6th Annual Postdoctoral Science Symposium

September 8, 2016

Organized by the MD Anderson Postdoctoral Association
Sponsored by The University of Texas MD Anderson Cancer Center Alumni & Faculty Association and the Office of Postdoctoral Affairs, a unit of Faculty and Academic Development

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

Symposium Agenda

September 8, 2016

Morning Sessions

9:00 am

Welcome Address
APSS Organizing Committee

9:10 – 10:00 am

Huda Y. Zoghbi, M.D.
Department of Molecular and Human Genetics,
Baylor College of Medicine
Title: **Rett Syndrome and MeCP2 disorders and the insight they provide into neuropsychiatric disorders**

10:00 – 10:50 am

Postdoctoral Fellows

Melinda Engevik, Ph.D.
Department of Pathology & Immunology,
Baylor College of Medicine
"Clostridium difficile chemotaxes *and forms biofilms with other gut microbes in intestinal mucus "*

Chunxu Gao, Ph.D.
Departments of Pathology and Immunology,
Baylor College of Medicine
"Microbiome-mediated suppression of inflammation-associated colon carcinogenesis by histamine production in Lactobacillus reuteri"

Amrita Mandal, Ph.D.
Department of Genetics,
The University of Texas MD Anderson Cancer Center
"Molecular pathways regulating stem cell behavior in epithelial bilayers during tissue homeostasis and regeneration"

Roger Lopez Bellido, Ph.D.
Department of Genetics,
The University of Texas MD Anderson Cancer Center
"A novel chemical nociception behavioral assay in Drosophila larvae"

Symposium Agenda

10:50 - 11:30 am

Poster Session 1 / Coffee Break

11:30 – 12:20 pm

Postdoctoral Fellows

Xiaofeng Zheng, Ph.D.
Department of Cancer Biology,
The University of Texas MD Anderson Cancer Center
"Epithelial-to-mesenchymal transition is dispensable for metastasis but induces chemoresistance in pancreatic cancer"

Umesh Karandikar, Ph.D.
Department of Molecular Virology and Microbiology,
Baylor College of Medicine
"Identifying intestinal cell types that can support replication of human norovirus in intestinal biopsies from immunocompromised patients and intestinal stem cell derived cultures"

Lalit Sehgal, Ph.D.
Department of Lymphoma/Myeloma,
The University of Texas MD Anderson Cancer Center
"Tumor microenvironment influences survival of mantle cell lymphoma through FGF/FGFR1 signaling"

Rasoul Pourebrahimabadi, Ph.D.
Department of Genetics,
The University of Texas MD Anderson Cancer Center
"Mutant p53 Gain-of-Function driven metastasis explored utilizing a traceable conditional osteosarcoma model"

12:20 – 1:25 pm

Breakout Sessions / Lunch

Keep it Simple: Lessons learned from teaching science to kids
with **Dr. *Ennio Tasciotti*** **S3.8003ab**
Institute for Academic Medicine, Houston Methodist

Show off what you're really working with through a winning cover letter and CV
with ***Dr. Toyin Babarinde*** **Rm. S3.8014**
Scientific Project Director, Neurosurgery,
The University of Texas MD Anderson Cancer Center

Symposium Agenda

How to promote yourself – for women postdocs
with ***TMC Women's Leadership Council*** **Onstead Auditorium**
Dr. Carmel B. Dyer – Associate Dean, Harris County Programs
John P. and Kathrine G. McGovern Medical School/UT Health
Dr. Toi Blakley Harris – Associate Provost of Institutional Diversity
Baylor College of Medicine
Dr. Laura A. Petersen – Chief, Section of Health Services Research
Baylor College of Medicine
Carolyn Smith – Senior Associate Dean
GSBS/Baylor College of Medicine
Dr. Julia D. Andrieni – President & CEO, Physicians Alliance for Quality,
Houston Methodist

How to Give a Great Elevator Speech
with **Dr. *Carrie Cameron*** **GSBS Large Classroom**
Department of Epidemiology,
The University of Texas MD Anderson Cancer Center

