# Register Today!

**September 24 - 25, 2013**

*Inaugural*

## Targeting Epigenetic Readers

*Second Annual*

## Targeting Histone Methyltransferases

**September 25 - 26, 2013**

*Seventh Annual*

## Next-Generation Histone Deacetylase Inhibitors

*Inaugural*

## Targeting Histone Demethylases

### 4 Epigenetic Targets and Therapies Events Feature:

- 3 Interactive Short Courses
- 60+ Speakers, Interactive Session and Panel Discussions
- Breakout Discussion Groups
- Exhibit Hall and Dedicated Poster Viewings
- Ample Networking Opportunities: Meet with Pharma, Biotech and Academic Stakeholders to Gain Strategic Insights into the Discovery and Development of Epigenetic Inhibitors

PART OF

11th Annual Discovery on TARGET

DiscoveryOnTarget.com

**September 24 - 26, 2013**

Westin Boston Waterfront, Boston, MA

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### PLENARY KEYNOTE SPEAKERS

**Towards a Patient-Based Drug Discovery**

**Stuart L. Schreiber, Ph.D.,** Director, Chemical Biology, Founding Member, Broad Institute of Harvard and MIT; Howard Hughes Medical Institute Investigator; Morris Loeb Professor of Chemistry and Chemical Biology, Harvard University

**Enteroendocrine Drug Discovery for Treatment of Metabolic Diseases**

**Paul L. Feldman, Ph.D.,** Senior Vice President, GlaxoSmithKline

### FEATURED SPEAKERS:

**Cheryl H. Arrowsmith,** Ph.D., Chief Scientist, Structural Genomics Consortium; Professor, Medical Biophysics; Canada Research Chair, StructuralProteomics, University of Toronto

**James E. Bradner,** M.D., Assistant Professor, Department of Medicine, Harvard Medical School and Investigator, Department of Medical Oncology, Dana-Farber Cancer Institute

**Peter J. Tummino,** Ph.D., Head, Biology, Cancer Epigenetics Discovery Performance Unit, Oncology R&D, GlaxoSmithKline Pharmaceuticals

Organized by
CHI Cambridge Healthtech Institute

## About the Epigenetic Drug Discovery Events:

Within the past few years, an expanding collection of epigenetic modulators - spanning multiple classes and disease implications - have been positioned as promising targets for therapeutic intervention. Since the approval of first-generation epigenetic therapies, a growing number of next-generation inhibitors targeting Histone Deacetylases (HDACs), Histone Methyltransferases (HMTs), Histone Demethylases (HDMs), and a distinct set of chromatin readers such as the BET family bromodomains, are currently being tested in preclinical and clinical stages. Collectively, this robust expanse of epigenetic targets has given rise to a new era in drug discovery, providing viable and much needed avenues for continued development of novel medicines. Cambridge Healthtech Institute is proud to host the industry's leading translational epigenetic congress, designed with complementary tracks covering the full landscape of the rapidly advancing arena of epigenetic targets and therapies. Join pharmaceutical, biotech and academic stakeholders September 24-26 in Boston, for interactive sessions, panel discussions and short courses all geared toward providing opportunities for active networking and collaborating, while gaining strategic insights into the discovery and development of epigenetic inhibitors.

For complete Epigenetic Drug Discovery Event details visit: **DiscoveryOnTarget.com/Epigenetic-Targets**

## Plenary Keynote Speakers

**Towards a Patient-Based Drug Discovery**

*Stuart L. Schreiber, Ph.D., Director, Chemical Biology, Founding Member, Broad Institute of Harvard and MIT; Howard Hughes Medical Institute Investigator; Morris Loeb Professor of Chemistry and Chemical Biology, Harvard University*

**Enteroendocrine Drug Discovery for Treatment of Metabolic Diseases**

*Paul L. Feldman, Ph.D., Senior Vice President, GlaxoSmithKline*

## Recommended Short Courses*

**MONDAY, SEPTEMBER 23 | 12:00 – 3:00 PM**

**SC3: Biochemical and Structure-Based Approaches to Epigenetic Drug Discovery**

*Instructors:*
*David Sheppard, Ph.D., Director, Computational Chemistry, BioFocus*
*Zhaohui Sunny Zhou, Ph.D., Faculty Fellow, Barnett Institute of Chemical and Biological Analysis; Associate Professor, Department of Chemistry and Chemical Biology, Northeastern University*
*Alan P. Graves, Ph.D., Investigator, Platform Technology and Sciences, GlaxoSmithKline*
*Dmitri Kireev, Ph.D., Director, Computational Drug Discovery Center for Integrative Chemical Biology and Drug Discovery Research Professor, University of North Carolina at Chapel Hill*
*Rich Cummings, Ph.D., Director, Lead Discovery, Constellation Pharmaceuticals*

**MONDAY, SEPTEMBER 23 | 3:30 – 6:30 PM**

**SC8: Characterization and Quantification of Histone Modifications**

*Instructors:*
*Alan Tackett, Ph.D., Associate Professor, Director UAMS Proteomics Facility, University of Arkansas for Medical Sciences*
*Sean Taverna, Ph.D., Assistant professor, Pharmacology & Molecular Sciences, IBBS Center for Epigenetics, Johns Hopkins University School of Medicine*
*Yingming Zhao, Ph.D., Professor, Cancer Research, University of Chicago*

**WEDNESDAY, SEPTEMBER 25 | 6:45 – 9:30 PM**

**Dinner will be provided**

**SC10: Tools for Detection and Utilization of Epigenetic Markers**

*Instructors:*
*Pamela Munster, M.D., Professor of Medicine, Director of Early Phase Clinical Trials' Program and Associate Director of Investigational Therapeutics, University of California, San Francisco*
*Sophie Lelièvre, D.V.M., LL.M., Ph.D., Associate Professor, Department of Basic Medical Sciences; Associate Director, Discovery Groups, NCI-Designated Purdue Center for Cancer Research, Purdue University*
*Jack Zhongyi Cheng, Ph.D., CEO, PTM Biolabs, Inc.*

