asbury Hall Foyer

10:30 a.m.-12:30 p.m. PLENARY SESSION 5: MODELS OF RAS-DRIVEN CANCERS
Grand Harbor Ballroom South

Signaling through individual RAS proteins in the absence of the Nf1-RASGAP
Nancy Ratner, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

Progress in pancreatic cancer modeling
David A. Tuveson, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY

Oncogenic K-RAS and the inflammatory microenvironment in pancreatic cancer
Marina Pasca di Magliano, University of Michigan, Ann Arbor, MI

Targeting aberrant RAS signaling in mouse cancer models
Kevin M. Shannon, University of California, San Francisco, CA

12:30 p.m.-3:00 p.m. POSTER SESSION B with LUNCH
Salons 5-8

*Short talks from proffered papers

RAS ONCOGENES: FROM BIOLOGY TO THERAPY

3:00 p.m.-4:00 p.m. PLENARY SESSION 6: POINT/COUNTERPOINT
Grand Harbor Ballroom South

4:00 p.m.-5:30 p.m. RAS INTERACTOME and NETWORKING
Grand Harbor Ballroom South

Justin Guinney, Sage Bionetworks, Seattle, WA

5:30 p.m. EVENING OFF/DINNER ON OWN

THURSDAY, FEBRUARY 27

7:00 a.m.-8:00 a.m. BREAKFAST
Salons 5-8

8:00 a.m.-10:00 a.m. PLENARY SESSION 7: RAS TARGETING 1
Grand Harbor Ballroom South

Discovery of K-RAS inhibitors for the treatment of cancer
Stephen W. Fesik, Vanderbilt University School of Medicine, Nashville, TN

Activation of RAS by post-translational modification: Ubiquitination and thiol oxidation
Sharon Campbell, University of North Carolina, Chapel Hill, NC

Title to be announced
Channing J. Der, University of North Carolina Comprehensive Cancer Center, Chapel Hill, NC

Crystal structure of K-Ras G12C bound to an active site inhibitor*
Ken Westover, UT Southwestern Medical Center, Dallas, TX

NF1*, *MET*, and *RIT1* mutations are RAS-pathway driver events in lung adenocarcinoma
Alice H. Berger, Broad Institute, Cambridge, MA

10:00 a.m.-10:30 a.m. BREAK
Salons 5-8

*Short talks from proffered papers

10:30 a.m.-12:30 p.m. PLENARY SESSION 8: RAS TARGETING 2

Grand Harbor Ballroom South

Exploiting context-dependent fragility in oncogenic RAS signaling networks

Michael A. White, University of Texas Southwestern Medical Center, Dallas, TX

Role of autophagy in cancer

Eileen P. White, UMDNJ-The Cancer Institute of New Jersey, New Brunswick, NJ

Oncogenic and WT RAS proteins: A family affair

Dafna Bar-Sagi, New York University Langone Medical Center, New York, NY

A systems biology approach to elucidate novel drug targets in KRAS mutant tumors*

David A. Wah, Columbia University, New York, NY

A novel model of metastatic melanoma driven by endogenous *N-RAS*^{Q61R} expression*

Christin E. Burd, The Ohio State University, Columbus, OH

12:30 p.m.-12:45 p.m. CLOSING REMARKS / DEPARTURE

Grand Harbor Ballroom South

*Short talks from proffered papers