

## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session A

Monday, Dec. 10, 2018

1:30 p.m.-3:30 p.m.

**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.

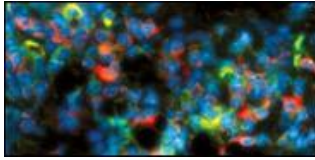
**A08 Genetic drug resistance screen identifies LZTR1 as regulator of RAS ubiquitination and signaling.** Johannes W. Bigenzahn, CeMM Center for Molecular Medicine, Vienna, Austria.

**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.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session A

Monday, Dec. 10, 2018

1:30 p.m.-3:30 p.m.

**A13 KRAS4A 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-Ras<sup>G13D</sup> 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.

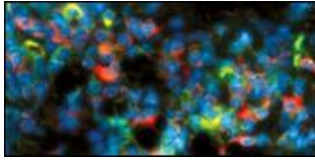
**A22 Kras drives changes in acinar-specific gene regulatory networks in early pancreatic neoplasia in conjunction with Bmi1.** Joyce K. Thompson, University of Michigan, Ann Arbor, MI.

**A23 A second site *Kras*<sup>G12D</sup> 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.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session A

Monday, Dec. 10, 2018

1:30 p.m.-3:30 p.m.

**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 Rio, 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.

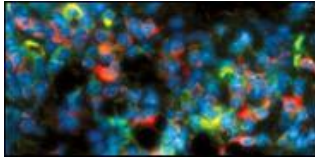
**A36 Neoadjuvant-like Ezh2 inhibition in Kras-driven lung cancer amplifies inflammation and creates new therapeutic vulnerabilities.** Gaetano Gargiulo, Max-Delbrück-Center for Molecular Medicine (MDC), Berlin, Germany.

**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.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session A

Monday, Dec. 10, 2018

1:30 p.m.-3:30 p.m.

**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.

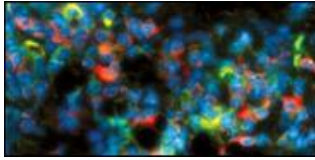
**A46 A pan-cancer RAS mutant library elucidates the transformation potential of RAS variants.** Amanda R. Moore, Genentech Inc, South San Francisco, CA.

**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.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session B

Tuesday, Dec. 11  
5:25 p.m.-7:25 p.m.

**B01 Inhibition of RAS signaling and tumorigenesis through targeting vulnerabilities in RAS biochemistry.** John P. O'Bryan, MUSC, UIC, Charleston, SC.

**B02 Dissecting tumor cell heterogeneity to identify therapeutic vulnerabilities in Kras-mutant lung cancer.** Aparna Padhye, University of Texas MD Anderson Cancer Center, Houston, TX.

**B03 Biologic and biochemical interactions of NF1 GAP on KRAS G13x mutations.** Dana Rabara, Frederick National Laboratory for Cancer Research, Frederick, MD.

**B04 High-level expression of oncogenic KRAS is required to transform LKB1 mutant tissue in vivo.** Briana B. Rackley, Emory University, Atlanta, GA.

**B05 ASN007, an oral ERK1/2 inhibitor, shows strong antitumor activity across a panel of KRAS subtype mutant cancer models.** Sanjeeva P. Reddy, Asana BioSciences, Lawrenceville, NJ.

**B06 ING2 loss sensitizes KRAS-mutated NSCLC to WEE1 inhibition through regulation of CHK1 expression.** Charles Ricordel, Université de Rennes 1, Rennes, France.

**B07 Inhibition of Ras signalling by targeting Son of Sevenless with Affimers.** Sophie E. Saunders, University of Leeds, Leeds, United Kingdom.

**B08 ETC inhibitors alter oncogenic KRAS signal transduction.** Kanika Sharma, Frederick National Laboratory for Cancer Research, Frederick, MD.

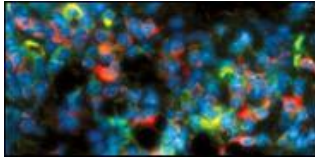
**B09 Combining proteomics and genetic screens to identify KRAS synthetic lethal interactions.** Shikha S. Sheth, Cancer Research Institute, Beth Israel Deaconess Cancer Center and Department of Medicine, Harvard Medical School, Boston, MA.

