A01 Novel state I structures of oncogenic KRAS4b mutants bound to GTP analog. Albert H. Chan, Frederick National Laboratory for Cancer Research, Frederick, MD.

A02 Probing amino acids residues chemical reactivity of KRAS 4b using N-hydroxysuccinimide esters. Oleg Chertov, Frederick National Laboratory for Cancer Research, Frederick, MD.

A03 Biochemical and structural analysis of the neurofibromin (NF1) protein and a potential role for protein destabilization in Rasopathy diseases. Dominic Esposito, Frederick National Laboratory for Cancer Research, Frederick, MD.

A04 Biophysical and biochemical characterization of Src-phosphorylated KRas. Teklab Gebregiworgis, Margaret Cancer Centre, University Health Network and Department of Medical Biophysics, University of Toronto, Toronto, ON, Canada.

A05 Context-dependent transformation with activated Ras isoforms in human normal epithelial cells. Minami Kumazaki, National Cancer Center Research Institute, Tokyo, Japan.

A06 Biophysical and biochemical characterization of KRAS G12C inhibition through the SMART™ platform. Earl W. May, Warp Drive Bio, Cambridge, MA.

A07 Inflammation enables pancreatic acinar cells to overcome resistance to oncogenic Kras by increasing its expression and plasma membrane localization. Mohamad Nabil Assi, Université Catholique de Louvain, de Duve Institute, Brussels, Belgium.

A08 Genetic drug resistance screen identifies LZTR1 as regulator of RAS ubiquitination and signaling. Johannes W. Bigenzahn, CeMM Center for Molecular Medicine, Vienna, Austria.

A09 Cooperative membrane interaction between G-domain and HVR defines unique diffusion behavior of KRAS4b. Debanjan Goswami, De Chen, John Columbus, Thomas Turbyville, FNLCR, NCI-Frederick, Frederick, MD.

A10 Quantitative biophysical analysis defining key components modulating KRAS recruitment to the plasma membrane. Frantz L. Jean-Francois, Frederick National Laboratory, Frederick, MD.

A11 Screening the Ras proteome microenvironment using APEX2. Stephanie P. Mo, University of Liverpool, Liverpool, United Kingdom.

A12 Mutations in RABL3 alter KRAS prenylation and are associated with hereditary pancreatic cancer. Sahar Nissim, Brigham and Women's Hospital, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA.
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A13 CRAS4A directly regulates hexokinase 1. Mark R. Philips, NYU Perlmutter Cancer Center, New York, NY.

A14 Mutations in the ubiquitin ligase adaptor LZTR1 drive human disease by dysregulating RAS ubiquitination and signaling. Anna Sablina, KULeuven/VIB, Leuven, Belgium.

A15 Axl-mediated activation of TBK1 drives epithelial plasticity in pancreatic cancer. Rolf A. Brekken, UT Southwestern, Dallas, TX.

A16 Understanding the principles of tissue repair that accelerate tumor initiation. Sara Gallini, Yale University, New Haven, CT.

A18 Characterization of K-RasG13D as a unique activating allele in a mouse model of colorectal cancer. Yi-Jang Lin, Beth Israel Deaconess Medical Center, Boston, MA.

A19 Conditional inactivation of SHOC2 in adult mice to study its role in tissue homeostasis. Sibel Sari, UCL Cancer Institute, London, United Kingdom.

A20 An essential role for Argonaute 2 in mouse models of KRAS driven cancers. Sunita Shankar, University of Michigan, Ann Arbor, MI.

A21 Loss of Argonaute 2 leads to oncogene-induced senescence in mutant RAS-driven cancer. Ronald F. Siebenaler, University of Michigan, Ann Arbor, MI.

A22 Kras drives changes in acinar-specific gene regulatory networks in early pancreatic neoplasia in conjunction with Bmi1. Joyce K. Thompson, University of Michigan, Ann Arbor, MI.

A23 A second site KrasG12D mutation that impairs PI3K binding rescues embryonic lethality, abrogates myeloproliferative disease, and delays lung tumorigenesis. Jasmine C. Wong, University of California, San Francisco, San Francisco, CA.

A24 New mouse models with KRASG12D or KRASG12V mutation in Amhr2-Cre mice develop different gynecologic tumors. Kwong-Kwok Wong, The University of Texas MD Anderson Cancer Center, Houston, TX.

A25 In vivo evidence validating the palmitoylation/depalmitoylation cycle as a therapeutic target in NRAS mutant hematologic cancers. Noemi A. Zambetti, Department of Pediatrics, University of California San Francisco, San Francisco, CA.