**Separate Registration Required for Short Courses*

# Conference-at-a-Glance

| | | | | | | |
|---|---|---|---|---|---|---|
| Mon. Sept. 23 | **Pre-Conference Short Courses*** | | | | | |
| | SC3: Biochemical and Structure-Based Approaches to Epigenetic Drug Discovery<br>SC8: Characterization and Quantification of Histone Modifications | | | | | |
| Tues. Sept. 24 | Targeting Epigenetic Readers | Targeting Histone Methyltransferases | GPCR-Based Drug Discovery | Functional Genomics Screening Strategies – Part 1 | Novel Strategies for Kinase Inhibitors | Antibodies Against Membrane Protein Targets – Part 1 |
| Wed.AM Sept. 25 | Targeting Epigenetic Readers | Targeting Histone Methyltransferases | GPCR-Based Drug Discovery | Functional Genomics Screening Strategies – Part 1 | Novel Strategies for Kinase Inhibitors | Antibodies Against Membrane Protein Targets– Part 1 |
| Wed. PM Sept. 25 | Next-Generation Histone Deacetylase Inhibitors | Targeting Histone Demethylases | GPCR-Targeted Therapeutic | Functional Genomics Screening Strategies – Part 2 | Cardio-Metabolic Drug Targets | Antibodies Against Membrane Protein Targets– Part 2 |
| 6:45 - 9:30 PM | **Conference Short Course*** | | | | | |
| | SC10: Tools for Detection and Utilization of Epigenetic Markers | | | | | |
| Thurs. Sept. 26 | Next- Generation Histone Deacetylase Inhibitors | Targeting Histone Demethylases | GPCR-Targeted Therapeutics | Functional Genomics Screening Strategies – Part 2 | Cardio-Metabolic Drug Targets | Antibodies Against Membrane Protein Targets– Part 2 |

**Separate Registration Required.*

## Present a poster and save $50!

Cambridge Healthtech Institute encourages attendees to gain further exposure by presenting their work in the poster sessions. To secure a poster board and inclusion in the conference materials, your abstract must be submitted, approved and your registration paid in full by August 16, 2013. Please see registration page for details.

**Reasons you should present your research poster at this conference:**

- Your poster will be exposed to our international delegation
- Receive $50 off your registration
- Your poster abstract will be published in our conference materials
- Your research will be seen by leaders from top pharmaceutical, biotech, academic and government institutes

**"In one day I wrote more notes than in full week-long conferences. Truly the state-of-the-art science at CHI!"**

**Assistant Professor, U. of Connecticut**

MEDIA PARTNERS

# Targeting Epigenetic Readers

Inhibiting Chromatin Readers - from Bromodomains to Beyond

**» SUGGESTED EVENT PACKAGE**

September 23: Biochemical and Structure-Based Approaches to Epigenetic Drug Discovery **Short Course 3**
September 23: Characterization and Quantification of Histone Modifications **Short Course 8**
September 24-25: Targeting Epigenetic Readers **Conference**
September 25-26: Next-Generation Histone Deacetylase Inhibitors **Conference**

## TUESDAY, SEPTEMBER 24

**7:00 am Registration and Morning Coffee**

## FEATURED SESSION: LEADERS IN EPIGENETIC DRUG DISCOVERY

**8:10 Chairperson's Opening Remarks**

**8:15 Drugging the Epigenome**

*Cheryl H. Arrowsmith, Ph.D., Chief Scientist, Structural Genomics Consortium; Professor, Medical Biophysics; Canada Research Chair, Structural Proteomics, University of Toronto*

The SGC is taking a protein family approach to understand the network of human proteins that deposit, recognize and remove acetyl and methyl marks on histones and non-histone proteins. We are generating potent, selective and cell-active antagonists of epigenetic regulatory proteins for use in target validation and biological discovery. I will discuss chemical tractability of these protein families and their potential for new classes of therapeutics.

**9:00 Drugging the Epigenome in Cancer**

*Peter J. Tummino, Ph.D., Head, Biology, Cancer Epigenetics Discovery Performance Unit, Oncology R&D, GlaxoSmithKline Pharmaceuticals*

Over the past several years, there is increasing evidence of epigenetic dysregulation in cancer. The writer, eraser, and reader proteins of histone epigenetic marks have become targets for small-molecule therapeutic intervention. A perspective on both the challenges and opportunities for drug discovery and development against these classes of targets will be presented, exemplifying GSK's experience with specific targets.

**9:45 Grand Opening Coffee Break in the Exhibit Hall with Poster Viewing**

## NOVEL PROBES AND INHIBITORS: MECHANISTIC INSIGHTS INTO TARGET VALIDATION

**10:45 Targeting Bromodomains in NUT Midline Carcinoma**

*Christopher A. French, M.D., Assistant Professor, Department of Pathology, Harvard Medical School*

I will first discuss the basic clinical aspects of NUT midline carcinoma, including its diagnosis and clinical behavior. Then, two mechanisms by which the causative BRD-NUT oncoproteins appear to block differentiation in this cancer. Finally, how we target these mechanisms using HDAC & BET bromodomain inhibitors.

**11:15 Sponsored Presentation** *(Opportunity Available)*

**11:45 CHD5 and H3: A Must-Read for Tumor Suppression**

*Alea A. Mills, Ph.D., Professor & Team Leader, Cold Spring Harbor Laboratory*

CHD5 binds unmodified histone 3 via its dual PHDs.This interaction is essential for CHD5 to modulate transcription, to inhibit proliferation, and to induce differentiation. Whereas wild type CHD5 inhibits tumor growth, mutations that perturb the CHD5:H3 interaction abolish CHD5's tumor suppressive activity, leading to enhanced tumorigenesis *in vivo*.

**12:15 pm Talk Title to be Announced (Work Currently Undisclosed)**

*John Trzupek, Ph.D., MBA, Principal Scientist, Biotherapeutics, External Chemistry Innovation, Pfizer*

**12:45 Luncheon Presentation** *(Sponsorship Opportunity Available)* **or Lunch on Your Own**

## PROGRESS TOWARDS NOVEL CHEMICAL READER ANTAGONISTS

**2:15 Chairperson's Opening Remarks**

**2:20 From Epigenetic Mechanism to Targeted Therapy**

*Ming-Ming Zhou, Ph.D., Professor & Chairman, Department of Structural & Chemical Biology, Icahn School of Medicine at Mount Sinai*

I will present the latest structural and mechanistic study of protein-protein interactions involving key transcription factors as well as core histones that are essential for gene transcriptional activation in chromatin. I will also discuss the functional implications of their new findings of the basic principles that govern the molecular interactions and regulation in gene expression, and a new strategy for developing targeted epigenetic therapy for cancer and chronic inflammation.

**2:50 Promoting Illiteracy: Inhibition of Methyl-Lysine Readers by Small Molecule Chemical Probes**

*Lindsey Ingerman James, Ph.D., Assistant Professor, Center for Integrative Chemical Biology & Drug Discovery, University of North Carolina, Chapel Hill*

I will describe efforts towards the discovery of UNC1215, a potent and selective probe for the methyl-lysine reading function of L3MBTL3. The potency, specificity, and cellular effects of UNC1215 establishes it as the first cell-active methyl-lysine reader anatagonist.