Afternoon Sessions

1:30 – 2:30 pm

Cullen Taniguchi, M.D., Ph.D.
Department of Radiation Oncology,
The University of Texas MD Anderson Cancer Center
Title: **Protecting the gut to improve outcomes in pancreatic cancer**

2:30 – 3:20 pm

Postdoctoral Fellows

Emanuela Gentile, Ph.D.
Department of Thoracic and Cardiovascular Surgery,
The University of Texas MD Anderson Cancer Center
*"*A novel cationic liquid crystalline nanoparticle for the delivery of synthetic RNAi-based therapeutics*"*

Maria Neus Bota Rabassedas, Ph.D.
Departments of Thoracic/Head & Neck Medical Oncology,
The University of Texas MD Anderson Cancer Center
"A 3D co-culture model to elucidate the molecular basis for interactions between cancer-associated fibroblasts and lung adenocarcinoma cells that drive early metastasis"

Symposium Agenda

Junji Xing, Ph.D.
Immunobiology and Transplantation Research,
Houston Methodist Research Institute
"Identification of TRIM29 in the control of innate immunity in the respiratory tract"

Jason Carey, Ph.D.
Department of Experimental Radiation Oncology,
The University of Texas MD Anderson Cancer Center
"Downregulation of c-myc sensitizes breast cancer cells to PARP inhibition independent of BRCA status"

3:20 – 4:00 pm

Poster Session 2 / Coffee Break

4:00 – 5:00 pm

Keynote

Mina Bissell, Ph.D.
Distinguished Scientist
Biological Systems and Engineering Division,
Lawrence Berkeley National Laboratory
"Why don't we get more cancer: The critical role of Extracellular Matrix and Microenvironment in metastasis and dormancy"

5:00 – 5:15

Award Ceremony and Closing Remarks
*with **Dr. Ethan Dmitrovski***

5:15 – 6:15 pm

Reception

Distinguished Speakers

Mina J. Bissell, Ph.D.

Keynote Speaker

Distinguished Scientist, Life Sciences Division
Lawrence Berkeley National Laboratory

Education:

Degree-Granting Education

1963	BA Chemistry, Harvard College, Cambridge, MA
1965	M.A Bacteriology & Biochemistry Harvard University, Cambridge, MA.
1969	Ph.D. Microbiology & Molecular Genetics Harvard University, Cambridge, MA

Postgraduate Training

1969-1970	Milton Fellow, Harvard University
1970-1972	ACS fellow, UCB
1979-Present	Faculty, Graduate group in Comparative Biochemistry, UCB
1992-2002	Director, Life Sciences Divison, LBNL
2002-Present	Distinguished Scientist, LBNL

Notable Honors/Awards

2015	Honorary Medal (STS/CCSS)
2015	Ernest W Bertner award, MDACC
2012	Lifetime Achievement Prize, LBNL

Research Interest:

Mina Bissell has been recognized for her lifetime contributions to the fields of breast cancer research, the enhanced role of extracellular matrix (ECM) and the nucleus environment to gene expression in normal and malignant tissues. These works have ushered and have changed some central paradigms that have strengthened the importance of context in the development of cancer.

Huda Y. Zoghbi, M.D.

Guest Speaker

Professor, Molecular and Human Genetics & HHMI Investigator
Baylor College of Medicine

Education:

Degree-Granting Education

1976 BS American university of Beirut, Beirut, Lebanon
1979 MD Meharry Medical College, Nashville, Tennessee

Postgraduate Training

1979-85 Post-Doctoral fellowship at BCM

Notable Honors/Awards

2014 Honorary Doctorate of Science, Yale University
2014 March of Dimes Prize in Developmental Biology
2014 Sckolnick Prize, MIT
2013 The Pearl Meister Greengard Prize, The Rockefeller University
2013 Dickinson Prize in Medicine
2011 Gruber Prize in Neuroscience
2004 Elected Fellow, National Academy of Sciences
2008 Texas Women's Hall of Fame award
2001 Bernard Sachs Award

Research Interest:

Huda Zoghbi and her collaborators have unraveled the genetic underpinnings of a number of devastating neurological disorders, including Rett syndrome and spinocerebellar ataxia type 1 (SCA1). Their discoveries have provided new ways of thinking about more common neurological disorders, including autism, intellectual disability, and Parkinson's disease, and could lead to better treatments.