**B10 Modeling the genetic heterogeneity of KRAS mutant lung adenocarcinomas for therapeutic discovery.** Kate D. Sutherland, The Walter and Eliza Hall of Medical Research, Melbourne, VIC, Australia.

**B11 O-GlcNAcylation is required for mutant KRAS-induced lung tumorigenesis.** Phuoc T. Tran, Johns Hopkins University School of Medicine, Baltimore, MD.

**B12 DOCK1 as a novel target for controlling RAS-driven cancer cell survival and invasion.** Takehito Uruno, Kyushu University, Medical Institute of Bioregulation, Fukuoka-city, Japan.

**B13 Parallel targeting of RAF/MEK/ERK pathway in RAS-mutant embryonal rhabdomyosarcoma.** Angelina V. Vaseva, Greehey Children's Cancer Research Institute, The University of Texas Health Science Center, San Antonio, TX.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session B

Tuesday, Dec. 11  
5:25 p.m.-7:25 p.m.

**B14 CRAF-mediated inactivation of epigenetic repressor complexes promotes KRAS-driven tumorigenesis.** Avinashnarayan Venkatanarayan, Genentech, Inc., South San Francisco, CA.

**B15 Dabrafenib-trametinib-induced pyrexia successfully treated with colchicine.** Jesus Vera, Mayo Clinic, Rochester, MN.

**B16 Role of RasGRF2 in AnxA6-mediated growth of TNBC cells.** Diva S. Whalen, Meharry Medical College, Nashville, TN.

**B17 MEK inhibition induces myogenic differentiation in RAS-driven rhabdomyosarcoma.** Marielle E. Yohe, NCI, Bethesda, MD.

**B18 *Clostridium perfringens* lethal toxin specifically targets RAS and disrupts RAS signaling pathway.** Maria Abreu-Blanco, Frederick National Laboratory for Cancer Research, Frederick, MD.

**B19 New tools to study the role of RAS/CRAF interaction in RAS-driven lung cancer.** Romain Baer, The Francis Crick Institute, London, United Kingdom.

**B20 Unbiased high-throughput screenings to identify combination therapies targeting RAS-mutated colorectal cancer.** Rajat Bhattacharya, University of Texas MD Anderson Cancer Center, Houston, TX.

**B21 Cancer-specific intracellular delivery of therapeutic antibodies against KRAS.** Kathlynn C. Brown, SRI International, Harrisonburg, VA.

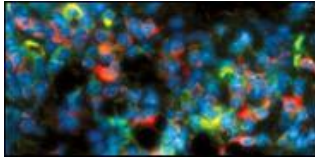
**B22 Identifying protein interactors of oncogenic Kras in pancreatic cancer cells via proximity labelling.** Derek K. Cheng, David A. Tuveson, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.

**B23 Insight towards therapeutic susceptibility of KRAS mutant cancers from MRTX1257: A prototype selective inhibitor of KRAS G12C.** James G. Christensen, Mirati Therapeutics, San Diego, CA.

**B24 Development of small-molecule RAS inhibitors using Affimer reagents.** Katarzyna Haza, University of Leeds, Leeds, United Kingdom.

**B25 Combined proteomic and genetic interaction mapping reveals new Ras pathway effectors and regulators.** Peter K. Jackson, Stanford University, Stanford, CA.

**B26 The SHOC2 phosphatase complex as a therapeutic target for ERK-pathway inhibition in RAS-driven tumors.** Greg G. Jones, University College London, London, United Kingdom.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session B

Tuesday, Dec. 11  
5:25 p.m.-7:25 p.m.

**B27 A DARPIn-based toolbox to understand and treat RAS-addicted cancers.** Jonas N. Kapp, University of Zurich, Zurich, Switzerland.

**B28 Direct targeting oncogenic Ras mutants by IgG-format cytosol-penetrating antibody.** Yong-Sung Kim, Ajou University, Suwon, Republic of Korea.

**B29 SHANK3 in oncogenic RAS signaling.** Johanna Lilja, Turku Centre for Biotechnology, University of Turku, Turku, Finland.

**B30 Structure-based drug discovery of MRTX1257, a selective, covalent KRAS G12C inhibitor with oral activity in animal models of cancer.** Matthew A. Marx, Mirati Therapeutics, San Diego, CA.