**3:20 Targeting Selective Inhibition of BET Proteins by Context Specific Engagement of Tandem Bromodomains with Coferons**

*Sponsored by* Coferon

*Lee Arnold, Ph.D., Vice President & CSO, Coferon Inc.*

Coferon's proprietary bioorthogonal chemistries enable self-assembly of large dimeric molecules upon macromolecular targets inside the cell. BET proteins use tandem bromodomains to bind acetylated histone proteins and direct a wide range of biological responses. BRD4 alone is linked to cancer, immunologic and cardiovascular outcomes, inferring a need for selective therapeutics. We have identified coferons that engage both bromodomains of BRD4 in different spatial orientations in cells, allowing us to probe the effects of selective inhibition.

**3:50 Refreshment Break in the Exhibit Hall with Poster Viewing**

**4:30 Disrupting the Reader**

*John M. Denu, Ph.D., Director, Epigenetics Theme, Wisconsin Institute for Discovery, University of Wisconsin*

This presentation will cover experimental strategies and assay platforms to reveal how protein readers interpret complex PTM language and how this information can be utilized to developed small-molecule disrupters (inhibitors) of readers implicated in disease.

**5:00 Histone Binding Mechanisms and Specificities of PHD Fingers**

*Tatiana Kutateladze, Ph.D., Associate Professor, Department of Pharmacology, Anschutz Medical Campus, University of Colorado*

Here, I summarize the structures and binding mechanisms of the PHD fingers that select for modified and unmodified histone H3 tails. I will compare the specificities of PHD fingers, Tudor and other histone readers, and discuss the significance of crosstalk between PTMs and the consequence of combinatorial readout for the selective recruitment of effectors to chromatin.

**5:30 Interactive Breakout Discussion Groups**

**6:30 Welcome Reception in the Exhibit Hall with Poster Viewing**

**7:30 Close of Day**

## WEDNESDAY, SEPTEMBER 25

**7:30 am Registration and Morning Coffee**

## THERAPEUTIC APPLICATIONS OF INHIBITING BET BROMODOMAINS

**8:00 Chairperson's Opening Remarks**

**8:05 Targeting MYCN with BET Bromodomain Inhibitors in Neuroblastoma**

*Kimberly Stegmaier, M.D., Associate Professor, Department of Pediatrics, Harvard Medical School; Independent Investigator, Pediatric Oncology, Dana-Farber Cancer Institute*

BET bromodomain inhibition has emerged as a promising strategy for the treatment of cancer, particularly hematological malignancies. However, biomarkers of response to this compound class have been lacking. We recently conducted high-throughput pharmacogenomic profiling of the BET bromodomain inhibitor JQ1 and identified MYCN amplification as a predictor of response.

**8:35 Bromodomain Inhibition as a Novel Therapeutic Treatment for Pulmonary Fibrosis**

*David C. Budd, Ph.D., Honorary Lecturer, Department of Inflammation, University College London Medical School*

The complex, multi-factorial mechanisms underlying the progression of Idiopathic Pulmonary Fibrosis (IPF) have hampered the identification and development of effective therapies. Inhibiting epigenetic readers, such as the Bromodomain and Extra Terminal Domain family with small molecule inhibitors may provide superior efficacy by antagonizing multiple profibrotic pathways.

**9:05 BET Proteins as Critical Links between Chronic Inflammation, Insulin-Resistant Obesity and Certain Cancers**

*Gerald V. Denis, Ph.D., Associate Professor, Cancer Research Center, Department of Pharmacology & Medicine, Boston University School of Medicine*

BET proteins are implicated in inflammation, adipogenesis/energy balance and cancer. Preliminary evidence supports a hypothesis that differential expression of certain BET proteins stratifies risk for a cluster of co-morbidities, including chronic inflammation and cancer, in insulin-resistant obesity.

**9:35 Monitoring Inhibition of Bromodomain Protein Interactions with Chromatin in Living Cells Using BRET**

*Sponsored by* Promega

*Danette L. Daniels, Ph.D., Group Leader, Functional Proteomics, Promega Corporation*

**10:05 Coffee Break in the Exhibit Hall with Poster Viewing**

**10:50 Mechanisms of BET Bromodomain Inhibition in the Control of Gene Expression**

*Robert J. Sims III, Ph.D., Senior Director, Biology, Constellation Pharmaceuticals, Inc.*

While suppression of MYC is a dominant consequence of BET bromodomain inhibition, BET proteins regulate additional genes and cancer cells display a range of responses to inhibitors. We have characterized the molecular impact of bromodomain inhibition, specifically in the context of chromatin re-organization and transcriptional control. We have examined both *de novo* and acquired resistance. Highly potent and selective BET inhibitors, optimized for clinical development, will also be discussed.

**11:20 Identification of Potent, BET Bromodomain Inhibitors for Treatment of Cancers**

*Hosahalli Subramanya, Ph.D., Senior Vice President, Structural Biology & Lead Generation, Aurigene Discovery Technologies, Ltd.*

This presentation covers strategies for identification of novel BET Bromodomain inhibitors using structure based drug design. Biochemical and cellular activity of multiple distinct series of compounds, and data from PK/PD and efficacy studies in xenograft models will be presented.

**11:50 Enjoy Lunch on Your Own**

**1:40 pm PLENARY KEYNOTE PRESENTATIONS**
*See Page 2 for Information*

**3:10-3:50 Refreshment Break in the Exhibit Hall with Poster Viewing**

**3:50 Close of Conference**

*Second Annual*

# Targeting Histone Methyltransferases

Advances in Second-Generation Epigenetic Therapies

**September 24 - 25, 2013**

**» SUGGESTED EVENT PACKAGE**

September 23: Biochemical and Structure-Based Approaches to Epigenetic Drug Discovery **Short Course 3**
September 23: Characterization and Quantification of Histone Modifications **Short Course 8**
September 24-25: Targeting Histone Methyltransferases **Conference**
September 25-26: Targeting Histone Demethylases **Conference**

## TUESDAY, SEPTEMBER 24

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**8:15 Drugging the Epigenome**

*Cheryl H. Arrowsmith, Ph.D., Chief Scientist, Structural Genomics Consortium; Professor, Medical Biophysics; Canada Research Chair, Structural Proteomics, University of Toronto*

The SGC is taking a protein family approach to understand the network of human proteins that deposit, recognize and remove acetyl and methyl marks on histones and non-histone proteins. We are generating potent, selective and cell-active antagonists of epigenetic regulatory proteins for use in target validation and biological discovery. I will discuss chemical tractability of these protein families and their potential for new classes of therapeutics.