Cullen M. Taniguchi, M.D., Ph.D.

Guest Speaker

Assistant Professor, Department of Radiation Oncology
MD Anderson Cancer Center, Houston, TX

Education:

Degree-Granting Education

2008 MD, Medicine, HMS, Boston, MA,
2005 PHD, Cell & Developmental Biology HMS, Boston, MA,
2000 M Phil, Economic and Social History, University of Oxford

Postgraduate Training

2008-2009 Transitional Internship, Santa Clara Valley Medical Center, San Jose, CA
2009-2013 Clinical Residency, Radiation Oncology, Stanford University, Stanford, CA
2013-2014 Research Fellowship, Radiation Oncology, Stanford University, Stanford, CA

Notable Honors/Awards

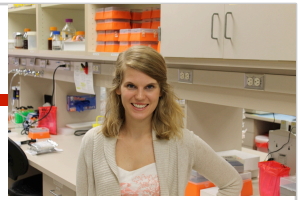
2012 MERIT Award, American Society of Clinical Oncology
2011 Resident Clinical Poster Award, 2nd Place, ASRO
2005 Basic Science Student Award, Endocrine Society
2004 Medical Scholar, American Diabetes Association
1998 Rhodes Scholarship
1998 Scholarship, Glaxo Wellcome
1997 Scholar, Howard Hughes Medical Institute
1996 Barry M Goldwater Scholarship

Research Interest:

Cullen Taniguchi is devoted to understanding the role of hypoxic and metabolic signaling in the initiation and progression of cancer, diabetes and other human disorders. Their group is particularly interested in dissecting the contribution of hypoxic and metabolic signaling in the tumor microenvironment.

Oral Speakers

Melinda Engevik, Ph.D.



Department of Pathology & Immunology, Baylor College of Medicine

Research Interest:

The long-term goal of my research is to elucidate the role of intestinal mucus in the setting of health and disease. As a post-doctoral fellow at Baylor College of Medicine, I have begun to examine the role of intestinal mucus in the setting of *Clostridium difficile* infection (CDI). CDI is the most common cause of bacterial-induced diarrhea and severe colitis in the United States, with an estimated 83,000 first disease recurrences and 29,000 deaths reported annually. The major risk factor for developing CDI is antibiotic-disruption of the gut microbiota, which promotes *C. difficile* spore germination and eventually toxin production. Currently little information exists on where *C. difficile* colonizes in the intestine and which resident microbes contribute to CDI. Our preliminary data indicates that *C. difficile* chemotaxes and adheres to mucus *in vitro* and *in vivo.* To define the role of intestinal mucus in CDI we are using several clinically relevant models: (1) mouse models, (2) biofilms, (3) bioreactors and (4) intestinal enteroids. These various models allow us to examine community dynamics and better understand the factors that contribute to *C. difficile* pathogenesis. This knowledge may provide evidence for exploring novel therapeutic approaches that modulate and bolster mucus production to limit CDI.

