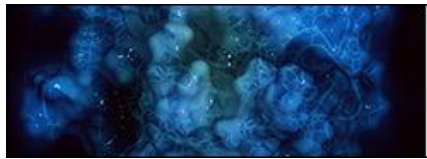


PROFERRED TALKS

- PR001 B045 **Targeting S6K2 overcomes resistance to RAS inhibition in NRAS-mutant melanoma**, Jessie Villanueva, The Wistar Institute, Philadelphia, United States
- PR002 A049 **Identification of a RAS-GTP Threshold for Malignant Transformation**, Sophie Krahnke, Frederick National Laboratory, Frederick, MD, United States
- PR003 A029 **MEK1/2 degraders uncover kinase-independent role of MEK1/2 in CRAF stabilization and maturation**, James Duncan, Fox Chase Cancer Center, Philadelphia, PA, United States
- PR004 A026 **Dissecting the critical ERK functions that support KRAS-driven pancreatic cancer**, Jennifer Klomp, Michigan State University, Grand Rapids, MI, United States
- PR005 A044 **KRAS amplification as de novo oncogenic alteration and therapeutic target in human cancers**, Mark Awad, Memorial Sloan Kettering Cancer Center, New York, NY, United States
- PR006 A045 **Comprehensive structure-function analysis reveals gain- and loss-of-function mechanisms impacting oncogenic KRAS activity**, Jason Kwon, Dana-Farber Cancer Institute, Boston, United States
- PR007 B007 **Anti-tumor efficacy of the selective oral KRAS G12D dual ON/OFF inhibitor VS-7375 as a single agent and in combination with targeted agents**, Jonathan Pachter, Verastem Oncology, Needham, MA, United States
- PR008 B004 **Preclinical activity of an orally bioavailable PROTAC pan-KRAS degrader versus inhibitors in mutant KRAS models**, Andrea Lopez-Arroyo, Arvinas Operations, Inc., New Haven, CT, United States
- PR009 A007 **The RAS(ON) multi-selective inhibitor, daraxonrasib (RMC-6236), induces Receptor Tyrosine Kinase (RTK) cell surface expression on pancreatic cancer cells, providing rationale for combinations with RTK targeting agents**, Ida Aronchik, Revolution Medicines, Redwood City, CA, United States
- PR010 A008 **ERK hyperactivation-induced lethality as a therapeutic strategy in RAS-driven tumors**, Alexa Cannon, Novartis Biomedical Research, Cambridge, MA, United States
- PR011 A032 **KRAS amplification creates a targetable pMHC antigen for T cell engager therapy to overcome KRAS inhibitor resistance**, Lauren Stopfer, Aethon Therapeutics, New York City, NY, United States
- PR012 A033 **Kras G12C and G12D driven lung cancers differ in oncogenic potency, immunogenicity, and relapse following Kras inhibition**, Esra Akbay, University of Texas Southwestern Medical Center, Dallas, TX, United States



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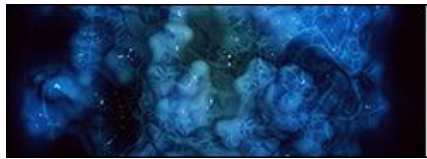
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PR013 B011 **Dynamic kinome reprogramming and metabolic rewiring drive adaptive resistance to RAS inhibition in pancreatic cancer**, Clint Stalneck, University of North Carolina at Chapel Hill, Chapel Hill, NC, United States

PR014 B012 **A PP2A molecular glue overcomes RAS/MAPK inhibitor resistance in KRAS-mutant non-small cell lung cancer**, Goutham Narla, The University of Michigan, Ann Arbor, MI, United States

PR015 B044 **Silent KRAS mutations confer altered sensitivity to targeted KRAS inhibition**, Andrew Waters, University of Cincinnati, Cincinnati, OH, United States

PR016 B048 **De novo design of Ras isoform selective binders**, Jason Zhang, UCLA, Los Angeles, CA, United States



POSTER SESSION A
Friday, March 6, 2026

A001 Uncovering the role of IMP2 in RAS signaling and colorectal cancer progression. Jessica Das. CUNY Graduate Center, New York, NY, United States.

