



RNAi & miRNA World Congress Epigenetics World Congress Genomics Automation Congress

**5-7 May 2010
Boston, MA, USA**



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RNAi & miRNA World Congress

5 May 2010

1:00 **Registration**

1:25 **Welcome and Introduction**

1:30 **Keynote Presentation:**

RNAi: New Promise in Human Therapy

Anastasia Khorova, CSO, RXi Pharmaceuticals

Since the discovery of RNAi, significant progress has been made in clarifying the basic biology of the mechanism and in successful utilization of the technology as an effective tool for functional genomics. A major hurdle still remains in the development of RNAi as a broad therapeutic class—delivery of RNAi compounds to appropriate tissues and efficient cellular uptake. Many diverse delivery approaches are being explored and several have reached the important benchmark of being tested in humans. Successful delivery will enable the targeting of novel and diverse targets as has been envisioned since the early days of RNAi research.

2:00 **In vivo Efficacy of Naked Chemically Stabilized siRNA Following Local and Systemic Administration**

Elena Feinstein, Chief Scientific Officer, Quark Pharmaceuticals

It is generally accepted that siRNA delivery is the major hurdle in development of siRNA therapeutics. Our strategy for developing siRNA therapeutics is based on selection of indications involving cells/tissues to which naked siRNA can be readily delivered.

2:30 TBC

3:00 **Coffee and Networking**

3:45 TBC

4:15 **LNA Discovery Technology and Single Stranded RNA Medicine - Executing the Pharmaceutical Potential of RNA Silencing**

Maj Hedtjaern, Groupleader, Partnered Programs Discovery, Santaris Pharma

4:45 **Combination of Oligo Configuration and Chemical Modification Provides Drug-Like Properties to RNAi Molecules**

Dmitry Samarsky, VP, Technology Development, RXi Pharmaceuticals

To become a viable drug, RNAi molecules should remain stable in biological fluids, avoid rapid clearance, penetrate the target tissue, be taken up by the target cells and, finally, activate the RISC for RNAi in the cytoplasm of these cells. The challenging task of satisfying all these requirements simultaneously can be addressed by combining particular oligo configurations and chemical modification patterns. To this end, a new class of siRNAs, 'self-delivering' RNAi compounds (sd-rxRNA™) have been developed and will be described.

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RNAi & miRNA World Congress

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- 9:00 **Keynote Presentation:
MicroRNAs in Cancer**
Frank Slack, Professor, 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. let-7 encodes a microRNA implicated in human 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.
- 9:30 **The MIRH1 Locus vs the Smad Pathway: Implications for Cancer Pathogenesis**
Andrei Thomas-Tikhonenko, Associate Professor, University of Pennsylvania
TGFbeta signaling and the Myc oncoprotein have a patently antagonistic relationship, whose exact molecular mechanism remains elusive. A new model will be presented, according to which Myc-activated MIRH1 microRNA locus targets several key components of the TGFbeta pathway, dampening cellular responses to this growth-limiting cytokine.
- 10:00 **Coffee and Networking**
- 10:45 TBA
Carlo M. Croce, Professor, Ohio State University
- 11:15 **A Tumor Suppressor microRNA in Melanoma**
Carl Novina, Assistant Professor, Dana-Farber Cancer Institute & Harvard Medical School
A microRNA expressed from the intron of a suspected melanoma tumor suppressor gene assumes the tumor suppressive function of its host gene.
- 11:45 **mir-200 Regulates Induction of Apoptosis through CD95 by Targeting FAP-1**
Marcus Peter, Professor, Ben May Department for Cancer Research, University of Chicago
miR-200 is a regulator of the epithelial-mesenchymal-transition (EMT) which resembles the process of tumor progression. We have identified the apoptosis inhibitor Fas associated phosphatase (FAP-1) as a target of miR-200 providing a mechanism of how miR-200 affects apoptosis sensitivity to the death ligand FasL during tumor progression.
- 12:15 **Technology Spotlight**
- 12:30 **Lunch**
- 1:30 **Poster Viewing Session 1**
- 2:00 TBA
Hannele Ruohola-Baker, Professor, University of Washington
- 2:30 **Deregulated mRNA/miRNA Expression Networks in Dopamine Neurons and Parkinson's Disease**
Kai-Christian Sonntag, Assistant Professor, McLean Hospital, Harvard Medical School
Dopamine (DA) neurons from Parkinson's disease (PD) patients demonstrate deregulated signaling pathways linked to PD pathogenesis. miRNAs in these neurons can be associated with the dysregulated gene expression network implying a potential role in disease mechanisms of PD.
- 3:00 **Coffee and Networking**
- 3:45 **microRNAs Changes Occur in Multiple Myeloma Cells in the Context of Bone Marrow Milieu**
Aldo Roccaro, Instructor in Medicine, Harvard Medical School, Dana-Farber Cancer Institute
It has been previously demonstrated that primary multiple myeloma (MM) cells are characterized by a specific microRNA (miRNA) signature compared to the related normal plasmacell counterpart; and that miRNAs play a crucial role in regulating MM pathogenesis. Nevertheless, miRNA changes that occur in MM cells in the context of the bone marrow microenvironment have not been previously examined. Therefore, characterization of miRNA profiling of MM cells in conjunction with bone marrow stromal cells (BMSCs) is important to better understand the underlying molecular changes that lead to initiation and progression of this disease
- 4:15 **MicroRNAs in Brain Tumor Growth and Migration**
Sean Lawler, Assistant Professor, Dept. of Neurological Surgery, The Ohio State University Comprehensive Cancer Center
MicroRNA alterations have been observed in the brain tumor glioblastoma multiforme. The presentation will discuss the function and therapeutic potential of some of these microRNAs related to growth, migration and stem cell-like properties of this tumor type.
- 4:45 **Drinks Reception**

