



AACR Special Conference in Cancer Research

## RAS ONCOGENESIS AND THERAPEUTICS

March 5-8, 2026 | Los Angeles, CA

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### 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

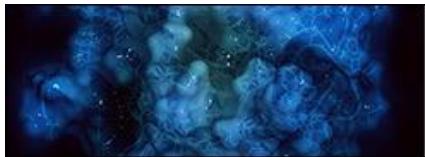
**PR008 B004 Preclinical activity of an orally bioavailable PROTAC pan-KRAS degrader versus inhibitors in mutant KRAS models**, Andrea Lopez-Arroyo, Arvinas Operations, Inc., New Haven, CT, United States

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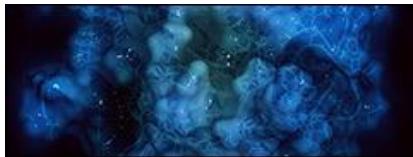
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**PR013 B011 Dynamic kinase reprogramming and metabolic rewiring drive adaptive resistance to RAS inhibition in pancreatic cancer**, Clint Stalnecker, University of North Carolina at Chapel Hill, Chapel Hill, NC, United States

**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.



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**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.



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**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.



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**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.



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**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  $\beta$ -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 Balias. 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 daraxorrasib (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 kinase reprogramming and metabolic rewiring drive adaptive resistance to RAS inhibition in pancreatic cancer.** Clint Stalnecker. 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.



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**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 ΔNp63 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.



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**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 Khairkhah. 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.



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**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.

**B042 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.