### Wednesday, January 4, 2017

5:00 p.m.-6:00 p.m. **Welcome and Opening Keynote** 

Legends Ballroom 1-4

A global genetic interaction network maps a wiring diagram of cellular function

Charles Boone, University of Toronto, Toronto, Ontario, Canada

6:00 p.m.-8:00 p.m. **Welcome Reception** 

The Edge

## Thursday, January 5, 2017

7:00 a.m.-8:00 a.m. **Breakfast** 

The Edge

8:00 a.m.-10:00 a.m. Plenary Session 1: Model Organisms to Identify Synthetic Lethal Interactions I

Legends Ballroom 1-4

Session Chair: Lars Zender, University Hospital Tuebingen, Tuebingen, Germany

To be announced Lars Zender

A functional variomic chemi-genetic screen in C. elegans identifies new synthetic lethal interactions with PARP inhibition that are conserved from worm to human\*

Nigel J. O'Neil, University of British Columbia, Vancouver, BC, Canada

To be announced

Nevan J. Krogan, University of California, San Francisco, CA

Synthetic lethality screen identifies novel druggable targets in the MYC pathway\*

Yulin Li, Stanford University, Stanford, CA

Exploring and exploiting aberrant self-fate programs in leukemia

Johannes Zuber, Research Institute of Molecular Pathology, Vienna, Austria

10:00 a.m.-10:30 a.m. **Break** 

Abbey Road

<sup>\*</sup>Short talk from proffered abstract

10:30 a.m.-12:30 p.m.

### Plenary Session 2: New Technology & Bioinformatics I

Legends Ballroom 1-4

Session Chair: Stephane Angers, University of Toronto, Toronto, Ontario, Canada

Massively parallel search for synthetic lethal vulnerabilities using CRISPRi and CRISPRa Jonathan Weissman, University of California, San Francisco, CA

Linking AP/MS-driven protein-protein interaction networks and combination CRISPR/sgRNA screens defines new Kras effectors and target candidates for non-small cell lung cancer\*

Peter K. Jackson, Stanford University School of Medicine, Stanford, CA

Leveraging genome-wide CRISPR screens and synthetic lethal interactions for novel cancer therapeutics

Stephane Angers

A CRISPR-based genetic interaction map identifies synergistic drug combinations for cancer\*

Kyuho Han, Stanford University, Stanford, CA

Defining, optimizing, and altering the specificities of CRISPR-Cas nucleases J. Keith Joung, Massachusetts General Hospital, Harvard Medical School, Charlestown, MA

12:30 p.m.-2:30 p.m.

#### Lunch on own

### 2:30 p.m.-4:30 p.m.

# Plenary Session 3: Finding Synthetic Lethal Interactions through Functional Genomics I

Legends Ballroom 1-4

Session Chair: René Bernards, Netherlands Cancer Institute, Amsterdam,

The Netherlands

Systematic interrogation of cancer dependencies

William C. Hahn, Dana-Farber Cancer Institute, Boston, MA

CRISPR pooled screening of hundreds of cancer cell lines identifies differential dependencies on epigenetic pathways and synthetic lethal relationships\* Alexandra R. Grassian, Epizyme, Inc., Cambridge, MA

Genome-wide CRISPR screens illuminate lymphoma pathogenesis and therapeutic resistance

Louis M. Staudt, National Cancer Institute, Bethesda, MD

CRISPR screens identified drivers of endocrine resistance and synthetic lethal vulnerabilities in breast cancer\*

Wei Li, Dana-Farber Cancer Institute, Boston, MA

Finding vulnerabilities of drug resistant cancers

René Bernards

#### 4:30 p.m.-6:30 p.m.

### **Reception and Poster Session A**

Penny Lane and Legends Ballroom 5-6

<sup>\*</sup>Short talk from proffered abstract

## Friday, January 6, 2017

7:00 a.m.-8:00 a.m. Breakfast

The Edge

8:00 a.m.-10:00 a.m. Plenary Session 4: Model Organisms to Identify Synthetic Lethal Interactions II

Legends Ballroom 1-4

Session Chair: Alejandro Sweet-Cordero, University of California, San Francisco, CA

Combinatorial screens in Drosophila cells

Norbert Perrimon, Harvard Medical School, Boston, MA

Collateral sensitivity in chemotherapy resistance\*

Simona Dalin, Massachusetts Institute of Technology, Cambridge, MA

Widespread rewiring of genetic interaction networks upon cancer pathway activation Michael Boutros, German Cancer Research Center, Heidelberg, Germany

Combinatorial CRISPR-Cas9 reveals many cancer synthetic lethal interactions are

private to cell type\*

John Paul Shen, University of California San Diego, La Jolla, CA

A novel KRAS specific vulnerability in the nutrient stress response

Alejandro Sweet-Cordero

10:00 a.m.-10:30 a.m. Break

Abbey Road

10:30 a.m.-12:30 p.m. Plenary Session 5: New Technology & Bioinformatics II

Legends Ballroom 1-4

Session Chair: Trey Ideker, University of California San Diego, La Jolla, CA

Understanding the complex biology of KRAS mutant cancers using genetic screens Tina Ling Yuan, Novartis Institutes for Biomedical Research, Cambridge, MA