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- 9:00 **Ago-associated Human usRNAs and Other Similar small RNAs**
Bino John, Asst Professor, University of Pittsburgh
Our study demonstrates that we must not ignore RNAs smaller than microRNAs as transient degradation products.
- 9:30 **A Tumor Targeting Delivery System for Realizing siRNA as an Anti-Cancer Nanomedicine**
Esther Chang, Professor, Georgetown University Medical Center
A tumor-targeting platform nanotechnology has been developed that can efficiently deliver therapeutic siRNA specifically to primary and metastatic cancer after systemic administration.
- 10:00 **Coffee and Networking**
- 10:45 **Therapeutic Gene Silencing in Leukocyte-implicated Diseases by Targeted and Stabilized Nanoparticles**
Dan Peer, Head, Laboratory of Nanomedicine, Tel Aviv University
Leukocytes are among the most difficult cells to transduce with RNAi. We developed a strategy that can target different subsets of leukocytes and selectively silence genes in vivo using targeted, stabilized nanoparticles (tsNPs). These carriers do not induce lymphocyte activation, interferon responses or release liver enzymes and are fully degradable. Three preclinical examples inflammatory bowel disease (IBD), blood cancer and viral infection will be discussed. We will show that tsNPs can be used for in vivo validation of new drug targets, for prevention of viral infection and for inducing therapeutic gene silencing in a preclinical setting and might be also suitable for RNAi delivery to other cell types outside the immune system.
- 11:15 **Albumin-Facilitated Extravasation of siRNA Across Vascular Endothelium Leading to Effective Gene Silencing in Cardiomyocytes**
Paul White, Senior Lecturer, Monash University
This study shows that conjugation of siRNA to albumin allows the siRNA to escape blood vessels and cause target knockdown in the mouse myocardium
- 11:45 **Inducible shRNA Expression in Mice and Rats for Gene Function Analysis**
Jost Seibler, Head of Technology Development, TaconicArtemis
We will introduce our transgenic RNAi technology, which allows inducible and reversible knock-down of single or multiple target genes in both mice and rats in a drug-like fashion in vivo.
- 12:15 **Technology Spotlight**
- 12:30 **Lunch**
- 1:30 **Poster Viewing Session 2**
- 2:00 **Sustained Over-expression of miRNA Mimics Using a Lentiviral Delivery System**
Kirk Brown, Research Scientist, Thermo Fisher
We have recently developed a new lentiviral-based miRNA expression platform. Strategies used to optimize robust miRNA expression and overall performance will be described.
- 2:30 **EXPANDING RNA Mammalian Interference Pathways**
Micheal McManus, Assistant Professor, UCSF
One of the biggest of shRNA technology is the difficulty in accessing cost-effective libraries to perform biological screens in mammalian cells. We have expanded this technology and make substantial improvements towards a next-generation RNAi library protocol.
- 3:00 **Coffee and Networking**
- 3:45 **A Genome-wide Lentiviral-RNAi Synthetic Lethality and Resistance Screen Identifies Novel Modulators of a Cancer Drug Sensitivity and Resistance in Human Breast Cancer Cells**
Attila Seyhan, Head/Principal Scientist, RNAi and compound screen, target ID and validation, Pfizer
The identification of novel mediators of cellular response to the legacy Wyeth cancer drug could lead to the development of potential combination therapies, the identification of pharmacodynamic or patient selection biomarkers, and expand our understanding of this cancer drug's mode-of-action.
- 4:15 **To Seq or not to Seq: A Comparison Study of Hypoxia Regulated microRNAs Detected by miRNA-Array and SOLiD DNA Sequencing Platforms**
Susan Mayelzadeh, Research Fellow, MD Anderson Cancer Centre
- 4:45 **Close of Conference**