**B31 Combination inhibitor strategies targeting KRAS effector signaling in KRAS-mutant pancreatic cancer.** Irem Ozkan-Dagliyan, University of North Carolina, Chapel Hill, NC.

**B32 Silencing of oncogenic KRAS by a mutant-favoring short interfering RNA.** Bjoern Papke, University of North Carolina at Chapel Hill, Chapel Hill, NC.

**B33 Inhibition of Ras using Affimers.** Ajinkya Rao, University of Leeds, Leeds, United Kingdom.

**B34 Targeted destruction of endogenous K-RAS using an Affinity directed PROtein Missile (AdPROM).** Sascha O. M. Roth, MRC Protein Phosphorylation and Ubiquitylation Unit, School of Life Sciences, University of Dundee, Dundee, United Kingdom.

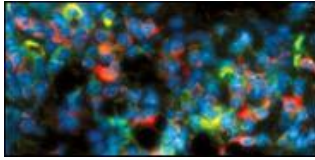
**B35 Discovery of K-Ras(G12D)-targeting peptide KRpep-2d and its optimization strategy.** Kotaro Sakamoto, ICHIMARU PHARCOS Co., Ltd., Motosu-shi, Gifu, Japan.

**B36 Berberine induces apoptosis in cervical carcinoma cells by inducing DNA damage and inhibition of RAS MAPK pathway.** Mayank Singh, All India Institute of Medical Sciences Delhi, New Delhi, India.

**B37 Development of inhibitors of the activated form of KRAS G12C.** Michelle L. Stewart, Warp Drive Bio, Cambridge, MA.

**B38 Ras clipping by bacterial toxin RRSP reduces viability and proliferation of Ras-dependent cancer cell lines in 2D and 3D in vitro models.** Vania Vidimar, Northwestern University, Chicago, IL.

**B39 Characterization of the interaction between KRAS and Argonaute 2.** Jessica Waninger, University of Michigan, Ann Arbor, MI.



## Targeting RAS-Driven Cancers

December 9-12, 2018 | San Diego, CA



### Poster Session B

Tuesday, Dec. 11  
5:25 p.m.-7:25 p.m.

**B40 Role of mutant HRAS in growth and drug sensitivity of head and neck squamous cell cancers (HNSCC).** Adrienne D. Cox, University of North Carolina at Chapel Hill, Chapel Hill, NC.

**B43 A systems biology approach to elucidate the mechanism of EGFR inhibitor sensitivity in mutant KRAS-driven colorectal cancer.** Thomas McFall, Salk Institute, La Jolla, CA.

**B44 KRAS and RAS signaling network is co-regulated and can be therapeutically blocked by targeting eIF4A dependent translation program.** Kamini Singh, Cancer Biology and Genetics Program, Memorial Sloan Kettering Cancer Center, New York, NY.

**B45 Surveillance of RAS-RAF dynamics in vivo: Tracking activity conformations and drug-induced interactions.** Eduard Stefan, University of Innsbruck, Innsbruck, Austria.

**B46 Systems modeling of Ras reveals systems mechanisms that dictate response to treatment.** Edward C. Stites, Salk Institute for Biological Studies, La Jolla, CA.

**B47 Systems-level dissection of tumor-macrophage crosstalk in ovarian cancer resistance to MEK inhibition.** Stephanie J. Wang, Massachusetts Institute of Technology, Cambridge, MA.

**B49 “Triple wild-type” co-mutational profile in early-stage KRAS-mutant lung cancer.** Colin R. Lindsay, University of Manchester, Manchester, United Kingdom.

**B50 ERK MAPK inhibition enhances the immunogenicity of KRAS-mutant colorectal cancer.** Meagan B. Ryan, Massachusetts General Hospital Cancer Center, Boston, MA.

**B51 MAPK regulation of an innate immune response in KRAS-mutant lung adenocarcinoma.** Daniel Sisler, University of Colorado-Anschutz Medical Campus, Aurora, CO.

**B52 KRAS-IRF2 axis drives immune suppression and immune therapy resistance in colorectal cancer.** Alan Wang, University of Texas MD Anderson Cancer Center, Houston, TX.

**B53 Differential response of distinct KRAS mutants to SHP2 inhibition.** Sara Mainardi, Molecular Carcinogenesis Division, The Netherlands Cancer Institute, Amsterdam, The Netherlands.