**9:00 Drugging the Epigenome in Cancer**

*Peter J. Tummino, Ph.D., Head, Biology, Cancer Epigenetics Discovery Performance Unit, Oncology R&D, GlaxoSmithKline Pharmaceuticals*

Over the past several years, there is increasing evidence of epigenetic dysregulation in cancer. The writer, eraser, and reader proteins of histone epigenetic marks have become targets for small-molecule therapeutic intervention. A perspective on both the challenges and opportunities for drug discovery and development against these classes of targets will be presented, exemplifying GSK's experience with specific targets.

**9:45 Grand Opening Coffee Break in the Exhibit Hall with Poster Viewing**

## STATE OF THE ART IN HMT PROBES & INHIBITORS

**10:45 A First-in-Class Chemical Probe for SETD7**

*Dafydd Owen, Ph.D., Associate Research Fellow, Medicinal Chemistry, Worldwide R&D, Pfizer*

Our program of pre-competitive chemical probe generation in collaboration with the SGC has delivered a first-in-class, highly HMT-selective, chemical tool for SETD7 that is free from restriction on use and available to all. The discovery, characterization, chemical biology and cellular phenotypes related to this compound will be described.

**11:15 Epigenetic Target Specificity and the Discovery of Epigenetic-Related *in vivo* Adverse Drug Reactions**

*Sponsored by* Cerep

*Manilduth Ramnath, Ph.D., Project Manager, Custom Services and Innovation, Cerep*

A sub-set of 1,000 pharmaceutically active molecules from the BioPrint® database were screened against five histone methyltransferases, five histone demethylases, three histone deacetylases, and three bromodomains. Certain classes of pharmacological compounds were found to modulate the activity of epigenetic proteins related to known adverse drug reactions.

**11:45 Targeting Histone Methyltransferases in Cancer Therapy**

*Sarah Knutson, Ph.D., Senior Scientist, Biological Sciences, Epizyme*

Epizyme has synthesized potent and selective small molecule inhibitors of several HMTs, including EZH2. The properties of such inhibitors, including their ability to selectively kill tumor cells bearing specific genetic alterations in cell culture and animal models, will be discussed.

**12:15 pm Therapeutic Applications of EZH2 Small Molecule Inhibitors**

*Patrick Trojer, Ph.D., Senior Director & Head, Biology, Constellation Pharmaceuticals*

Constellation has discovered and developed EZH2 and EZH1 small molecule inhibitors with *in vitro* and *in vivo* efficacy in various cancer models. We have established a good PK-PD-efficacy relationship and examined molecular consequences of target engagement.

**12:45 Elimination of Serial Dilution to Improve Dose-Response Analyses**

*Sponsored by* hp

*Ken Ward, Ph.D., Pharma Product Development R&D, Hewlett-Packard Company*

Placing the appropriate number of picoliter droplets of compound stock directly into assay-ready plates allows the direct formation of doses without the need for serial dilution. This non-contact method also creates better dose-response curves by enabling randomization of doses throughout plates to reduce edge effects and through the use of finely space doses to improve estimates of IC50s. These improvements will be illustrated using results from the numerous pharma labs now using this new technology.

## STATE OF THE ART IN HMT PROBES & INHIBITORS (CONT.)

**2:15 Chairperson's Opening Remarks**

**2:20 Targeting H3K4 Methylation by the MLL1 Complex for the Treatment of Mixed Lineage Leukemia**

*Yali Dou, Ph.D., Associate Professor, Pathology,*

*University of Michigan*

Here we report the development of a potent and specific small-molecule inhibitor (MM-401) for MLL1 activity. Using MM-401, we demonstrate the selective inhibition of MLL1 activity by blocking the assembly of the MLL1 complex. Furthermore, MM-401 shows high efficacy against MLL leukemia through inducing apoptosis, cell cycle arrest and myeloid differentiation.

**2:50 Small Molecule Epigenetic Intervention of Disease via Histone Lysine Methyltransferases**

*Matthew Fuchter, Ph.D., Senior Lecturer, Synthetic and Medicinal Chemistry, Department of Chemistry, Imperial College London*

Natural and synthetic inhibitor classes will be highlighted, focusing on the mechanism of action of these inhibitors, as well as the design and synthesis of novel analogues. Our strategy for the identification and progression of inhibitors targeting the H3K27me3 mark in cancer using cell-based (MDA-MB-231) assays will be discussed. We have identified novel small molecules that induce re-expression of genes, reverse H3K27me3 mediated gene silencing and induce inhibition of tumor cell growth.

**3:20 Sponsored Presentation** *(Opportunity Available)*

**3:50 Refreshment Break in the Exhibit Hall with Poster Viewing**

**4:30 Harnessing EZH2 Enzymatic Activities by Targeting PRC2 Associated Cofactors**

*Gang "Greg" Wang, Ph.D., Assistant Professor, Department of Biochemistry & Biophysics, University of North Carolina, Chapel Hill*

Recently, we have identified a novel PRC2-regulatory mechanism where PHF1 or PHF19 directs PRC2 chromatin-association and promotes EZH2 enzymatic activities. Targeting PRC2-associated cofactors represents an alternative approach to harness PRC2 enzymatic activities, in addition to the recently developed small-molecule inhibitors that target EZH2 directly.

**5:00 Targeting the Histone Methyltransferase MMSET in Cancer**

*Irfan Asangani, Ph.D., Research Investigator, Pathology, Michigan Center for Translational Pathology*

We have molecularly ordered two HMTases, EZH2 and MMSET that have established genetic links to oncogenesis. EZH2-MMSET HMTase axis is coordinated by a microRNA network and that the oncogenic functions of EZH2 require MMSET activity. We identified 3-hydraxino-2-quinoxalinethiol (MCTP39) that attenuates MMSET activity in vivo. MCTP39 was shown to preferentially inhibit cancer cell lines that have elevated levels of MMSET as well as t(4;14) MMSET translocation positive multiple myeloma cells.

**5:30 Interactive Breakout Discussion Groups**

**6:30 Welcome Reception in the Exhibit Hall with Poster Viewing**

**7:30 Close of Day**

## WEDNESDAY, SEPTEMBER 25

**7:30 am Registration and Morning Coffee**

## APPROACHES FOR HMT INHIBITOR DEVELOPMENT

**8:00 Chairperson's Opening Remarks**

**8:05 Global Analysis of Methylation and Functional Annotation of Methyltransferases: Chemo-Enzymatic Approaches**

*Zhaohui Sunny Zhou, Ph.D., Associate Professor, Department of Chemistry and Chemical Biology, Northeastern University*

Our chemo-enzymatic approaches enables the selective tagging (e.g., derivatization) of modified amino acids based on their unique chemical reactivities or enzymatic specificities. Spectroscopic, affinity and mass tags can be judiciously incorporated to facilitate the subsequent detection and quantification. Moreover, affinity enrichment markedly reduces sample complexity.