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Epigenetics World Congress

6 May 2010

- 8:00 **Registration**
- 8:55 **Welcome and Introduction**
- 9:00 **Epigenomics of Differential Diagnosis - Are we there yet?**
Victor Levenson, Associate Professor, Rush University Medical Center
Analysis of methylation in cell-free circulating DNA in blood holds significant promise for non-invasive detection of various diseases. Its potential has been conclusively demonstrated for neoplastic, neurodegenerative, and other diseases, although every time healthy controls have been compared to disease. As the result, the question remains – What exactly is detected? Does every disease have its own DNA methylation signature or are signatures organ-specific, and for each organ inflammation or benign lesions produce the same signatures as cancer? So far, accumulated data argue for disease-specific DNA methylation. DNA methylation patterns are very different in plasma of patients with chronic pancreatitis or adenocarcinoma of the pancreas, suggesting that inflammatory process has its own unique pattern. Similarly, plasma patterns from patients with benign ovarian lesions are different from those from patients with ovarian cancer. If these findings are confirmed for other diseases, analysis of plasma DNA methylation may provide a universal platform for minimally invasive differential diagnosis of different diseases.
- 9:30 TBA
Enal Razvi
- 10:00 **Coffee and Networking in Exhibition Hall**
- 10:45 **Histone Methylation: The Missing Link Between Chromatin and DNA Repair**
Brenden Price, Assistant Professor, Dana-Farber Cancer Institute
DNA repair requires significant alteration in chromatin architecture. Here, we discuss a crucial role for the histone modification H3K9me3 in co-ordinating the activation of DNA damage response pathways, and explain how histone methylation controls activation of the Tip60 acetyltransferase.
- 11:15 TBA
Axel Schumacher, Project Leader, The Krembil Epigenomics Laboratory
- 11:45 **Resetting the Epigenetic Code by Histone Deacetylase Inhibition Leads to Abrogation of Autoimmune Diabetes via Differential Regulation of Cytokines**
Sundararajan Jayaraman, Associate Professor, University of Illinois
Epigenetic modifications of the genome can prevent the occurrence of autoimmune diabetes in mice. This is associated with differential modulation of cytokine gene expression.
We found that treatment with a histone deacetylase inhibitor prevented the occurrence of overt type 1 diabetes in non-obese diabetic mice. Drug treatment induced transient histone acetylation in the pancreas and spleen, abrogated inflammation of the islets, and preserved β cell function without numerically altering dendritic cells and T regulatory cells in the spleen. The expression of IFN- γ mRNA and protein levels was significantly enhanced in stimulated splenocytes derived from cured mice while TNF- α mRNA but not protein expression was reduced in non-T cells. The expression of IL-4, IL-17, IL-18 and iNOS was not modulated by drug treatment. These results suggest that epigenetic modulation of the genome can lead to protection against autoimmune diabetes via differential cytokine expression.
- 12:15 **Technology Spotlight**
- 12:30 **Lunch**
- 1:30 **Poster Viewing Session 1**
- 2:00 **Searching the PDB for Novel Readers of Methyl Marks**
Matthieu Schapira, Associate Professor, University of Toronto
The majority of known methyl-lysine readers (PHD, Chromo, MBT, Tudor, PWWP domains and Ankyrin repeats) possess an aromatic cage composed of two or more Phe, Tyr or Trp residues that may additionally include an Asp or Glu residue. A computer algorithm was specifically developed to screen all experimental structures of human proteins in the Protein Databank for solvent-accessible aromatic cages. The genes identified include known and novel putative readers of the histone code that may be involved in epigenetic signaling. Ligands co-crystallized to diverse aromatic cages indicate specific chemotypes that may be exploited by medicinal chemists to develop inhibitors against readers of methyl marks.
- 2:30 **Development of Diabetic Complications as a Result of Prior Poor Glycemic Control are Mediated by Persistent Activating Epigenetic Changes of Methyl-writing and -erasing Enzymes**
Assam El-Osta, Head of Epigenetics in Human Health and Disease, Baker IDI Heart and Diabetes Institute
Defining the molecular events that lead to a phenomenon described as “metabolic memory” which is associated with endothelial cell dysfunction will provide critical insights into the interpretation of persistent epigenetic gene-activating events.
- 3:00 **Coffee and Networking in Exhibition Hall**
- 3:45 **Dynamic DNA Methylation Programs Persistent Adverse Effects of Early-life Stress**
Chris Murgatroyd, Post-Doc, Max-Planck Institute of Psychiatry
Early-life stress (ELS) has long lasting effects on the brain. Maternal separation in mice persistently alters the offspring's hormonal responses to stress; this included elevated vasopressin (AVP) in the hypothalamus and treatment with a receptor antagonist was able to reverse the effects of early-life stress. The altered AVP expression was associated with sustained DNA hypomethylation of a region in the AVP enhancer that serves as a binding site for the methyl-CpG binding protein 2 (MeCP2). Neuronal activity was able to control the ability of MeCP2 to regulate transcription of the AVP gene and induce epigenetic marking. Thus, ELS can dynamically control DNA methylation in postmitotic neurons to generate stable changes in AVP expression that trigger neuroendocrine and behavioral alterations which are frequent features in depression.
- 4:15 **Epigenomic Diversity of Colorectal Cancer Indicated by LINE-1 Methylation in a Database of 869 Tumors**
Shuji Ogino, Associate Professor of Pathology, Harvard Medical School
Colorectal cancer is not a single disease, but a heterogeneous group of diseases at the molecular level. Our data indicate diversity of its epigenomic status, implying the necessity of considering its epigenomic heterogeneity when developing targeted therapy.
- 4:45 **Drinks Reception**