A002 KRAS inhibitors as sensitizing agents in treatment-refractory rectal cancer. Chao Wu. Department of Colon & Rectal Surgery, The University of Texas MD Anderson Cancer Center, Houston, TX, United States.

A003 Enhancing the efficacy of KRAS-targeted therapy in colorectal cancer via Aurora kinase A inhibition. Zhaojin Liu. University of Southern California, Los Angeles, CA, United States.

A004 Experimental and Computational Validation of Alcea rosea Compounds in Blocking Colorectal Cancer Progression. Ruhban Parry. University of Kashmir, Srinagar, 190003, India.

A005 D3S-002: A Purposefully Designed ERK1/2 Inhibitor Achieving Low-Dose, Pulsatile Target Inhibition for Combination with D3S-001, a New-Generation KRAS G12C Inhibitor. Jing Zhang. D3 Bio, Inc., Shanghai, Taiwan (Greater China).

A006 Synergistic co-targeting of KRAS G12V and pan-TEAD by an EGFR-directed, inverted chimeric RNAi molecule. Chad Pecot. University of North Carolina - Lineberger Cancer Center, Chapel Hill, NC, United States.

B045 Targeting S6K2 overcomes resistance to RAS inhibition in NRAS-mutant melanoma. Jessie Villanueva. The Wistar Institute, Philadelphia, United States.

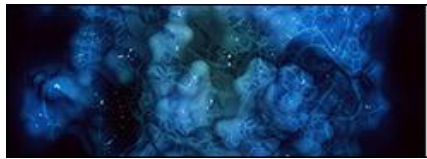
A049 Identification of a RAS-GTP Threshold for Malignant Transformation. Sophie Krahnke. Frederick National Laboratory, Frederick, MD, United States.

A009 Synergistically overcoming KRAS-driven radioresistance and immune evasion in PDAC with alpha particle radiotherapy and anti-CTLA-4. Marco Reis. UT MD Anderson and Rice University, Houston, TX, United States.

A010 Advancing new rational drug combinations to treat mutant KRAS-driven pancreatic adenocarcinoma. Brajendra Tripathi. National Cancer Institute, National Institutes of Health, Bethesda, MD, United States.

A011 Decoding the codons: Uncovering KRAS mutant isoform-specific vulnerabilities in non-small cell lung cancer. Will McDaid. University of Manchester, MANCHESTER, United Kingdom.

A012 Combined AXL and KRAS inhibition synergize and drive immune recruitment in KRAS-mutant NSCLC. Fredrik Thege. The Ohio State University Comprehensive Cancer Center, Columbus, OH, United States.



A013 Targeted therapy-induced chromosomal instability dictates mitotic dependency on Aurora Kinase A. Chendi Li. Massachusetts General Hospital/ Harvard Medical School, Boston, MA, United States.

A014 Mapping the genetic landscape of KRAS mutant cancer cells using combinatorial CRISPR screens. Rand Arafeh. Dana Farber Cancer Institute, Harvard Medical School & Broad Institute of MIT and Harvard, Boston, MA, United States.

A015 AB801, a potent and selective clinical-stage AXL inhibitor, enhances the anti-tumor efficacy and duration of response of KRAS inhibitors. Ester Fernandez-Salas. Arcus Biosciences, Hayward, CA, United States.

A016 GAP mimetics display synergy with K-Ras Switch-II inhibition. Patrick Pfaff. Department of Cellular and Molecular Pharmacology and Howard Hughes Medical Institute, University of California, San Francisco (UCSF), San Francisco, CA, United States.