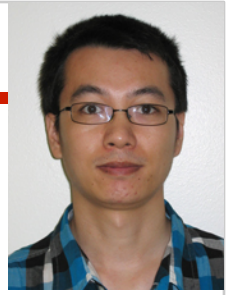
Abstract:

Background: *Clostridium difficile* infection (CDI) is one of the most costly nosocomial infections. It is estimated that CDI is responsible for 3 billion dollars of health care expenditures each year. Studies have shown that antibiotic disruption of the gut microbiota creates a favorable niche for *C. difficile* spore germination and proliferation. In order to reach the epithelium, gastrointestinal (GI) pathogens such as *C. difficile* must likely associate with the intestinal mucins. Several pathogens have been demonstrated to adhere and manipulate mucus; however few studies have addressed this interplay with *C. difficile*. As mucus is among the first lines of epithelial defense, this information may hold the key for *C. difficile* host colonization and be a potential preventative treatment target. **Methods & Results**: Our data demonstrate that *C. difficile* R20291 and three clinical isolates are capable of chemotaxis towards intestinal mucus derived from both stool and the mucus-secreting cell lines LS174T and HT29-MTX cells as determined by the chemotaxis capillary assay and ibid chambers. *C. difficile* chemotaxes specifically toward cleaved O-linked glycans and mucin monosaccharides, including: fucose, mannose, glucose, galactose, N-acetyl-galactocosamine (GalNAc), and N-acetyl-glucosamine (GlcNAc). *C. difficile* did not migrate towards sialic acid or the amino acid cysteine. Using CFDA-SE tagged bacteria we demonstrated that *C. difficile* is capable of adhering to stool mucus in a glycan dependent manner. Computational modeling of *C. difficile* adhesion proteins demonstrated the ability of several proteins to bind to mucin glycan structures, providing further evidence for *C. difficile* mucin adherence. Adherence of *C. difficile* to mucin glycans promoted the formation of biofilms as denoted by crystal violet, ruby red biofilm matrix stain, and calcofluor staining. Bioreactors outfitted with suspended mucin coverslips were used to examine *C. difficile* mucin chemotaxis and biofilm formation in the setting of a complex gut microbiome. C. difficile was found to form a biofilm with other gut microbes as analyzed by fluorescence in situ hybridization (FISH) and RNA sequencing. In patients with recurrent CDI and pseudomembranous colitis, *C. difficile* was found to produce a biofilm which localized to the mucus adjacent to the epithelium as determined by FISH. **Conclusions:** Collectively, these studies suggest that *C. difficile* adheres to mucin glycans and adherence promotes biofilm formation *in vitro* and *in vivo*. Adherence to mucins and formation of biofilms would allow *C. difficile* to efficiently deliver toxins in close proximity to the host, thus providing a potential mechanism for persistent colonization and infection in CDI.

Email: melinda.engevik@bcm.edu

Chunxu Gao, Ph.D.

Department of Pathology and Immunology, Baylor College of Medicine



Research Interest:

My research interest is to understand the nature of the gut microbiome and explore how microbes affect the health and disease status in the host. I seek to understand the molecular mechanisms of microbiome-mediated signaling in intestinal inflammation and carcinogenesis.

Abstract:

Colorectal cancer (CRC) is the third most common cancer and leading cause of cancer related mortality. Patients with inflammatory bowel disease have an increased lifetime risk of CRC compared to the general population. The role of the intestinal microbiome in CRC development has recently been investigated, and manipulation of the gut microbiome by probiotics was shown to suppress CRC in rodent models.