Epigenetics World Congress

7 May 2010

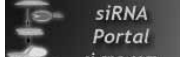
- 9:00 **Keynote Presentation:**
Rudolf Jaenisch, Professor, Whitehead Institute for Biomedical Research
- 9:30 TBA
- 10:00 **Coffee and Networking in Exhibition Hall**
- 10:45 **ATRX Partners with Cohesin and MeCP2 and Contributes to Developmental Silencing of Imprinted Genes in the Brain**
Nathalie Berube, Assistant Professor, University of Western Ontario and Children's Health Research Institute
Human developmental disorders caused by chromatin dysfunction often display overlapping clinical manifestations, such as cognitive deficits, but the underlying molecular links are poorly defined. Here we show that ATRX, MeCP2 and cohesin, chromatin regulators implicated in ATR-X, RTT and CdLS syndromes, respectively, interact in the brain and co-localize at the H19 imprinting control region (H19 ICR) with preferential binding on the maternal allele. Importantly, we show that ATRX loss results in altered enrichment of cohesin, CTCF and of histone modifications at the H19 ICR, without affecting DNA methylation on the paternal allele. ATRX was also required for normal occupancy of cohesin, CTCF and MeCP2 at a second imprinting control region within the Gtl2/Dlk1 imprinted domain. Finally, we show that loss of ATRX interferes with the postnatal silencing of the maternal H19 gene along with a larger network of imprinted genes. We propose that ATRX, cohesin and MeCP2 cooperate in the developmental silencing a subset of imprinted genes in the postnatal brain.
- 11:15 **The Effect of EBV Transformation on Genome-wide Methylation Pattern**
Karolina Aberg, Assistant Professor, Virginia Commonwealth University
The goal of this study is to evaluate the effect of the Epstein-Barr virus (EBV) transformation on methylation patterns in human lymphocytes. This will shed light on whether (whole-genome) methylation studies can be performed using samples from the increasing number of repositories that often create cell lines to be able to continuously supply DNA for multiple investigations. We used a genome-wide approach to investigating DNA extracted from whole blood, in duplicates, and EBV transformed DNA from 10 unrelated individuals. Genomic DNA was fragmented and enriched for methylated regions using MethylMiner, which captures DNA fragments that contain methylated CpG sites. The methylation-enriched fraction from each sample (N=30) was hybridized to 45 million probes on the Affymetrix GeneChip® Human Tiling 2.0R Array Set. Preliminary results indicate that the correlation between the two samples extracted from whole blood and the EBV transformed DNA is high (0.88 – 0.98) meaning that the transformation largely preserve the rank of the probe intensities. However, if we use a mixture modeling approach that enables us to study only the methylated regions, there are clear indications that the EBV transformation disturbs the methylation patterns of individual subjects.
- 11:45 **Imprinted Genes Show Upregulation Similar to Dosage Compensation of X-linked Genes**
Ismail Zaitoun, Post-Doc, University of Wisconsin-Madison