A017 RAS inhibition and cytotoxic chemotherapy target complementary cell states in pancreatic cancer. Kenneth Olive. Columbia University Irving Medical Center, New York, NY, United States.

A018 Functional chemo-genomic screening identifies novel combination therapies to treat RAS-driven multiple myeloma. Omar S. Al-Odat. NCI, Bethesda, MD, United States.

A019 Enhancing RAS therapies by targeting PIKfyve in pancreatic cancer. Caleb Cheng. University of Michigan, Ann Arbor, MI, United States.

A020 Applying a functional precision medicine platform, Optim.AI™, to identify novel KRAS inhibitor-based combinations in pancreatic cancer. Edward Chow. KYAN Technologies Pte Ltd, Singapore, Singapore.

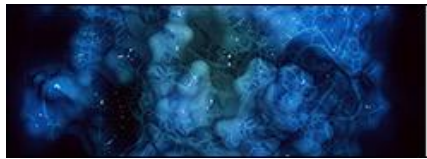
A021 Enhancing therapeutic efficacy of RAS-targeted cancer therapy via co-targeting DNA topoisomerase II. Zhen Chen. Winship Cancer Institute of Emory University, Atlanta, GA, United States.

A022 Combination benefit of treatment with focal adhesion kinase inhibitor narmafotinib and KRAS inhibitors. Christopher Burns. Amplia Therapeutics, Melbourne, VIC, Australia.

A023 Combined RAS and ICB inhibition targets NF-κB–driven immune evasion in chemoresistant pancreatic cancer. Kevin Christian Gulay. University of California San Diego, San Diego, CA, United States.

A024 Characterization of rutin binding to HRAS and MAPK3 and its clinical prognostic relevance in lung cancer: Using in silico and clinical prognostic experimental design. Dr. Hossam Kamli. King Khalid University, Abha, Saudi Arabia.

A025 Unveiling Synergistic Potential of VHL and KEAP1-based PROTACs for Targeted Protein Degradation. Sehbanul Islam. University of Pennsylvania, Philadelphia, PA, United States.



A029 MEK1/2 degraders uncover kinase-independent role of MEK1/2 in CRAF stabilization and maturation. James Duncan. Fox Chase Cancer Center, Philadelphia, PA, United States.

A027 Molecular dynamics driving phenotypic divergence among KRAS mutants in pancreatic tumorigenesis. David Falvo. Weill Cornell Medicine, New York, NY, United States.

A028 Non-genetic resistance to multi-selective RAS(ON) inhibitors in NRAS-mutant melanoma. Hee Won Yang. Columbia University, New York, NY, United States.

A026 Dissecting the critical ERK functions that support KRAS-driven pancreatic cancer. Jennifer Klomp. Michigan State University, Grand Rapids, MI, United States.

A030 Branched Actin-driven Cell Membrane Protrusions Regulate Oncogenic KRAS Molecular Signaling. Gabriel Muhire Gihana. UT Southwestern Medical Center, Dallas, TX, United States.

A031 A metabolic weak spot: GJB3–SLC7A11 Synthetic lethality in colon cancer. Disha Acharya. Indian Institute of Technology, Dharwad, Chikkamalligwad, Dharwad, India.

A044 KRAS amplification as de novo oncogenic alteration and therapeutic target in human cancers. Mark Awad. Memorial Sloan Kettering Cancer Center, New York, NY, United States.

A045 Comprehensive structure-function analysis reveals gain- and loss-of-function mechanisms impacting oncogenic KRAS activity. Jason Kwon. Dana-Farber Cancer Institute, Boston, United States.

A034 Targeting phosphodiesterase 10A by ADT-030 normalizes the tumor immune microenvironment by inhibiting RAS-MAPK signaling in both cancer cells and myeloid-derived suppressor cells. Gang Zhou. Augusta University, Augusta, GA, United States.