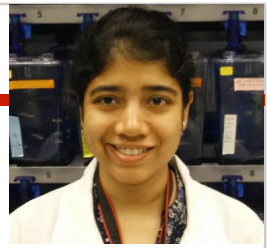
Histidine decarboxylase (HDC) deficiency has been shown to promote inflammation-associated CRC by accumulation of $CD11b^{+}Gr\text{-}1^{+}$ immature myeloid cells (IMCs). Here, we addressed the ability of histamine-producing probiotic lactobacilli to complement histamine deficiency in *Hdc*$^{-/-}$mice by inter-kingdom complementation. We demonstrate that the gut microbe *hdc*$^{+}$ *Lactobacillus reuteri* ATCC PTA 6475 converts histidine to histamine in the mouse intestine. Evidence includes significantly increased colonic *hdc* mRNA and fecal histamine quantities in *Hdc*$^{-/-}$ mice. In an azoxymethane (AOM)/dextran sodium sulfate (DSS)-induced inflammation associated CRC model, *L. reuteri* 6475 administration diminished the numbers and sizes of colon tumors and colonic uptake of [18F]fluorodeoxyglucose by positron emission tomography. Administration of *L. reuteri* 6475 suppressed KC, IL-22, IL-6, TNF, and IL-1α gene expression in colonic mucosa and reduced the amounts of pro-inflammatory, cancer-associated cytokines, KC, IL-22 and IL-6, in plasma. *L. reuteri* 6475 also decreased the relative numbers of $CD11b^{+}Gr\text{-}1^{+}$ IMCs in the spleen. Collectively, histamine generating *L. reuteri* 6475 suppresses inflammation-associated colon carcinogenesis. The anti-tumorigenic effect of *L. reuteri* 6475 was further compared with its isogenic histidine decarboxylase (*hdcA*)-deficient strain. Oral intake of *hdcA*-deficient *L. reuteri* which is unable to generate histamine did not yield increased fecal histamine quantities in *Hdc*$^{-/-}$ mice. In the AOM/DSS-induced CRC model, *hdcA*-deficient *L. reuteri* did not suppress colon carcinogenesis. These results indicate that the enzymatic machinery, histidine decarboxylase, must be present in *L. reuteri* 6475 to generate histamine as the bioactive compound *in vivo* for the suppression of colonic tumorigenesis.

Our findings link luminal conversion of dietary components by gut microbes and probiotic-mediated suppression of CRC. Microbiome-mediated suppression of carcinogenesis may open new avenues for identification of therapeutic targets and for prevention strategies in oncology, and may provide clues for discovery and development of next generation probiotics.

Email: cgao@bcm.edu

Amrita Mandal, Ph.D.

Department of Genetics,
MD Anderson Cancer Center



Research Interest:

My research interest at the Eisenhoffer lab primarily focuses on deciphering the mechanisms by which cell division and cell death is maintained for proper epithelial homeostasis. Epithelia provide the first layer of defense for any organ and abnormalities in epithelial cell death and proliferation have been implicated in several human diseases including cancer. Although our knowledge about how death and division is correlated during normal epithelia development is rather incomplete. We use zebrafish, a powerful model system to answer these questions in vivo. Studying intrinsic properties of cell behavior such as how cells communicate with each other to make the decision to proliferate or die is fascinating. The findings from this study will not only help us to understand epithelial biology in molecular detail but also have the potential to generate novel therapeutics aimed at healing cancer. As a scientist I dream about the day when disease like cancer will only be considered as regular cough & fever.

Abstract:

Epithelial tissue acts as a protective barrier for the body and organ surfaces. In order to maintain a barrier function, the process of cell renewal and turnover must involve equivalent numbers of cell division and death. Epithelial cell turnover is key for development, tissue homeostasis and regeneration, and alterations are thought to underlie numerous many human diseases, including cancer, asthma and colitis. Here we investigate the maintenance of overall cell numbers and the ability to sustain a functional barrier by perturbing a stem cell population in a living epithelial tissue. Towards that aim we have used zebrafish as a model organism to study epithelial stem cell biology *in vivo*. The developing zebrafish epidermis is a bilayered epithelium that is similar in molecular character and organization to the epithelial coating of mammalian organs, with a differentiated keratinocytes in the apical layer and stem cells residing in the basal layer. To investigate stem cells in a living epithelium, we use a GAL4 enhancer trap line that is expressed in a subset of the basal stem cell population in the developing zebrafish. When combined with UAS effector lines, the epithelial GAL4 line provides the opportunity to visualize specific stem cells for imaging, overexpress genes of interest, and target cells for ablation with spatial and temporal control. For our studies, we utilize an assay that allows induction of apoptosis specifically in a subset of the basal stem cells. Live imaging and fixed tissue analysis revealed the apoptotic cells are rapidly cleared from the tissue, and that the loss of these cells promotes proliferation of the remaining stem cells. RNA sequencing and transcriptional analysis identified molecular pathways associated with the observed stem cell death and division. Current studies are focused on validation of these new molecular targets using qRT-PCR analysis and in situ hybridization. To characterize the function of the validated candidates, we are employing Crispr-cas9 genome editing to perturb the identified genes and determine the specific role in stem cell maintenance under normal physiological conditions and after damage. Together, our novel approach allows for direct visualization and experimental manipulation of distinct subsets of stem cells within a living epidermis. The findings generated from this study will provide insight into the molecular mechanisms driving stem cell dynamics in epithelial tissues and will reveal novel therapeutic targets that can be stimulated for regeneration or are altered during pathogenesis.