Genomic imprinting is a parent-of-origin-specific monoallelic expression of a subset of genes in placental mammals. Imprinted genes, which belong to families with different biological functions, control growth and development in a dose-dependent manner and their aberrant expression is associated with defects in development, growth, and behavior. Imprinted genes show allele-specific histone modification patterns; silenced alleles are bound by suppressive modifications, while expressed alleles are associated with permissive histone modifications, suggesting that imprinted genes would show upregulation in gene expression. To investigate this in mice, we calculated the intensity of expression of 59 imprinted genes relative to the rest of the genome by analyzing microarray data. Quantitative real-time PCR (qPCR) was performed to confirm microarray results. Expression of imprinted genes was found to be upregulated in a wide spectrum of adult and embryonic mouse tissue types. Consistent with their functions in growth and development, imprinted genes were found to be highly expressed in extraembryonic tissues and progressively upregulated during early embryonic development. In conclusion, upregulation of imprinted genes found in this study is similar to the dosage compensation (twofold upregulation) recently reported for X-linked genes.
- 12:15 **Technology Spotlight**
- 12:30 **Lunch**
- 1:30 **Poster Viewing Session 1**
- 2:00 TBA
Claes Wahlestedt, Professor, Director of Neuroscience Discovery, The Scripps Research Institute
- 2:30 **Decreased Histone-acetylation Status and Increased HDAC Activity Characterize Waldenstrom's Macroglobulinemia Tumor Cells**
Aldo Roccaro, Instructor in Medicine, Harvard Medical School, Dana-Farber Cancer Institute
Epigenetic regulation of gene expression, including histone acetylation, is commonly deregulated in many malignancies leading to aberrant transcription, but histone acetylation status in low grade lymphoplasmacytic lymphoma, such as Waldenstrom's Macroglobulinemia (WM) has not been evaluated yet.
- 3:00 **Coffee and Networking in Exhibition Hall**
- 3:45 **Epigenome Sequencing comes of age in Development, Differentiation and Diseases Mechanism Researches**
Li Ling, Project Leader, BGI
We have generated the first human diploid methylome profile in a blood monocyte cell. In our study, the first high resolution human DNA methylation profile map was generated, which was determined by 20X coverage genome-wide resequencing after bisulfate treatment. The study performed herein not only provides a comprehensive methods for detailed understanding of epigenetic regulation mechanisms, but also serves as a catalyst for future studies of the epigenetic mechanisms that regulates development, differentiation and immune regulation mechanisms.
- 4:15 **Ewing's Sarcoma Family of Tumors (ESFT): A Case of Oncogene-mediated Epigenetic-addiction?**
Idriss Bennani-Baiti, Children's Cancer Research Institute Associate Investigator
Ewing's sarcoma offers a good model to investigating the epigenetic bases of cancer. We present our findings on several histone-modifying enzymes such as Aurora-B, BRCA1, EZH2, MLLT3, p300, and jumonji proteins that contribute to the oncogenic phenotype of many cancers.
- 4:45 Close of Conference