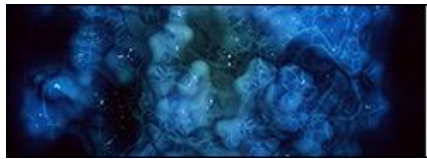
A035 TCR-T cell therapy targeting mutant KRAS in pancreatic cancer using a humanized co-culture and mouse model system. Georg Hilfenhaus. Charité-Universitätsmedizin Berlin, Berlin, Germany.

A036 Developing the next generation of T cell engager (TCE): Combining high-throughput immunoassays with generative AI to develop TCEs toward multiple RAS mutations. Dubravka Pezic. Synteny Biotechnology, London, United Kingdom.

A037 Oncogenic KRAS signaling reversibly suppresses immunotherapy responses in non-small cell lung cancer. Drithi Patel. Thomas Jefferson University, Philadelphia, PA, United States.

A038 Targeting RAS in gynecologic cancers. Elizabeth Stover. Dana-Farber Cancer Institute, Boston, MA, United States.

A039 Towards the discovery of a panKRAS binder via the use of antibody derivatives. Davide Cardella. The Institute of Cancer Research, Sutton, United Kingdom.



A040 Remarkable in vivo responses to Ras targeting drug Daraxonrasib in a chemotherapy-resistant MMTV-Kras G12D syngeneic mammary tumor model. Mehrnoosh Arabi. Tulane School of Medicine, New Orleans, LA, United States.

A041 Genetic landscape of intracholecystic papillary neoplasms (ICPNs) of the gallbladder - STK11 as a characteristic driver gene for ICPNs-. Satomi Saito. Department of Investigative Pathology, Tohoku University, Sendai, Japan.

A042 Clinicopathological and molecular profiling of pancreatic adenocarcinoma: The Moroccan experience. Fatima El Agy. 1. Laboratory of Biomedical and Translational Research, Faculty of Medicine, Pharmacy, and Dentistry of Fez, Sidi Mohamed Ben Abdellah University, Fez, Morocco, FEZ, Morocco.

A043 Phosphorylation Protects Oncogenic RAS from LZTR1-Mediated Degradation. Lin Zhang. NIH, Bethesda, MD, United States.

B007 Anti-tumor efficacy of the selective oral KRAS G12D dual ON/OFF inhibitor VS-7375 as a single agent and in combination with targeted agents. Jonathan Pachter. Verastem Oncology, Needham, MA, United States.

B004 Preclinical activity of an orally bioavailable PROTAC pan-KRAS degrader versus inhibitors in mutant KRAS models. Andrea López-Arroyo. Arvinas Operations, Inc., New Haven, CT, United States.

A046 Discovery and characterization of a novel cryptic pocket in KRAS. Michel Maira. Novartis Biomedical Research Oncology, Basel, Switzerland.

A047 KRAS G12C and G12D mutants exhibit distinct conformational flexibility in the helix 3–switch 2 pocket that drives differential protein function. ALOK SHARMA. NCI RAS Initiative, Cancer Research Technology Program, Frederick National Laboratory for Cancer Research, Leidos Biomedical Research, Inc., FREDERICK, MD, United States.

A048 Structure of SHOC2-KRAS-PP1C complex reveals RAS isoform-specific determinants and insights into targeting complex assembly by RAS inhibitors. Jacob Potter. Frederick National Laboratory for Cancer Research, Frederick, MD, United States.

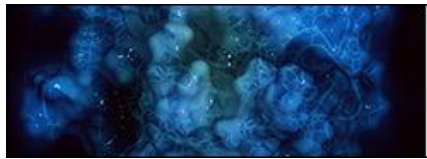
A007 The RAS(ON) multi-selective inhibitor, daraxonrasib (RMC-6236), induces Receptor Tyrosine Kinase (RTK) cell surface expression on pancreatic cancer cells, providing rationale for combinations with RTK targeting agents. Ida Aronchik. Revolution Medicines, Redwood City, CA, United States.