Harnessing synthetic lethality to predict clinical outcomes of cancer treatment\* Joo Sang Lee, University of Maryland, College Park, MD

Genetic screens to study cancer

David M. Sabatini, MIT Whitehead Institute for Biomedical Research, Cambridge, MA

ScreenBEAM: A novel meta-analysis algorithm for functional genomics screens via bayesian hierarchical modeling\*

Jiyang Yu, St. Jude Children's Research Hospital, Memphis, TN

Systematic mapping and modeling of genetic interaction networks in cancer cells

Trey Ideker

<sup>\*</sup>Short talk from proffered abstract

12:30 p.m.-2:30 p.m. Lunch and Poster Session B

The Edge and Legends Ballroom 5-6

2:30 p.m.-5:00 p.m. Plenary Session 6: Finding Synthetic Lethal Interactions through Functional

**Genomics II** 

Legends Ballroom 1-4

Session Chair: Angelique Whitehurst, UT Southwestern Simmons Comprehensive

Cancer Center, Dallas, TX

Synthetic-lethal strategies for MYC-driven cancers

Andrei Goga, University of California, San Francisco, CA

MCL1-dependent triple-negative breast cancers are exquisitely sensitive to

concomitant inhibition of proteasome and RNA splicing functions\*
Fabio Petrocca, Boston University School of Medicine, Boston, MA

Genome-wide in-vivo tumor xenograft CRISPR knockout screening for identifying

KRAS mutant synthetic lethal interactions\*

Edwin H. Yau, University of California San Diego, La Jolla, CA

Mechanistic participation of cancer testis antigens in tumor initiation and progression Angelique Whitehurst, UT Southwestern Simmons Comprehensive Cancer Center,

Dallas, TX

CRISPRi screening with targeted therapeutics classifies functional long non-coding

RNAs in DLBCL\*

Daniel E. Webster, National Cancer Institute, Bethesda, MD

MEK inhibitors block growth of Ataxia Telangiectasia Mutated (ATM) mutant lung

tumors\*

Ferran Fece de la Cruz, Ludwig Institute for Cancer Research, University of Oxford,

Oxford, United Kingdom

Exploiting genetic deficiencies in cancer therapy

Alan Ashworth, UCSF Helen Diller Family Comprehensive Cancer Center,

San Francisco, CA

5:00 p.m. Evening off

Saturday, January 7, 2017

7:00 a.m.-8:00 a.m. Breakfast

The Edge

<sup>\*</sup>Short talk from proffered abstract

8:00 a.m.-10:00 a.m.

### **Plenary Session 7: Chemical Biology**

Legends Ballroom 1-4

Session Chair: Sourav Bandyopadhyay, UCSF Helen Diller Family Comprehensive

Cancer Center, San Francisco, CA

Chemical-genetic interaction maps for precision therapies in breast and ovarian cancers

Sourav Bandyopadhyay

Rho-associated kinase 1 inhibition is synthetically lethal with von Hippel-Lindau deficiency in clear cell renal cell carcinoma\*

Olga V. Razorenova, University of California, Irvine, CA

Systematic, network-based elucidation of synthetic lethal proteins and synergistic compounds

Andrea Califano, Columbia University, New York, NY

A druggable transcriptional vulnerability in NRF2-dependent lung cancer\* Liron Bar-Peled, The Scripps Research Institute, La Jolla, CA

Exploiting the heterogeneity of mutant Kras lung tumors to improve therapy Carla Martins, University of Cambridge, Cambridge, England

10:00 a.m.-10:30 a.m.

#### **Break**

Abbey Road

#### 10:30 a.m.-12:30 p.m.

### **Plenary Session 8: Resistance Against Drug Combinations**

Legends Ballroom 1-4

Session Chair: Ryan B. Corcoran, Massachusetts General Hospital Cancer Center, Boston, MA

Overcoming drug resistance and tumor heterogeneity in gastrointestinal cancers Ryan B. Corcoran

Single-cell analysis reveals an adaptive, slowly-dividing, de-differentiated, drug-resistant cell state selectively inhibitable by drug combinations\*
Mohammad Fallahi-Sichani, Harvard Medical School, Boston, MA

PRMT5 as a therapeutic target in MTAP-deleted cancers Frederick H. Wilson, Dana-Farber Cancer Institute, Boston, MA

Leveraging synthetic lethality to target convergent therapeutic resistance\* Kris Wood, Duke University, Durham, NC

To be announced

Neal Rosen, Memorial Sloan Kettering Cancer Center, New York, NY

Not designated for CME

#### 12:30 p.m.

#### **Closing Remarks**

<sup>\*</sup>Short talk from proffered abstract