Genomics Automation Congress

6 May 2010

- 8:00 **Registration**
- 8:55 **Welcome and Introduction**
- 9:00 TBA
Greg Caporaso, Colorado University
- 9:30 TBA
Steven Jones, Genome Sciences Centre, British Columbia Cancer Research Centre
- 10:00 **Coffee and Networking in the Exhibition Hall**
- 10:45 **Automated Multiplex High-Throughput Next-Generation Sequencing Of microRNA**
Francois Vigneault, Ragon Institute Fellow, Harvard Medical School, Ragon Institute Fellow
We demonstrate the use of bar-coding for next-generation sequencing of microRNA for the Illumina sequencing platform, including tips and trick from the bench for efficient library preparation and the use of automated liquid handling robots.
- 11:15 **Next Generation Microarray and Sequencing Technologies for Genome-wide Dosage Assays in Yeast and Man**
Corey Nislow, Assistant Professor, Department of Molecular Genetics, University of Toronto
We have been developing novel high-density microarray platforms and multiplex NGS technologies for highly parallel analysis of gene-dose assays to understand gene functions using small molecule probes.
- 11:45 **Expression Profiling of microRNAs in Ovarian Cancer using Deep Sequencing**
Chad Creighton, Assistant Professor, Division of Biostatistics, Dan L. Duncan Cancer Center
By comprehensively profiling expression of microRNAs and genes in ovarian cancer, we have identified strong candidate microRNAs and their target genes that may contribute to the pathogenesis of this disease.
- 12:15 **Technology Spotlight**
- 12:30 **Lunch**
- 1:30 **Poster Viewing Session 1**
- 2:00 TBA
Micheal Metzker, Associate Professor, Human Genome Sequencing Center, Baylor College of Medicine
- 2:30 **Electron Tunneling for Label-free Sequencing?**
Stephen Lindsay, Edward and Nadine Carson Professor of Physics and Chemistry, Arizona State University
Electron tunneling through DNA bases exiting a nanopore has been proposed as a route to rapid, label-free, single molecule sequencing. In unpublished work, we have shown that all four bases can be identified via the distinctive signal generated as they transit a fixed electrode gap functionalized with a recognition molecule that traps each base transiently with a unique pattern of hydrogen bonds. Even at a read time of 1 ms/base, parallel readouts are needed to increase accuracy and speed. We have shown that metallic carbon nanotubes can be used as nanopores, integrating nanopore and electrode in a device that might be mass-produced.
- 3:00 **Coffee and Networking in the Exhibition Hall**
- 3:45 TBA
Ewen Kirkness, Investigator in Genomic Medicine, J. Craig Venter Institute
- 4:15 TBA
Masako Suzuki, Dept. Of Genetics, Albert Einstein Colledge of Medicine
- 4:45 **Drinks Reception**

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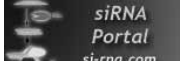
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- 9:00 **Diagnostic Sequencing using Second Generation Technologies** Birgit Funke, Associate Director at the Laboratory for Molecular Medicine, Harvard Medical School
Medical sequencing for diseases with locus and allelic heterogeneity has been limited by the high cost and low throughput of traditional sequencing technologies. "Second generation" sequencing (SGS) technologies allow parallel processing of a large number of genes and therefore offer great promise for medical sequencing; however, their use in clinical laboratories is still in its infancy. Our laboratory offers clinical resequencing for hypertrophic and dilated cardiomyopathy (HCM, DCM) using an array-based platform that interrogates 11 and 19 genes, respectively.
- 9:30 TBA
Stuart Brown, Associate Professor, New York University Langone Medical Center
- 10:00 **Coffee and Networking in Exhibition Hall**
- 10:45 **Preparing your Template, One Size Doesn't Fit All**
Masoud Toloue, Senior Scientist, Bioo Scientific
The demand for preparative steps that deliver fast and unbiased small nucleic acid sequence for investigation cannot be overemphasized. Current isolation, fragmentation, ligation and purification methods lead to bias, require significant preparatory time and are highly inefficient. To circumvent these obstacles we have developed several strategies that not only eliminate small RNA circularization and linker dimers, but allow for cross genome analysis using multiplexed barcodes, improving next gen. sequencing and experimental design. It has become obvious that the use of the same preparatory methods to identify and quantify rare transcripts and perform large scale comparative evolutionary studies is not ideal and that there are clear advantages for particular applications over others. Recent efforts directed towards the development of new template preparations illustrate this.
- 11:15 TBA
Nick Bergman, Head of Genomics Group, NBACC
- 11:45 **Medical Practices, Regulation, and Patents: How They Might Affect the Transition to Full-genome Sequence Analysis**
Robert Cook-Deegan, Director of the IGSP's Center for Genome Ethics, Law & Policy, Duke University
- 12:15 **Technology Spotlight**
- 12:30 **Lunch**
- 1:30 **Poster Viewing Session 2**
- 2:00 TBC
- 2:30 TBC
- 3:00 **Coffee and Networking in Exhibition Hall**
- 3:45 TBC
- 4:15 TBC
- 4:45 Close of conference

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