A050 Mutant allele copy number gains define distinct molecular subtypes of oncogene-driven cancers and are associated with response to allele-specific KRAS G12C inhibitor sotorasib in NSCLC. Maria Perry. Memorial Sloan Kettering Cancer Center, New York, NY, United States.



A051 Recurrent RRAS and RRAS2 mutations in lung cancer define actionable oncogenic drivers with therapeutic susceptibility to pan-RAS inhibition. Alexander Pfeil. Memorial Sloan Kettering Cancer Center, Chapel Hill, NC, United States.

A052 ADT-030: A novel PDE10 inhibitor with robust and durable antitumor activity and the capacity to overcome resistance common to RAS inhibitors through dual blockade of RAS and β -catenin signaling. Gary Piazza. Auburn University, Auburn, AL, United States.



POSTER SESSION B
Saturday, March 7, 2026

B001 eIF2B Selectively Anchors and Activates Mutant KRAS4B. Hyungdong Kim. Lady Davis Institute for Medical Research, Sir Mortimer B. Davis Jewish General Hospital, Montreal, Quebec, H3T 1E2, Canada / Graduate Program in Clinical and Translational Research, Faculty of Medicine, McGill University, Montreal, Quebec, H4A 3J1, Canada, Montreal, QC, Canada.

B002 Structural and functional analysis of the resistance mechanisms of KRAS-G12C secondary mutations to switch-II pocket inhibitors. Matthew Whitley. Frederick National Laboratory for Cancer Research, Frederick, MD, United States.

B003 Pan-Cancer Systematic Meta-Analysis of RAS Oncogene Mutational Landscapes and Therapeutic Vulnerabilities. Shivi Kumar. University of Pennsylvania, Philadelphia, TX, United States.

A008 ERK hyperactivation-induced lethality as a therapeutic strategy in RAS-driven tumors. Alexa Cannon. Novartis Biomedical Research, Cambridge, MA, United States.

B005 Noncovalent Pan-KRAS inhibitors: Broad-spectrum targeting of KRAS mutations in cancer. Greg Jones. Pfizer, San Diego, CA, United States.

B006 Screening a disulfide library against engineered cysteine RAS mutants reveals cryptic binding pockets. Trent Balius. Frederick National Laboratory for Cancer Research, Frederick, MD, United States.

A032 KRAS amplification creates a targetable pMHC antigen for T cell engager therapy to overcome KRAS inhibitor resistance. Lauren Stopfer. Aethon Therapeutics, New York City, NY, United States.

B008 Pharmacokinetics of daraxonrasib (RMC-6236): transporter and enzyme impact on a first-in-class pan-RAS molecular glue. Davinia Arguedas. Netherlands Cancer Institute (NKI), Amsterdam, Netherlands.

B010 Mutational landscape of RTK–RAS signaling pathway in acute myeloid leukemia: Insights from whole exome sequencing. HARSH GOEL. ALL INDIA INSTITUTE OF MEDICAL SCIENCES, NEW DELHI, India.

A033 Kras G12C and G12D driven lung cancers differ in oncogenic potency, immunogenicity, and relapse following Kras inhibition. Esra Akbay. University of Texas Southwestern Medical Center, Dallas, TX, United States.

B011 Dynamic kinome reprogramming and metabolic rewiring drive adaptive resistance to RAS inhibition in pancreatic cancer. Clint Stalneck. University of North Carolina at Chapel Hill, Chapel Hill, NC, United States.

B013 Reversing the ferroptosis-resistant phenotype of KEAP1 -mutant lung adenocarcinoma through glutaminase 1 inhibition enhances the efficacy of KRAS inhibitors. Amirali Karimi. The University of Texas MD Anderson Cancer Center, Houston, TX, United States.



B014 A mechanistically distinct pan-RAS inhibitor, ADT-007, with robust antitumor activity evades resistance common to mutant-specific and pan-RAS Inhibitors. Gary Piazza. Auburn University, Auburn, AL, United States.

B015 Leveraging patient-derived organoids and matched 2D models to elucidate resistance mechanisms to KRAS inhibition in pancreatic cancer. Nicolas Lecomte. David M. Rubenstein Center for Pancreatic Cancer Research, Memorial Sloan Kettering Cancer Center, New York City, NY, United States.

B016 hSSB2 Activators as a Dual Therapeutic Strategy for Overcoming RAS/MAPK Inhibitor Resistance While Protecting Normal Epithelia in Aggressive Cancers. Andrew Norris. BCN Biosciences, Pasadena, CA, United States.

B017 Mechanisms regulating resistance to KRAS inhibitors driven by Δ Np63 in lung adenocarcinoma. Santanu Adhikary. H. Lee Moffitt Cancer Center and Research Institute, Tampa, FL, United States.

B018 ZNF384 Orchestrates Cell-State Reprogramming and Kinome Remodeling in KRAS G12C Inhibitor Resistance. Chendi Li. Massachusetts General Hospital Cancer Center/Harvard Medical School, Boston, MA, United States.

B019 NFAT5 regulates KRAS target therapy resistance in pancreatic cancer. Pingping Hou. Rutgers New Jersey Medical School, Newark, NJ, United States.

B020 TRIM7 Inhibition Blocks RTK/RAS Pathway-Driven Tumor Proliferation Independent of Mutation and in the Setting of KRASi Resistance. George Fromm. Kayak Therapeutics, Inc., Durham, NC, United States.

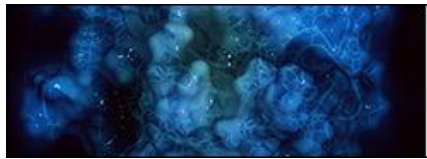
B021 Genome-wide CRISPR Screen Identifies Menin as a Mediator of Encorafenib Plus Cetuximab Resistance in BRAF V600E -mutant Colorectal Cancer. Akash Srivaths. Olivia Newton-John Cancer Research Institute, Melbourne, Australia.

B023 Identification of TME-dependent resistance and emergence of persister cells to KRAS inhibition. Niloofar Khairkhah. University of Michigan, Ann Arbor, MI, United States.

B024 KRAS inhibition is an effective therapy for appendiceal adenocarcinoma: Single cell profiling identifies tumor intrinsic and microenvironmental mechanisms unique to appendix cancer. John Paul Shen. Univ of Texas MD Anderson, Houston, TX, United States.

B025 Potential limitation of oncogenic KRAS-targeted therapy in lung cancer due to stromal remodeling. Brock Humphries. University of Michigan, Ann Arbor, MI, United States.

B026 RAF1 gene amplification drives RAS inhibitor resistance via nuclear translocation and interactions with PLK1 and Aurora A kinases. Hitendra Solanki. H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, United States.



B027 Functional genomics studies identify determinants of response vs. resistance to pharmacological inhibitors of RAS in multiple myeloma. Constantine Mitsiades. Dana-Farber Cancer Institute, Boston, MA, United States.

B028 Adaptive Signaling Rewiring Enables Rapid, Sequential Resistance to KRAS and Pan-RAS Inhibitors. Ines Pulido. University of Illinois Chicago, Chicago, United States.

B029 Immune checkpoint-mediated resistance to KRAS G12D inhibition. Niloofar Khairkhan. University of Michigan, Ann Arbor, MI, United States.

B030 A window trial in metastatic pancreatic ductal adenocarcinoma reveals resistance mechanisms to targeting the KRAS-MEK pathway. Motoyuki Tsuda. Oregon Health and Science University, Portland, OR, United States.

B031 A high-throughput combination screen identifies NT-1 as a superior compound to overcome KRAS G12D inhibitor resistance. Natalie Thielen. Albert Einstein College of Medicine, Bronx, NY, United States.