**8:35 Hit-to-Lead Strategies for Epigenetic Targets at GSK**

*Alan P. Graves, Ph.D., Investigator, Platform Technology and Sciences, GlaxoSmithKline*

I will discuss screening strategies used at GSK to identify hits for epigenetic targets with a focus on hit discovery and lead optimization for EZH2. Structural insights used to build the EZH2 homology model and SAR that guided the predicted binding mode of our lead series will be described.

**9:05 Chemical Tractability of Protein Methyltransferases: Lessons Learned from Protein Structures and Screening Campaigns**

*Matthieu Schapira, Ph.D., Principal Investigator, Computational Chemistry, Structural Genomics Consortium*

The Structural Genomics Consortium is screening multiple chemical libraries from academic and industry partners against a large panel of protein methyltransferases. I will review hit rates observed so far, highlight resulting chemical probes and complex structures, and discuss the chemical tractability of the cofactor and substrate binding sites.

**9:35 Sponsored Presentation** *(Opportunity Available)*

**10:05 Coffee Break in the Exhibit Hall with Poster Viewing**

**10:50 Histone Methyltransferase Inhibitors Targeting Cancer**

*Yongcheng Song, Ph.D., Assistant Professor, Pharmacology, Baylor College of Medicine*

Several series of novel compounds inhibiting DOT1L as well as other histone methyltransferases were discovered and developed in our lab. The biochemical and biophysical studies of these compounds as well as their activities against MLL leukemia and other cancers will be discussed.

**11:20 The Structure and Activity of Type II Arginine Methyltransferases**

*Stephen Antonysamy, Ph.D., Principal Research Scientist, Structural Biology, Eli Lilly*

**11:50 Enjoy Lunch on Your Own**

**1:40 pm PLENARY KEYNOTE PRESENTATIONS**

*See Page 2 for Information*

**3:10-3:50 Refreshment Break in the Exhibit Hall with Poster Viewing**

**3:50 Close of Conference**

# Next-Generation Histone Deacetylase Inhibitors

## Targeting HDACs for Oncology, Inflammation, Neurodegeneration, Diabetes and Other Indications

**» SUGGESTED EVENT PACKAGE**

September 23: Characterization and Quantification of Histone Modifications **Short Course 8**
September 24-25: Targeting Epigenetic Readers **Conference**
September 25:Tools for Epigenetic Biomarker Discovery **Dinner Short Course 10**
September 25-26: Next-Generation Histone Deacetylase Inhibitors **Conference**

### WEDNESDAY, SEPTEMBER 25

**11:50 am Conference Registration**

### DESIGNING THE IDEAL INHIBITOR

**1:30 pm Chairperson's Opening Remarks**

**1:40 PLENARY KEYNOTE PRESENTATIONS**
*See Page 2 for Details*

**3:10-3:50 Coffee Break in the Exhibit Hall with Poster Viewing**

**3:50 Chairperson's Opening Remarks**

**4:00 FEATURED PRESENTATION: Targeting Lysine Acetylation in Human Disease**

*James E. Bradner, M.D., Assistant Professor, Department of Medicine, Harvard Medical School and Investigator, Department of Medical Oncology, Dana-Farber Cancer Institute*

Sidechain acetylation of lysine has emerged as a prevalent post-translational modification mediating cell signaling pathways of broad relevance to developmental and disease biology. This presentation will review the history of lysine acetylation, the development of first and next-generation histone deacetylase inhibitors, as well as the recent devevelopment of direct-acting bromodomain inhibitors for cancer therapy. Particular emphasis will be placed on new insights into chromatin biology revealed through the lens of chemical biology, *in vitro* and *in vivo*.

**4:30 Selective Bioluminescent HDAC Assays for Cell-Based Drug Development**

*Sponsored by*

Promega

*Andrew L. Niles, Senior Research Scientist, Promega Corporation*

**5:00 Sirtuins: Aging, Diseases and Circadian Control**

*Leonard P. Guarente, Ph.D., Novartis Professor of Biology, Harvard University*

In this talk, I will describe how mamamlian SIRT1 impacts tissue maintenance and diseases of aging by deacetyling nuclear transcription factors that govern key physiological pathways. Moreover, I will also describe new data showing the importance of central circadian control in the brain in mouse longevity studies. I will also show how SIRT1 mediates circadian control in the brain by deacetylating the central pacemaker BMAL, and demonstrate that this regulation of circadian proteins declines with normal aging in mice.

**5:30 Chemogenomic Approaches to Spatiotemporal Regulation of HDAC Activity**

*Ralph Mazitschek, Ph.D., Assistant Professor, Center for Systems Biology, Chemical Biology Platform, Massachusetts General Hospital*

HDAC inhibitors have been used as tool compounds to study basic biology and recognized as promising therapeutics for the treatment of cancer and beyond. However, systemic exposure is often not well tolerated, or does not provide the required resolution in biological model systems. To address these shortcomings we have developed a new approach to control HDAC activity with greater spatial and temporal resolution.

**6:00 Novel Lysine Acylation Pathways and Acetylation-Independent Mechanisms of HDACs**

*Yingming Zhao, Ph.D., Professor, The Ben May Department for Cancer Research, University of Chicago*

We have previously identified several new lysine acylation pathways: lysine propionylation, lysine butyrylation, lysine crotonylation, lysine malonylation, and lysine succinylation. We now report the identification and characterization of the regulatory enzymes that can modulate status of these PTMs. We will also report proteomics screenings that identify the substrates for the new lysine acylations and their regulatory enzymes.

**6:30 Close of Day**

### THURSDAY, SEPTEMBER 26

**7:30 am Registration**

### HDACi FOR CARDIOVASCULAR INDICATIONS

**8:00 Breakfast Interactive Breakout Discussion Groups**

**9:05 Chairperson's Opening Remarks**

**9:10 HDAC Inhibitors for the Treatment of Pathological Muscle Remodeling**

*Timothy A. McKinsey, Ph.D., Associate Professor and Associate Division Head for Translational Research, Department of Medicine, Division of Cardiology, University of Colorado Denver*

Preclinical findings have suggested unforeseen potential for small molecule HDAC inhibitors for the treatment of heart failure and associated conditions. However, since broad-spectrum, 'pan' HDAC inhibition is associated with

toxicities such as thrombocytopenia, many remain skeptical of the prospects of translating these findings to the clinic. I will highlight roles of HDACs in striated muscle and the therapeutic potential of isoform-selective HDAC inhibitors for the treatment of pathological muscle remodeling.

**9:40 HDAC Inhibition to Target Heart Disease**

*Joseph Hill, M.D., Ph.D., Professor, Internal Medicine and Molecular Biology; Chief of Cardiology, University of Texas Southwestern Medical Center; Director, Harry S. Moss Heart Center*

Gene deletion and over-expression studies have revealed important functions of enzymes involved in controlling protein acetylation in pathological cardiac remodeling, including ventricular hypertrophy, apoptosis, necrosis, metabolism, contractility, and fibrosis. Further, pre-clinical studies have revealed that pharmacological inhibition of HDACs can blunt pathological change induced by pressure overload or ischemia/reperfusion injury. We will review these data.

**10:10 Coffee Break in the Exhibit Hall with Poster Viewing**

**10:55 HDAC Inhibition and Cardiac Protection**

*Ting Zhao, M.D., Associate Professor, Department of Surgery, Roger Williams Medical Center, Boston University Medical School*

HDAC inhibition increased the improvement of myocardial functional recovery in infarcted heart, prevented myocardial remodeling and enhanced the formation of new myocytes and microvessels. Re-introduction of TSA-treated cardiac progenitor cells into MI heart restored myocardial functional improvement. Results indicate that HDAC inhibition preserves cardiac performance and stimulates cardiac endogenous regeneration.

## HDACi FOR CNS INDICATIONS

**11:25 HDACi in the Treatment of Muscular Dystrophies: Targeting Cellular and Molecular Networks that Control Muscle Repair**

*Puri Pier Lorenzo, M.D., Ph.D., IRCCS Fondazione Santa Lucia, Pharmacology and Epigenetics, Rome, Italy; Associate Professor, Sanford-Burnham Institute for Medical Research*

Our studies revealed that HDAC inhibitors (HDACi) exert a beneficial effect in mouse models of muscular dystrophies, by promoting compensatory regeneration and preventing the formation of fibrotic scars and fatty deposition. Currently, HDACi are being tested in clinical trials on patients affected by Duchenne Muscular Dystrophies. We will present our latest studies on the cell types and the intracellular networks that mediate the therapeutic effects of HDACi in dystrophic muscles.

**11:55 Regulation of Excitatory and Inhibitory Synaptic Functions by HDAC2**

*Qiang Zhou, Ph.D., Scientist, Department of Neuroscience, Genentech, Inc.*

We found that HDAC2 levels affect excitatory and inhibitory synaptic functions in opposite manner – HDAC2 suppresses excitatory but enhances inhibitory synaptic transmission, in a cell-autonomous fashion. Regulating the abundance of postsynaptic GABAA receptors underlies the impact of HDAC2 on inhibitory synaptic transmission. We suggest that by influencing the balance between synaptic excitation and inhibition, HDAC2 contributes to the functions and diseases of the nervous system.

**12:25 pm Sponsored Presentation** *(Opportunity Available)*

**12:55 Luncheon Presentation** *(Sponsorship Opportunity Available)* **or Lunch on Your Own**

## HDACi FOR TARGETING IMMUNE AND METABOLIC DISORDERS

**2:25 Chairperson's Opening Remarks**

**2:30 Mechanisms By Which the HDAC3 Inhibitor BRD3308 Reduces Insulin Resistance *in vivo***

*Gerald Shulman, M.D., Ph.D., Professor of Internal Medicine & Cellular and Molecular Physiology, Yale University; Investigator, Howard Hughes Medical Institute*

In this presentation I will review recent studies that examine the cellular and molecular mechanisms by which the HDAC3 inhibitor BRD3308 reduces insulin resistance and hyperglycemia in rodent models of non-alcoholic fatty liver disease and type 2 diabetes.

**3:00 Inhibition of HDAC3 Protects Beta-Cell Function**

*Bridget K. Wagner, Ph.D., Director, Pancreatic Cell Biology, Chemical Biology Program, The Broad Institute of MIT and Harvard*

Pancreatic beta-cell failure is central to the etiologies of both type 1 (T1D) and type 2 diabetes (T2D). Chemical compounds that promote beta-cell survival and function may therefore be of potential clinical benefit to patients with T1D or T2D. HDAC inhibitors have shown some promise in rodent models of diabetes. Here, I discuss the role of HDAC3 in beta-cell apoptosis, and the use of a selective HDAC3 inhibitor to promote beta-cell survival.

**3:30 Ice Cream Refreshment Break in the Exhibit Hall with Poster Viewing**

**4:00 Design of Class I Isoform Selective Inhibitors for Use in Metabolic Indications**

*Edward Holson Ph.D., Director, Medicinal Chemistry, Stanley Center for Psychiatric Research, The Broad Institute of MIT and Harvard*

**4:30 HDAC6 and Immune Sexual Dimorphism: New Approaches to Autoimmunity**

*Wayne W. Hancock, M.D., Ph.D., Professor of Pathology and Chief of Transplant Immunology, Children's Hospital of Philadelphia and University of Pennsylvania*

Between puberty and menopause, the incidence of most autoimmune diseases is markedly higher in females than males. We find that the suppressive functions of Foxp3+ Tregs are significantly less in females vs. males, and that pharmacologic inhibition of the estrogen receptor boosts Treg function. We will discuss *in vitro* and *in vivo* data from murine and clinical studies indicating the key role that HDAC6 targeting has in boosting Treg function especially in females, and protecting against development and progression of autoimmunity, without inducing undesired global impairment of immune functions.

**5:00 Immuno-Modulatory Activity of HDAC Inhibitors**

*Tso-Pang Yao, Ph.D., Associate Professor, Department of Pharmacology and Cancer Biology, Duke University*

The growing appreciation of versatile functions of HDAC family members presents both a challenge and opportunity for developing therapeutic utilities of HDAC inhibitors. I will discuss the biology and potential utility of HDAC inhibitors in the immune response.

**5:30 A Novel Zinc Binding Group Enables Selective Class IIa HDAC Inhibition and Alters Immune Responses**

*Mercedes Lobera, Ph.D., Head, Chemistry, Tempero Pharmaceuticals, Inc.*

**6:00 Close of Conference**

# Targeting Histone Demethylases

An Emerging Class of Epigenetic Targets

**» SUGGESTED EVENT PACKAGE**

September 23: Biochemical and Structure-Based Approaches to Epigenetic Drug Discovery **Short Course 3**
September 23: Characterization and Quantification of Histone Modifications **Short Course 8**
September 24-25: Targeting Histone Methyltransferases **Conference**
September 25-26: Targeting Histone Demethylases **Conference**

## WEDNESDAY, SEPTEMBER 25

**11:50 am Registration**

**1:30 pm Chairperson's Opening Remarks**

**1:40 PLENARY KEYNOTE PRESENTATIONS**
*See Page 2 for Details*

**3:10-3:50 Refreshment Break in the Exhibit Hall with Poster Viewing**

### TOWARDS DISCOVERY OF HIGH-QUALITY DEMETHYLASE PROBES

**3:50 Chairperson's Opening Remarks**

**4:00 Targeting H3K9me2 Writers and Erasers**

*Xiaodong Cheng, Ph.D., Professor of Biochemistry & Georgia Research Alliance Eminent Scholar, Emory University School of Medicine*

I will discuss the design of potent inhibitors of H3K9me2 writers (G9a/GLP) and erasers (PHF8/KIAA1718) by adding a lysine or methyl-lysine mimic.

**4:30 Sponsored Presentations** *(Opportunities Available)*

**5:00 Structure and Specificity of JMJD2 Histone Demethylases**

*Raymond C. Trievel, Ph.D., Associate Professor of Biological Chemistry, University of Michigan Medical School*

We determined the crystal structure of a JMJD2D:H3K9me3 peptide complex and compared it to structures of JMJD2A bound H3K9me3 and H3K36me3 peptides. These structural comparisons coupled with kinetics analysis of JMJD2A and JMJD2D demonstrated that subtle variations in the histone binding clefts of the JMJD2 demethylases impart substantial differences in their respective methylation site specificities. These studies will inform the design of inhibitorstargeting JMJD2-linked cancers.

**5:30 Strategies for Identifying New Chemical Probes for Histone Lysine Demethylases**

*Brian Lohse, Ph.D., Associate Professor, Drug Design and Pharmacology, University of Copenhagen*

Here we present past, present and future work in our group, with focus on discovery and synthesis of new chemical probes and inhibitors for the histone lysine demethylases KDM4 and LSD. The work includes small molecules, substrate-based inhibitors and DNA-encoded peptide libraries, to obtain selective inhibitors.

**6:00 Nitric Oxide is an Endogenously Produced Epigenetic Regulatory Molecule**

*Douglas Thomas, Ph.D., Associate Professor, Medicinal Chemistry, University of Illinois at Chicago*

We have recently demonstrated 3 novel and distinct mechanisms whereby the free radical nitric oxide (NO) can affect histone methylation patterns: direct inhibition of JMJC-demethylase activity, reduction in iron cofactor availability, and regulation methyl-modifying enzyme gene expression. This model is the first description of NO as an endogenously produced epigenetic regulatory modulator and provides a novel explanation for non-classical gene regulation by NO.

**6:30 Close of Day**

## THURSDAY, SEPTEMBER 26

**7:30 am Registration**

### CLINICAL INSIGHTS INTO TARGETED THERAPY

**8:00 Breakfast Interactive Breakout Discussion Groups**

**9:05 Chairperson's Opening Remarks**

**9:10 Chromatin Regulators as Therapeutic Targets in Breast Cancer**

*Kornelia Polyak, M.D., Ph.D., Associate Professor, Department of Medicine, Harvard Medical School*

I will discuss histone demethylases as lineage-specific oncoogenes in breast cancer.

**9:40 Chromatin Modulators Provide a New Insight into Cancer Genomes**

*Johnathan R. Whetstine, Ph.D., Assistant Professor, Medicine, Harvard Medical School and Massachusetts General Hospital Cancer Center*

To date, enzymes that are capable of promoting site-specific copy number changes have yet to be identified. We have recently been able to demonstrate that H3K9/36me3 lysine demethylase KDM4A overexpression leads to localized copy gains without global chromosome instability. The copy gain occurs within a single cell cycle, requires S-phase and is not stable but regenerated each cell division. The regions with increased copy number are re-replicated and have increased KDM4A, MCM and DNA polymerase occupancy. Suv39h1/KMT1A or HP1γ overexpression suppresses the copy gain, while H3K9/K36 methylation interference promotes gain. Furthermore, analysis of tumors with KMD4A amplification supported these observations. Our results demonstrate that overexpression of a chromatin modifier results in site-specific copy gains. These data begin to establish how copy number changes could originate during tumorigenesis and demonstrate that transient overexpression of chromatin

modulators could promote copy change.

**10:10 Coffee Break in the Exhibit Hall with Poster Viewing**

**10:55 Epigenetic Therapy for Cancer Treatment**

*Lorraine Gudas, Ph.D., Chairman & Revlon Pharmaceutical Professor, Pharmacology and Toxicology, Pharmacology Department, Weill Cornell Medical College*

I will present data on the use of various combinations of drugs that influence the epigenome, such as histone demethylase enzymes, to treat head and neck cancer (HNSCC) in a murine model of head and neck carcinogenesis. I will also discuss some human clinical trials with these drugs.

**11:25 Epigenetic Reprogramming in Pancreatic Cancer: The Emerging Role of Histone Demethylases**

*Alexandros Tzatsos, M.D., Ph.D., Instructor, Medicine, Harvard Medical School; Assistant Geneticist, Massachusetts General Hospital Cancer Center*

We have identified members of the Jumonji-domain containing histone demethylase family as important oncogenes in the development pancreatic cancer. Gain and loss-of-function experiments coupled to genome-wide gene expression and chromatin immunoprecipitation studies led to the discovery of a key epigenetic program, through which histone demethylase KDM2B subverts cellular differentiation and sustains metabolic homeostasis, leading to tumor progression.

**11:55 Development of Histone Demethylase Inhibitors for Oncological and Neurodegenerative Disease**

*Tamara Maes, Ph.D., Co-Founder, Vice President & CSO, Oryzon Genomics*

Oryzon's LSD1 inhibitors were shown to selectively abrogate the clonogenic potential of acute myeloid leukemia cells with MLL translocations, sparing the repopulating potential of normal hematopoietic stem cells. ORY-1001 is a potent, selective LSD1 inhibitor, with excellent pharmacological characteristics. ORY-1001 reduces leukemic stem cell potential, potently inhibits colony formation, overcomes the differentiation block in AML cell lines, and induces apoptosis/inhibits proliferation at sub-nanomolar concentrations in selected AML cell lines. ORY-1001 has received a positive opinion for orphan drug status for AML from the EMA and will start Phase I studies in Q4 2013.