Email: AMandal@mdanderson.org

Roger Lopez-Bellido, Ph.D.

Department of Genetics,
MD Anderson Cancer Center

Research Interest:
I am interested in how different noxious stimuli (thermal, mechanical, and chemical) induce distinct nociceptive responses, using *Drosophila* as a model organism.

Abstract:
Different noxious stimuli (thermal, mechanical, electrical or chemical) provoke aversive pain behaviors in humans and in animals. The molecular/genetic bases of thermal and mechanical pain responses is beginning to be understood. However, the molecular/genetic underpinning of chemically-provoked pain has not yet been developed, in part due to a lack of simple genetically tractable models to study this biology. **Objective**: Our goal is to use *Drosophila* genetics to establish a genetically tractable system to study the molecular genetic basis of chemically-induced pain. **Methods**: We developed a new model of chemical nociception (pain) in *Drosophila* by exposing larvae to solutions with increasing concentrations of hydrochloric acid (HCl). Concentrations ranging from 1.0 % to 9.0 % produced an increasingly intense behavioral reaction which was measured as an aversive rolling response. 0.5% HCl was subthreshold and provoked no rolling. Therefore, this concentration was used to study chemical allodynia (lowering of the response threshold in response to tissue injury). We also developed an ultraviolet (UV)-induced nociceptive sensitization model that induced chemical allodynia in larvae. **Results**: To determine which class(es) of sensory neuron play a role in chemical nociception process we genetically silenced the four `different classes of multidendritic peripheral sensory neurons (classes I-IV). Surprisingly, our results revealed that classes I, II and IV are required for the response to HCl, with class IV playing the main role. This response by multiple cell types has not been observed with other sensory modalities (heat, cold, mechanical). While UV-treated larvae developed slight chemical allodynia physical puncture wounding induced a stronger sensitization response, perhaps because this injury involves a breach in the cuticle barrier. Finally, our preliminary findings showed that loss of function mutants of piezo (an ion channel mediating mechanosensory transduction), painless (a transient receptor potential (TRP) ion channel), and Pvr (PDGFR/VEGFR like receptor tyrosine kinase) and its ligands regulate chemical nociception in *Drosophila*, as all of them displayed reduced sensitivity to noxious chemical stimuli.
Conclusion: We developed a novel assay to study chemically-induced pain in *Drosophila* larvae. Nociceptive behavioral responses to HCl are mediated by the I, II and IV classes of sensory neurons. Painless, Piezo, and Pvr and its ligands are key mediators of chemical nociception in *Drosophila*. The high genetic resolving power of *Drosophila* will not only improve our basic understanding of chemical pain mechanisms, but also help us to identify novel therapeutic targets for pain treatment that may ultimately prove useful in humans.

Email: RLopez5@mdanderson.org

Xiaofeng Zheng, Ph.D.

Department of Cancer Biology, MD Anderson Cancer Center

Research Interest:

Dr. Xiaofeng Zheng's research interests focus on the epithelial–mesenchymal transition (EMT) and cancer metastasis, drug resistance, immunotherapy and the function of stroma in pancreatic cancer progression using mouse models. Her recent findings published in Nature revealed that EMT is dispensable for metastasis but induces chemoresistance in pancreatic cancer, which challenged a prevailing theory in the field: the role of EMT as a crucial effector of cancer metastasis.

