COVERAGE INCLUDES:

- microRNA in Biomarker and Diagnostic Development
- microRNA in Therapeutic Development
- microRNA in Human Development and Disease
- microRNA and Cancer Mechanism
- microRNA and Cancer Stem Cells

miRNA as Diagnostic Biomarkers and Targets for Therapeutic Development

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mRNA IN BIOMARKER AND DIAGNOSTIC DEVELOPMENT

8:40-8:45 Chairperson's Opening Remarks

8:45-9:10 miRNAs as Diagnostics and Therapeutics in Cancer

Frank Slack, Ph.D., Professor, Department of Molecular, Cellular and Developmental Biology, Yale University

MicroRNAs are small non-coding RNAs that regulate gene expression to control important aspects of development and metabolism such as cell differentiation, apoptosis and lifespan. Mi-7 encodes a microRNA implicated in human cancer. Specifically, human let-7 is poorly expressed or deleted in lung cancer, and over-expression of let-7 in lung cancer cells inhibits their growth, demonstrating a role for let-7 as a tumor suppressor in lung tissue. Let-7 is expressed in the developing mammalian lung and regulates the expression of important oncogenes implicated in lung cancer, suggesting a mechanism for let-7's involvement in cancer. We are focused on the role of let-7 and other oncomiRs in regulating proto-oncogene expression during development and cancer, and on using miRNAs to suppress tumorigenesis.

9:10-9:35 microRNAs: Biomarkers for Cancer Therapy

Glen J. Weiss, M.D., Co-Head, Lung Cancer Unit, The Translational Genomics Research Institute (TGen); Director, Thoracic Oncology, TGen Clinical Research Services at Scottsdale Healthcare

A single microRNA can impact hundreds of targets and can affect pathways controlling oncogenic processes. Data will be presented illustrating how using microRNA can impact cancer treatment decision making, the validation of microRNAs associated with resistance and/or sensitivity to chemotherapy and targeted therapy and how microRNAs could be used as therapeutics.

9:35-10:00 Developing miRNA Mimics and Biomarkers

Lee P. Lim, Ph.D., Research Fellow, Sirna Therapeutics, Merck Research Labs

Progress in understanding the biology of microRNAs has opened up a new area for oligonucleotide therapeutics, where working with ~22nt RNAs that can regulate hundreds of genes presents novel challenges and opportunities. I will describe our work exploring structures and chemistries for miRNA mimetics, as well as work on the use of miRNAs as plasma biomarkers.

10:00-10:25 Multiplexing microRNA and Protein Expression Analysis for Cancer Diagnostics

Lorenzo F. Sempere, Ph.D., Research Assistant Professor of Medicine, Department of Medicine, Dartmouth-Hitchcock Norris Cancer Center

Visualization of microRNA expression within individual cells by in situ hybridization provides an independent tool to clinically validate results of high-throughput expression profiling experiments. Here we describe a rapid and sensitive fluorescence-based assay with multiplexing capability for co-detection of microRNA and clinically relevant protein markers on formalin-fixed paraffin-embedded specimens. We provide several examples in which the cancer cells, and supportive and/or reactive microenvironment elements, are the principal source of microRNA deregulation in solid tumors. We discuss implementation of a fully automated platform from detection to expression analysis of selected biomarkers for high-throughput microRNA-based diagnostic applications.

10:25-11:00 Networking Coffee Break
microRNA in Human Disease and Development

2:00-2:30 Prognostic Value of miRNAs in Colorectal Cancer
Upender Manne, Ph.D., Associate Professor, Pathology, University of Alabama at Birmingham

Although promising results from experimental models are available, the utility of miRNAs should be validated in order to develop a miRNA-based therapy for colorectal cancers (CRCs). We have demonstrated that miRNAs are stable in archival CRC samples stored for up to 28 years, and analyses of a panel of six miRNAs suggest that, after treatment for CRC, patients with higher levels, specifically miRNA-21 and miR-10a, had an increased risk of death. However, for African-American patients, not for Caucasians, the presence of higher levels of miR-181b and miR-203 indicated a poorer prognosis. Further prospective validation is required prior to application in oncologic practice.