A26 Precise characterization and comparison of KRAS proteoforms by top-down mass spectrometry. Caroline J. DeHart, Northwestern University, Evanston, IL.
A28 USP21 promotes stemness of pancreatic cancer cells and bypass of KRAS extinction. Pingping Hou, University of Texas MD Anderson Cancer Center, Houston, TX.

A29 The gastric cancer-associated mutations R5W and Y42C in the RAS homologous RHOA protein cause distinct biochemical alterations, exhibit gain-of-function signaling and oncogenic activities. Antje Schaefer, University of North Carolina at Chapel Hill, Chapel Hill, NC.

A30 Neurofibromatosis type 1 (NF1) regulates the RAS-related GTPases, RRAS and RRAS2, independent of RAS activity in melanoma cells. Jillian M. Silva, University of California San Francisco, San Francisco, CA.

A31 Germline RASopathy mutations provide functional insights into the Raf cysteine-rich domain (CRD). Russell Spencer-Smith, NCI-Frederick, Frederick, MD.

A32 Selective contribution of the SHOC2 phosphatase complex to ERK pathway dynamics highlights its potential as a therapeutic target. Isabel Boned del Río, University College London, Cancer Institute, London, United Kingdom.

A33 Combinations with CDK4/6 inhibitors to treat cancers with mutations in both KRAS and CDKN2A. Sean G. Buchanan, Eli Lilly, Indianapolis, IN.

A34 Defining KRAS mutation-specific kinome signatures and vulnerabilities in colorectal cancer. James Duncan, Fox Chase Cancer Center, Philadelphia, PA.

A36 Neoadjuvant-like Ezh2 inhibition in Kras-driven lung cancer amplifies inflammation and creates new therapeutic vulnerabilities. Gaetano Gargiulo, Max-Delbrück-Center for Molecular Medicine (MDC), Berlin, Germany.

A37 Mapping KRAS signaling pathways using the Mammalian-Membrane Two-Hybrid (MaMTH) assay to elucidate novel therapeutic targets. Ingrid Claudia Grozavu, University of Toronto, Toronto, ON, Canada.

A39 The role of YAP in regulating glycogen metabolism in pancreatic cancer. Sung Eun (Monica) Kim, University of California San Francisco, San Francisco, CA.

A40 Combinatorial knockout of Rap1GDS1 and RhoA leads to lethality in KRAS-driven non-small cell lung cancer. Kaja Kostyrko, University of California San Francisco, San Francisco, CA.

A41 Nucleotide metabolism heterogeneity in mutant KRAS pancreatic cancer. Thuc M. Le, University of California Los Angeles, Los Angeles, CA.
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A42 Bromodomain inhibitors suppress Nrf2-dependent HO-1 positive macrophage accumulation in murine models of KRAS-mutated pancreatic cancer. Ana S. Leal, Michigan State University, East Lansing, MI.

A43 Investigating novel inhibitors of the IMP-1-KRAS mRNA interaction. Victor Liu, University of Northern British Columbia, Prince George, BC, Canada.

A44 SHP2 inhibition overcomes RTK-mediated pathway reactivation in KRAS-mutant tumors treated with MEK inhibitors. Hengyu Lu, Novartis Institutes for BioMedical Research, Cambridge, MA.

A45 Protective autophagy elicited by RAF→MEK→ERK inhibition suggests a treatment strategy for RAS-driven cancers. Martin McMahon, Huntsman Cancer Institute, University of Utah, Salt Lake City, UT.

A46 A pan-cancer RAS mutant library elucidates the transformation potential of RAS variants. Amanda R. Moore, Genentech Inc, South San Francisco, CA.

A47 Targeting glutaminolysis potentiates the efficacy of chemotherapy in RAS-driven pancreatic cancers. Suman Mukhopadhyay, Frederick National Laboratory, Frederick, MD.

A48 Therapeutic reactivation of the protein phosphatase 2A (PP2A) for the treatment of KRAS-driven cancers. Goutham Narla, University of Michigan, Ann Arbor, MI.

A49 Specific Kras codon 12 and 13 mutations display different tumor initiation in pancreatic cancer. Maria Paz Zafra Martin, Sandra and Edward Meyer Cancer Center, Department of Medicine, Weill Cornell Medicine, New York.

A50 Molecular Targeting of HuR Oncoprotein for Melanoma Treatment. Rebaz Ahmed, Univ. of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma.