Sixth AACR-IASLC International Joint Conference

LUNG CANCER TRANSLATIONAL SCIENCE FROM THE BENCH TO THE CLINIC

January 11-14, 2020 | Marriott Marquis San Diego | San Diego, CA

Proceedings Supplement

Session Chairs

PLENARY SESSION 1: THERAPEUTIC TARGETING AND VULNERABILITIES OF RAS-DRIVEN LUNG CANCER
Session Chair: John V. Heymach, The University of Texas MD Anderson Cancer Center, Houston, TX

PLENARY SESSION 2: LUNG PRENEOPLASIA AND EARLY DETECTION
Session Chair: Pierre Massion, Vanderbilt-Ingram Cancer Center, Nashville, TN

PLENARY SESSION 3: METABOLISM
Session Chair: David B. Shackelford, UCLA David Geffen School of Medicine, Los Angeles, CA

POSTER SESSION A HIGHLIGHTS
Session Chair: Christine M. Lovly, Vanderbilt University School of Medicine, Nashville, TN

PLENARY SESSION 4: PRECISION IMMUNOTHERAPY
Session Chair: Katerina A. Politi, Yale Cancer Center, New Haven, CT

PLENARY SESSION 5: TARGETING TUMOR SUPPRESSORS AND "UNDRUGGABLE" TARGETS
Session Chair: Kris C. Wood, Duke University, Durham, NC

PLENARY SESSION 6: CELLULAR THERAPIES, VACCINES, AND NEW IO MODALITIES
Session Chair: Edward B. Garon, University of California Los Angeles, Los Angeles, CA

PLENARY SESSION 7: SMALL-CELL LUNG CANCER
Session Chair: David G. McFadden, UT Southwestern Medical Center, Dallas, TX

PLENARY SESSION 8: METASTASIS AND TUMOR PROGRESSION
Session Chair: David Feldser, University of Pennsylvania, Philadelphia, PA

POSTER SESSION B HIGHLIGHTS
Session Chair: Trever G. Bivona, University of California San Francisco, San Francisco, CA

PLENARY SESSION 9: GENOMIC MECHANISMS OF RESISTANCE
Session Chair: Robert C. Doebele, University of Colorado Denver, Aurora, CO

PLENARY SESSION 10: NONGENETIC MECHANISMS OF RESISTANCE
Session Chair: Christine M. Lovly, Vanderbilt University School of Medicine, Nashville, TN

Updated Talk Title / Change in Speaker Order

PLENARY SESSION 9: GENOMIC MECHANISMS OF RESISTANCE

8:00 a.m.-8:25 a.m.	**Title to be announced** Robert C. Doebele
8:25 a.m.-8:50 a.m.	**Targeting osimertinib resistance in EGFR-mutant NSCLC: A clinical perspective** Zofia Piotrowska, Massachusetts General Hospital, Boston, MA
8:50 a.m.-9:15 a.m.	**Investigating and overcoming primary resistance of EGFR and HER2 (ERBB2) exon 20 mutant NSCLC** Jacqulyne Robichaux, The University of Texas MD Anderson Cancer Center, Houston, TX
9:15 a.m.-9:35 a.m.	**Genetic contributors to tumor progression and drug resistance in EGFR mutant lung cancer** Katerina A. Politi, Yale Cancer Center, New Haven, CT
9:35 a.m.-9:45 a.m.	**Advocate presentation** Janet Freeman-Daily, Cofounder, The ROS1ders
9:45 a.m.-9:55 a.m.	**Advocate presentation** Jill Feldman, Cofounder, EGFR Resisters

Updated Location

Poster Highlight Sessions A and B will take place in Pacific Ballroom 23/24.