2:30-2:55 Nanopore-Facilitated Single Molecule Detection of Circulating microRNAs in Lung Cancer Patients
Li-Qun Andrew Gu, Ph.D., Associate Professor, Biological Engineering and Dalton Cardiovascular Research Center, University of Missouri

Developing new methods for lung cancer screening and early diagnosis is a critical issue for saving lung cancer patients’ lives. microRNAs (miRNAs) are small regulating RNA molecules that have been recognized as cancer biomarkers. We developed a nanopore sensor that is combined with a programmable oligonucleotide probe for selective and sensitive single molecule detection of miRNAs in lung cancer patient plasma samples. The sensor also demonstrated ability to discriminate miRNAs containing single nucleotide differences. This simple, sensitive, label-free technique requiring no amplification for miRNA detection has the potential for noninvasive and cost-effective early diagnosis of lung cancer.

3:00-3:20 microRNA Signatures and Roles in Prostate Cancer Progression
Cassandra Belair, Ph.D., Researcher, Urology, University of California, San Francisco

To test the global role of miRNAs in prostate cancer progression, we have crossed a prostate specific cre to PTEN- and DGC8R8-conditioned knockout mice. The loss of DGC8R alone had no phenotype, while the loss of PTEN resulted in progression through hyperplasia, dysplasia, and microinvasion, which was blocked with their simultaneous loss. Expansion of basal-like cells was also blocked in the PTEN-DGC8R double knockouts. An analysis of miRNAs in the plasma of human prostate cancer patients identified miRNA signatures associated with risk for progression. We are evaluating the functional roles of these miRNAs in progression using our in vivo model.

3:20-3:50 Networking Refreshment Break in the Exhibit Hall with Poster Viewing

4:00-4:25 Extracellular microRNA: A New Source of Biomarkers
Kai Wang, Ph.D., DABT, Senior Research Scientist, Institute for Systems Biology

MicroRNAs are small, non-coding RNAs that play an important role in regulating various biological processes in cells. Recently, some miRNAs have also been found in extracellular space. We examined the presence of miRNAs in a variety of normal human body fluids, from tears to breast milk, with the goal of assessing the distribution of miRNAs and examining the potential use of miRNAs as biomarkers. Our results indicate that miRNAs are present in all fluids tested and show distinct compositions in different fluid types. As an example, with a limited number of urine samples from individuals with several physiopathological conditions, we demonstrated the potential for using changes in the urine miRNA spectrum as biomarkers. This finding suggests the possibility of using the levels of specific miRNAs in body fluids to detect various pathological conditions.

4:25-4:50 microRNA and Thyroid Cancer
Honey Reddi, Ph.D., Assistant Professor, Medicine, Mayo Clinic

Thyroid cancer accounts for 96% of endocrine malignancies affecting about 3 million individuals in the U.S. Pre-operative assessment of thyroid nodules by currently used cytological methods poses a significant clinical challenge due to a 20% non-diagnostic rate. This results in unnecessary surgical intervention, wherein only 8-17% of cases are malignant, thereby prompting the need for additional methods of detection. We have identified a panel of miRs that is currently being tested for its potential to be used as a robust clinical assay for pre-operative diagnosis of malignancy and non-invasive follow-up of thyroid cancer patients. Also, some of the mechanisms by which these individual miRs are differently regulated will be discussed.

5:30-5:55 Towards Enhancing the Shelf-Life of ex vivo Stored Therapeutic Blood Cells: Study of microRNA-Target miRNA Interactions in Platelets
C.D. Atreya, Ph.D., Associate Director for Research, Office of Blood Research and Review, CBER, FDA

Recently we have shown that miRNAs do exist, and their profiles change in platelets during ex-vivo storage. Here we demonstrate selected miRNA-miRNA interactions that have relevance to the apoptotic pathway. Understanding these interactions would help facilitate pathways for developing strategies to enhance the shelf-life of the platelets, the most sought-after therapeutics in transfusion medicine.

5:55-6:30 Networking Reception in the Exhibit Hall with Poster Viewing
Implications of miRNAs in Colorectal Cancer and Cancer Stem Cells
Jingfang Ju, Ph.D., Co-Director of Translational Research, Pathology, Stony Brook University
We have recently developed a novel approach to discover both degraded and non-degraded miRNA mediated targets. We have discovered that several miRNAs (miR-140, miR-192, miR-215) play key roles in colorectal cancer by impacting cell proliferation, cell cycle control and chemoresistance. Some of the key targets (TS, DHFR, DTL, HDAC4) and pathways have been identified. The therapeutic potential and prognosis significance of these miRNAs were also investigated both in vitro and in vivo.

microRNAs: Native Regulators and Potential Therapeutics for Cancer Stem Cells
Liang Xu, M.D., Ph.D., Associate Professor, Department of Molecular Biosciences, University of Kansas
microRNAs have been implicated in cancer initiation and progression and are involved in cancer stem cell dysregulation. We show that CD44+/CD133+ cancer cells are enriched with cancer stem cells that have high levels of Notch/Bcl-2 and loss of miR-34. miR-34 restoration reduces the cancer stem cell population, inhibits tumorsphere growth and tumor-initiation in vivo via modulating Bcl-2 and Notch, supporting that miR-34 is involved in cancer stem cell self-renewal. Currently, we are exploring nanoparticle-targeted delivery of miR-34 to cancer stem cells as a novel molecular cancer therapy. Tumor targeted miRNA therapy holds promise as a novel molecular therapy targeting cancer stem cells.

Networking Coffee Break in the Exhibit Hall with Poster Viewing

TECHNOLOGY SHOWCASE
10:45-11:15 am Discovery of a miRNA-Based RT-qPCR Signature Able to Detect Early Stage Colorectal Cancer in Blood Plasma
Adam Baker, Ph.D, Director, Diagnostics & Pharma Partnering, Exiqon A/S
Colorectal cancer (CRC) ranks 2nd in number of deaths among cancers of the western world. There is therefore a clear unmet need for a sensitive screening assay to select at-risk individuals for definitive diagnosis by colonoscopy.
To screen for miRNAs in blood plasma, we developed an LNA-enhanced miRNA RT-qPCR platform. Using this platform, we performed a two-phased discovery program in plasma samples from stage II/III CRC patients and age- and gender-matched colonoscopy-verified healthy controls.
11:15 am-12:00 pm Sponsored Presentations (Opportunities Available)
Contact Jon Stroup at jstroup@healthtech.com or 781-972-5483

12:00-1:30 Lunch on Your Own
microRNA AND CANCER MECHANISM
1:30-1:35 Chairperson’s Opening Remarks
1:35-2:00 Causes and Consequences of microRNA Dysregulation in Cancer
Carlo M. Croce, M.D., Professor and Chair, Department of Molecular Virology, Immunology and Medical Genetics; Director, Institute of Genetics, Ohio State University
During the past several years it has become clear that alterations in the expression of microRNA genes contribute to the pathogenesis of most, perhaps all, human malignancies. These alterations can be caused by a variety of mechanisms, including deletions, amplifications or mutations involving microRNA loci, by epigenetic silencing or by dysregulation of transcription factors targeting specific microRNAs. Since malignant cells show dependence on the dysregulated expression of microRNA genes, which in turn control or are controlled by dysregulation of multiple protein coding oncogenes or tumor suppressor genes, these small RNAs provide important opportunities for development of future microRNA based therapies.

2:00-2:25 microRNA Regulation by Different Akt Isoforms
Philip Tischlis, M.D., Jane F. Desforges Professor of Hematology and Oncology, Tufts Medical Center

2:25-2:50 microRNA Alterations that Contribute to Melanoma Metastasis
Eva Hernando, Ph.D., Assistant Professor, Department of Pathology, NYU School of Medicine
Recent evidence supports the contribution of altered microRNAs to melanoma progression. Using miRNA expression arrays, we identified miRNAs 30b and 30d (chr 8q24) as overexpressed in metastatic melanoma, with higher levels associated with increased stage. Ectopic expression of miR-30d enhanced melanoma cell invasion in vitro and metastasis in vivo.

Collectively, our results expand our understanding of the mechanisms that control melanoma metastasis, potentially revealing novel therapeutic avenues for patients for whom no viable approaches are currently available.

microRNAs and Prostate Cancer
Aurora Esquila-Kerscher, Ph.D., Assistant Professor, Department of Microbiology and Molecular Cell Biology, Leroy T. Canoles Jr. Cancer Research Center, Eastern Virginia Medical School

2:50-3:15 Networking Refreshment Break in the Exhibit Hall with Poster Viewing

3:15-4:15 Networking Refreshment Break in the Exhibit Hall with Poster Viewing

4:15-4:40 microRNA Binding Site SNPs and Cancer Risk, Response and Outcome
Joanne Weidhaas, M.D., Ph.D., Assistant Professor, Department of Therapeutic Radiology, Yale University School of Medicine

4:40-5:05 Functional Investigation of microRNAs Using Genomics and Proteomics
Jun S. Wei, Ph.D., Staff Scientist, Pediatric Oncology Branch, Oncogenomics Section, National Cancer Institute
microRNAs are an important class of gene expression regulators. This talk will give an overview of the important role of microRNA in human cancers. Studies of microRNA expression in pediatric malignancies using functional, genomic and proteomic approaches for clinical use such as diagnostic, prognostic biomarkers and as potential therapeutic targets will be discussed. New technologies such as next-generation sequencing to identify novel microRNAs in human cancers will also be introduced.

5:05-5:30 Discovery and Function of Tumor Virus microRNAs
Christopher S. Sullivan, Ph.D., Assistant Professor, Department of Molecular Genetics and Microbiology, The University of Texas at Austin
We, and others, have shown that human tumor viruses from the Polyoma and Herpes families encode microRNAs. While an understanding of the functions for most viral microRNAs remains incomplete, multiple studies have indicated that viral microRNAs will play an important role in down-regulating the expression of both host and viral-encoded genes. Here we present our efforts to identify new tumor virus-encoded miRNAs. We show that some of these newly-identified RNAs play a role in autoregulation of viral gene expression and may act via a mechanism that has so far escaped detection for virus-encoded transcripts.

5:30-5:55 Kaposi’s Sarcoma-Associated Herpesvirus miRNAs and their Roles in B Cell Development and Tumorgenesis
Rolf Renne, Ph.D., Professor, Department of Molecular Genetics and Microbiology, University of Florida
Kaposi’s sarcoma-associated herpesvirus (KSHV) is the causative agent for
KS and B-cell lymphomas, and expresses 17 miRNAs. To date, little is known about the roles that these miRNAs play in pathogenesis. One KSHV miRNA, miR-K12-11, shares 100% seed sequence homology with hsa-miR-155, an oncogenic miRNA that plays important roles in B-cell differentiation. Ectopic miRNA expression in human cord blood progenitors during hematopoiesis in NOD/1LtSz-scid IL2R-γc- mice revealed that miR-K12-11 induces splenic B cell expansion and mimics miR-155. In addition, results from Ago-2/CLIP-Seq experiments performed in latently infected lymphoma cells will be discussed.

**WEDNESDAY, MARCH 30**

7:30-8:15 am  **Breakfast Presentation (Sponsorship Opportunity Available) or Morning Coffee**
Contact Jon Stroup at jstroup@healthtech.com or 781-972-5483

**microRNA IN HUMAN DEVELOPMENT AND DISEASE**

8:30-8:35  **Chairperson’s Opening Remarks**

8:35-9:00  **Inverse Modifications in microRNAs -132 and -211 Contribute to Synapse and Cholinergic Malfunctioning in Alzheimer’s Disease**

Hermona Soreq, Ph.D., Professor, Molecular Neuroscience, Biological Chemistry, The Hebrew University of Jerusalem

Alzheimer’s disease (AD) notably involves failed synaptic functioning and premature death of cholinergic neurons, but the underlying mechanism(s) and possible interrelationships between these two phenomena are yet incompletely understood. Using a global high-throughput screening, we discovered increased exon inclusion events and corresponding decreases in the exon exclusion regulators, heteronuclear ribonucleoprotein particles (hnRNPs) in the entorhinal cortex from AD patients compared to non-demented controls. This was accompanied by increased microRNA (miR-211) which co-targets three different hnRNP miRNAs; and lenti- viral-mediated knockdown of these hnRNPs caused synapse loss in cultured neurons and learning and memory impairments in brain-injected mice. Furthermore, in vivo destruction of cholinergic neurons, but not APP or TAU mutations, reduced brain hnRNP levels, and the synaptogenesis regulating and acetylcholinesterase (AChE)-targeted miR-132 was drastically reduced in the AD entorhinal cortex, possibly attributing part of the loss of cholinergic input to this change. Together, our findings suggest that AD involves a feed-forward loop of miR-211 increases which mediate hnRNP depletion, leading to synapse loss; and parallel miR-132 decreases which cause AChE elevation, impair cholinergic signaling and enhance neuroinflammation while exacerbating hnRNPs loss.

9:00-9:25  **microRNAs that Regulate Adipocyte Differentiation and Function**

Harvey F. Lodish, Ph.D., Professor, Biology and Biological Engineering, Whitehead Institute for Biomedical Research, MIT

Brown adipocytes are specialized for heat generation and energy expenditure. To investigate the role of miRNAs in the lineage determination of brown adipocytes, we compared the global miRNA expression profiles of brown fat, white fat and skeletal muscle. We identified a brown fat-enriched miRNA cluster that was upregulated during brown fat adipogenesis and induced by ectopic expression of PRDM16 in both primary white preadipocytes and myoblasts. This miRNA cluster serves as an important downstream effector of PRDM16 and contributes to brown adipocyte lineage determination primarily by repressing white fat adipogenesis and myogenesis.

9:25-9:50  **Opposing Functions and Differential Regulation of the Bicistronic Cardiac miRNAs, miR-1 and miR-133a**

Kathryn N. Ivey, Ph.D., Staff Research Investigator, Gladstone Institute of Cardiovascular Disease, University of California, San Francisco

miR-1 and miR-133a are bicistronic muscle-specific miRNAs that are essential for heart development and function. Although they are co-transcribed, they have opposing effects on differentiation and accumulate to dramatically different levels. Therefore, we hypothesized that interaction of miR-1 or miR-133a with distinct proteins may differentially control their biogenesis or stability. Using a biochemical approach, we identified several proteins that uniquely interact with miR-1, but not miR-133a, and specifically affect the accumulation of miR-1. This represents the first description of differential regulation of bicistronic miRNA accumulation and is likely to have broader implications for miRNA control.

9:50-10:15  **Talk Title to be Announced**

Speaker to be Announced.

10:15-10:50  **Networking Coffee Break**

**microRNA IN THERAPEUTIC DEVELOPMENT**

10:50-11:15  **A microRNA Screen to Identify Modulators of Sensitivity to BCL2 Inhibitor ABT-263 (Navitoclax)**

Lloyd T Lam, Ph.D., Senior Scientist II, Global Pharmaceutical R&D, Tumor Genomics, Abbott Oncology

ABT-263 is a first-in-class BCL2 family inhibitor that restores the ability of cancer cells to undergo apoptosis. However, many cancer cells are resistant to ABT-263 due to high expression of a BCL2 family member MCL1. A functional genomics approach was used to explore the role of microRNAs in determining ABT-263 sensitivity. All the identified microRNAs restore apoptosis in the presence of ABT-263 by reducing MCL1 protein expression. This approach can facilitate the identification of microRNA modulators to other cancer agents and the design of microRNA replacement therapies.

11:15-11:40  **Novel Approach for Targeted Deregulation of miRNAs for Cancer Therapy**

Fazul H. Sarkar, Ph.D., Professor of Pathology, Barbara Ann Karmanos Cancer Institute, Wayne State University School of Medicine

Emerging evidence suggests the acquisition of epithelial-to-mesenchymal transition (EMT), a transient process that is typically found during tumor progression in vivo, which is reminiscent of cancer stem-like cell (CSC) characteristics, contributes to tumor progression and metastasis. Moreover, these cells within a tumor mass are also highly resistant to conventional therapeutics, suggesting that novel targeted strategies must be discovered so that these resistant cells could be eliminated in order to make improvement in the treatment of most solid tumors because the EMT (CSC) phenotypic cells are believed to be the root cause of tumor recurrence and metastasis. We have discovered a novel compound that is very effective in eliminating EMT (CSC) phenotypic cells both in vitro and in vivo, which was found to be due to specific deregulation of miRNAs. This subject will be discussed during the presentation.

11:40 am-12:05 pm  **miR-21 Overexpression in Breast Cancer Confers Herceptin Resistance by Downregulation of PTEN**

Dihua Yu, M.D., Ph.D., Professor and Deputy Chair, Department of Molecular and Cellular Oncology; Hubert L. and Olive Stringer Distinguished Chair in Basic Science; Director, Cancer Biology Program, The University of Texas MD Anderson Cancer Center

Ablation of expression of certain microRNAs can lead to therapeutic drug resistance. Our lab previously found that PTEN loss confers Herceptin resistance in ErbB2-overexpressing breast cancers. Other studies in hepatocellular carcinoma have shown that miR-21 targets the 3’UTR of PTEN miRNA leading to downregulation of PTEN protein levels. Based on these findings, we hypothesized that overexpression of miR-21 may confer Herceptin resistance in breast cancer patients by downregulation of PTEN. I will discuss our exciting data from cell culture, animal model, and human breast cancer patients demonstrating miR21 overexpression can be found in almost 60% of ErbB2 positive breast cancers, and miR21 overexpression in breast cancer cells confers Herceptin resistance by down-regulation of PTEN and modulation of apoptotic pathways. Furthermore, targeting miR21 can sensitize ErbB2 positive breast cancers to Herceptin treatment in vivo. These data clearly indicated a new direction and strategy of overcoming Herceptin resistance to further improve the clinical management of breast cancer patients.
microRNAs are endogenous non-coding RNAs that post-transcriptionally regulate gene expression. Each microRNA can regulate hundreds of genes that have been evolutionarily selected to regulate biological pathways. The pathways involved are central to many areas of biology, including development, cancer, metabolism, and immunity. The ability of microRNAs to modulate disease pathways by influencing multiple, functionally-linked genes makes targeting or augmenting them an exciting new approach for drug discovery. Recent genetic and pharmacologic studies have suggested opportunities for microRNA therapeutics in multiple disease areas. However, the nontraditional nature of these drug targets presents unique challenges in the process of developing targeted therapeutics. In this talk I will discuss some of these challenges as well as give examples within the areas of fibrosis and cancer where exciting new data demonstrate the potential power of microRNA-based therapeutics.

12:30 Close of Conference
Pricing and Registration Information

Program Pricing (March 28-30)

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