**12:25 pm Sponsored Presentation** *(Opportunity Available)*

**12:55 Luncheon Presentation** *(Sponsorship Opportunity Available)* **or Lunch on Your Own**

## NOVEL MODULATORS & INSIGHTS INTO INHIBITION

**2:25 Chairperson's Opening Remarks**

**2:30 Targeting the Histone Demethylome**

*Udo Oppermann, Ph.D., Professor, Molecular Biology; Director, Molecular Laboratory Sciences, Botnar Research Centre; Principal Investigator, Epigenetics and Metabolism, Structural Genomics Consortium, University of Oxford*

Recent data suggest that HDMs are chemically tractable targets, and that demethylase-selective small molecules may be useful tools to dissect chromatin driven biological processes. Data will be presented to illustrate the usefulness of these tool compounds to understand demethylase involvement in oncology and stem cell biology.

**3:00 Targeting Histone Demethylation in Cancer**

*Ryan Kruger, Ph.D., Manager, Cancer Epigenetics Discovery Performance Unit, Oncology R&D, GlaxoSmithKline Pharmaceuticals*

This presentation will highlight progress GSK has made with potent selective inhibitors of LSD-1 and their use as treatment for cancer.

**3:30 Ice Cream Refreshment Break in the Exhibit Hall with Poster Viewing**

**4:00 Targeting Lysine-Specific Demethylase 1 with Polyamine Analogues to Induce Expression of Aberrantly Silenced Genes**

*Robert A. Casero, Jr., Ph.D., Professor of Oncology, The Sidney Kimmel Comprehensive Cancer Center, Johns Hopkins University School of Medicine*

I will present data demonstrating that different classes of our polyamine and oligoamine analogues are effective inhibitors of the lysine-specific demethylase 1 (LSD1/KDM1). These results indicate that treatment of tumor cells with these compounds results in the re-expression of several aberrantly silenced genes and that they are effective in inhibiting tumor cell growth, both *in vitro* and in established tumors *in vivo*.

**4:30 JIB-04 is a Novel Small Molecule Inhibitor of Jumonji Demethylases with Anti-Cancer Activity *In Vivo***

*Elisabeth Martinez, Ph.D., Assistant Professor, Pharmacology, University of Texas Southwestern Medical Center*

Here, we report the discovery and characterization of a Jumonji enzyme inhibitor, JIB-04, and its activity *in vitro*, in cells and *in vivo*. The small molecule inhibits trimethyl demethylase enzyme activity by a unique mechanism, alters the transcription of growth related genes and causes cell death specifically in cancer cells, while sparing normal cells. In mouse models of lung and breast cancer, it decreases demethylase activity, blunts tumor growth and delays cancer-induced death. We find that in human populations, patients with tumors that express high levels of Jumonji demethylases have poorer survival, defining a relevant subpopulation that may benefit from a future therapeutic intervention functionally related to JIB-04. In conclusion, we have identified and characterized the anti-cancer properties of a novel inhibitor of Jumonji histone demethylases. This is the first report of a modulator of demethylases with activity *in vivo*.

**5:00 JARID1 Demethylases as Cancer Targets**

*Qin Yan, Ph.D., Assistant Professor, Pathology, Yale University School of Medicine*

My laboratory focuses on the roles and regulatory mechanisms of the JARID1/KDM5 histone demethylases, which demethylate the active H3K4me3/2 marks. We showed with genetically engineered mouse cancer models that JARID1A loss inhibits endocrine tumor formation. We have also identified lead compounds that inhibit JARID1 enzymes. We will discuss the mechanisms by which the JARID1 enzymes promote tumorigenesis and our effort to identify their small molecule inhibitors.

**5:30 The Therapeutic Potential of Jumonji Histone Demethylase Inhibitors**

*Peter Staller, Ph.D., Director, Oncology Research, EpiTherapeutics ApS*

EpiTherapeutics is developing novel cancer drugs targeting histone demethylases and I will present our recent progress in obtaining specific and potent inhibitors of the putative oncoprotein jumonji C-domain histone H3K4 demethylase KDM5B. *In vitro* and *in vivo* data will be discussed.

**6:00 Close of Conference**

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**Discounted Room Rate: $269 s/d**

**Discounted Room Rate Cut-off Date: August 26, 2013**

Please visit our conference website or call the hotel directly to reserve your sleeping accommodations. You will need to identify yourself as a Cambridge Healthtech Institute conference attendee to receive the discounted room rate with the host hotel. Reservations made after the cut-off date or after the group room block has been filled (whichever comes first) will be accepted on a space and rate-availability basis. Rooms are limited, so please book early.

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Monday, September 23

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|---|
| SC1: New Class of Kinase Inhibitors: Covalent Modifiers |
| SC2: Practical Aspects of Structure-Based Drug Discovery with GPCRs |
| SC3: Biochemical and Structure-Based Approaches to Epigenetic Drug Discovery |
| SC4: Allosteric Modulators of GPCRs |
| SC5: Advancing Tools and Technologies for Fragment-Based Design |
| SC6: Setting Up Effective RNAi Screens: Getting From Design to Data |
| SC7: Production and Presentation of Integral Membrane Proteins for Antibody Discovery |
| SC8: Characterization and Quantification of Histone Modifications |

Wednesday, September 25

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| SC9: Setting Up Effective Functional Screens Using 3D Cell Cultures |
| SC10: Tools for Epigenetic Biomarker Discovery |

If you are unable to attend but would like to purchase the Discovery On Target CD for $750 (plus shipping), please visit DiscoveryOnTarget.com. Massachusetts delivery will include sales tax.

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| | | |
|---|---|---|
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| | | |
|---|---|---|
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| September 24 - 25 | September 25 - 26 |
|---|---|
| Track 1: Targeting Epigenetic Readers | Track 7: Next-Generation Histone Deacetylase Inhibitors |
| Track 2: Targeting Histone Methyltransferases | Track 8: Targeting Histone Demethylases |
| Track 3: GPCR-Based Drug Discovery | Track 9: GPCR-Targeted Therapeutics |
| Track 4: Functional Genomics Screening Strategies - Part 1 | Track 10: Functional Genomics Screening Strategies - Part 2 |
| Track 5: Novel Strategies for Kinase Inhibitors | Track 11: Cardio-Metabolic Drug Targets |
| Track 6: Antibodies Against Membrane Protein Targets - Part 1 | Track 12: Antibodies Against Membrane Protein Targets - Part 2 |

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