B033 Resistance to the pan-RAS(ON) tri-complex inhibitor daraxonrasib is overcome by direct multi-KRAS inhibitors, QTX3034 and QTX3544. Jillian Silva. Quanta Therapeutics, South San Francisco, CA, United States.

B034 Genetic and non-genetic mechanisms of resistance to KRAS inhibition in CRC. Lukas Dow. Weill Cornell Medicine, New York, NY, United States.

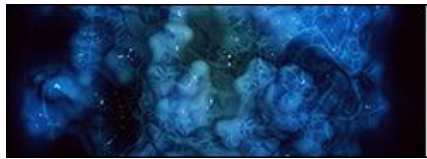
B035 Investigating adaptive responses in KRAS/LKB1 co-mutant tumors. Mikoto Kobayashi. University of Michigan, Ann Arbor, MI, United States.

B036 FYN antagonizes response to RAS inhibition in pancreatic ductal adenocarcinoma. Hsiu-Chi Ting. Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, United States.

B037 Comparison of RAS G12C(ON) and KRAS G12C(OFF) inhibitor activity in parental or KRAS G12C-amplified tumors via mathematical modeling. Muhammad Ali Al-Radhawi. Revolution Medicines, Redwood City, CA, United States.

B038 Paneth-like transition drives resistance to dual targeting of KRAS and EGFR in colorectal cancer. Yijun Gao. Sun Yat-sen University Cancer Center, Guangzhou, Taiwan (Greater China).

B040 Understanding and Overcoming RAS-pathway Mediated Therapeutic Resistance and Disease Progression in Myeloproliferative Neoplasms. Subyeta Chowdhury. Memorial Sloan Kettering Cancer Center, New York, NY, United States.



B041 TIMP1-Mediated regulation of angiogenesis differs between KRAS-Dependent and KRAS-Independent cells in NSCLC. Ilamathi M-Thirusenthilarasan. Penn State College of Medicine, Hershey, PA, United States.

B042 Therapeutic options for cancers with oncogenic Ras mutations. Andrew Wolfe. Hunter College, New York, NY, United States.

B043 Multi-Omic mapping reveals stress-MAPK-dependent mechanisms of resistance to KRAS G12C inhibitors in colorectal cancer. Kasturi Nayak. Quantitative Biosciences Institute and Department of Medicine, University of California San Francisco, San Francisco, CA, United States.

B012 A PP2A molecular glue overcomes RAS/MAPK inhibitor resistance in KRAS-mutant non-small cell lung cancer. Goutham Narla. The University of Michigan, Ann Arbor, MI, United States.

B044 Silent KRAS mutations confer altered sensitivity to targeted KRAS inhibition. Andrew Waters. University of Cincinnati, Cincinnati, OH, United States.

B046 Pan-RAS Inhibition as a novel therapeutic strategy for RAS-driven rhabdomyosarcoma. Patience Odeniyide. The Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins University, Baltimore, MD, United States.

B047 Pre-treatment with azacytidine sensitizes RAS-mutated secondary AML to the pan-RAS inhibitor RMC-7977. Tessa Seale. Johns Hopkins University School of Medicine, Baltimore, MD, United States.

B048 De novo design of Ras isoform selective binders. Jason Zhang. UCLA, Los Angeles, CA, United States.

B049 Overcoming treatment adaptation to RAS(ON) inhibition in NRAS/HRAS mutant pediatric tumors. Anand Patel. St. Jude Children's Research Hospital, Memphis, TN, United Arab Emirates.

B050 Identifying and characterizing the role of the adapter protein, LNK in NRAS mutant melanoma. Meghan Vrkoc. McGill University, Montreal, QC, Canada.

B051 Targeting RAS to stimulate pyroptosis and antitumor immunity in acral melanoma. Geethanjali Annamalai. Thomas Jefferson University, Philadelphia, PA, United States.