Renumbered Poster

B27 IHH acts as a tumor suppressor of lung adenocarcinoma by repressing reactive oxygen species. Sahba Kasiri[1], Baozhi Chen[1], Alexandra Wilson[1], Annika Reczek[1], Simbarashe Mazambani[2], Jashkaran Gadhvi[2], Evan Noel[1], Ummay Marriam[1], Barbara Mino[3], Wei Lu[3], Luc Girard[1], Luisa Solis[3], Katherine Luby-Phelps[1], Justin Bishop[1], Jung-whan Kim[2], James Kim[1]. [1]UT Southwestern Medical Center, Dallas, TX, [2]The University of Texas at Dallas, Dallas, TX, [3]The University of Texas MD Anderson Cancer Center, Houston, TX.

This is now being presented as Poster A01.

Late Abstract

A15 Cancer-associated mesenchymal cells influence lung cancer metastatic phenotypes in vitro and in vivo. Austin Krueger, Douglas J. Saforo, Leah J. Siskind, Levi J. Beverly. University of Louisville, Louisville, KY.

Lung cancer is the leading cause of cancer deaths worldwide among both men and women. The vast majority of all cancer deaths are caused by metastatic dissemination of the disease. The extracellular environment surrounding and within a tumor, the tumor microenvironment, comprises a variety of components and multiple cell types. The interactions between different cell types and their associated extracellular matrices (ECM) are thought to play a role in cancer progression and metastasis, as well as therapeutic responses to cytotoxic and immunotherapies. The precise molecular details for how interactions between individual components of the tumor microenvironment impact cancer progression and metastasis are not well understood. Elucidating complex interactions within the tumor microenvironment is essential for identifying novel therapeutic targets, but has proved challenging because isolating and studying cancer-associated stromal cells from primary lung tumors has remained difficult. To begin to fill this knowledge gap, we developed a rapid, reliable, and reproducible culture system that allows for the isolation and expansion of large quantities of primary cancer-associated mesenchymal (CaM) cells. Briefly, by combining fibroblast-derived extracellular matrices with hypoxic culture conditions, we developed a microenvironmental mimetic cell culture system. Primary lung cancer biopsies are dispersed and put into this system, and the resulting cells that expand are CaM cells with stem-like properties. We show that CaM cells and their deposited extracellular matrices alter signaling pathways and migration of lung cancer cells in vitro, and when coinjected with lung cancer cells, CaM cells induce metastasis in vivo. Thus, the overall hypothesis of this work is that CaM cells drive lung cancer metastasis by reprogramming the extracellular milieu and inducing metastatic signaling within the cancer cells. We are now working to determine the specific mechanisms by which primary CaM cells influence metastatic phenotypes of cancer cells. Further, these experiments will lead to novel therapeutic targets by identifying interactions and signaling events that are only initiated by CaM cells and their deposited ECM within the tumor microenvironment.

Presenting Author Changes

A14 Circulating ensembles of tumor-associated cells are ubiquitous in lung cancers. Dadasaheb B. Akolkar[1], Sewanti Limaye[2], Darshana Patil[1], Pradip Fulmali[1], Pooja Fulmali[1], Sachin Apurwa[1], Sushant Pawar[1], Vineet Datta[1], Cynthe Sims[1], Ajay Srinivasan[1], Rajan Datar[1]. [1]Datar Cancer Genetics Limited, Nasik, Maharashtra, India, [2]Kokilaben Dhirubhai Ambani Hospital, Mumbai, India.

The presenting author has changed from Dadasaheb B. Akolkar to Cynthe Sims.

B35 Circulating tumor-associated cells in lung cancers are resistance-educated per previous chemotherapy treatments. Dadasaheb B. Akolkar[1], Sewanti Limaye[2], Darshana Patil[1], Sanket Patil[1], Vishakha Mhase[1], Sachin Apurwa[1], Sushant Pawar[1], Vipul Todarwal[1], Vineet Datta[1], Cynthe Sims[1], Ajay Srinivasan[1], Rajan Datar[1]. [1]Datar Cancer Genetics Limited, Nasik, Maharashtra, India, [2]Kokilaben Dhirubhai Ambani Hospital, Mumbai, India.

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