Abstract:

Diagnosis of pancreatic ductal adenocarcinoma (PDAC) is associated with a dismal prognosis despite current best therapies; therefore new treatment strategies are urgently required. Numerous studies have suggested that epithelial-to-mesenchymal transition (EMT) contributes to early-stage dissemination of cancer cells and is pivotal for invasion and metastasis of PDAC1–4. EMT is associated with phenotypic conversion of epithelial cells into mesenchymallike cells in cell culture conditions, although such defined mesenchymal conversion (with spindle-shaped morphology) of epithelial cells in vivo is rare, with quasi-mesenchymal phenotypes occasionally observed in the tumour (partial EMT)5,6. Most studies exploring the functional role of EMT in tumours have depended on cell-culture-induced loss-of-function and gain-of-function experiments involving EMT-inducing transcription factors such as Twist, Snail and Zeb1 (refs 2,3,7–10). Therefore, the functional contribution of EMT to invasion and metastasis remains unclear4,6, and genetically engineered mouse models to address a causal connection are lacking. Here we functionally probe the role of EMT in PDAC by generating mouse models of PDAC with deletion of Snail or Twist, two key transcription factors responsible for EMT. EMT suppression in the primary tumour does not alter the emergence of invasive PDAC, systemic dissemination or metastasis. Suppression of EMT leads to an increase in cancer cell proliferation with enhanced expression of nucleoside transporters in tumours, contributing to enhanced sensitivity to gemcitabine treatment and increased overall survival of mice. Collectively, our study suggests that Snail- or Twist-induced EMT is not rate-limiting for invasion and metastasis, but highlights the importance of combining EMT inhibition with chemotherapy for the treatment of pancreatic cancer.

Email: XZheng5@mdanderson.org

Umesh Karandikar, Ph.D.

Department of Molecular Virology and Microbiology, Baylor College of Medicine

Research Interest:

Human noroviruses have remained uncultivable under laboratory conditions. Lack of our understanding of the cell types that can support replication of these viruses in patients has impeded our efforts to grow them. I have identified intestinal cell types that can support replication of human norovirus in patient biopsies and the stem cell based model system - human intestinal enteroids (HIEs). My long-term interests include identification of the molecular players involved in the cross-talk between intestinal epithelium and immune cells during viral infections & pathogenesis and homeostasis using HIEs.

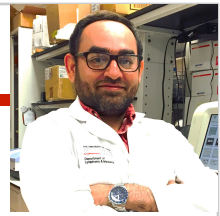
Abstract:

Human noroviruses (HuNoVs) can cause chronic infections in solid organ and hematopoietic stem cell transplant (HSCT) patients. The overall objective of this study was to characterize histopathological changes during HuNoV infection, to determine the cell types that may be permissive for HuNoV replication in the immunocompromised transplant patients and extend these results by testing if stem cell derived epithelial cell cultures can support HuNoV replication.

We analyzed biopsies from HuNoV-infected and noninfected (control) immunocompromised transplant patients to assess histopathological changes in conjunction with detection of HuNoV antigens to identify the infected cell types. HuNoV infection in immunocompromised patients was associated with histopathological changes such as disorganization and flattening of the intestinal epithelium. The HuNoV major capsid protein, VP1, was detected in all segments of the small intestine, in areas of biopsies that showed histopathological changes. Specifically, VP1 was detected in enterocytes, macrophages, T cells and dendritic cells. HuNoV replication was investigated by detecting the non-structural proteins, RdRp and VPg. We detected RdRp and VPg along with VP1 in duodenal and jejunal enterocytes. We extended these observations to tissue stem cell derived human intestinal epithelial cultures [human intestinal enteroids (HIEs)] and showed that enterocytes can support the replication of HuNoVs based on detection of HuNoV VP1 in combination with either the non-structural proteins RdRp, VPg or dsRNA. Goblet cells and enteroendocrine cells are not infected by HuNoV in small intestine-derived HIEs. These results provide insights into histological changes due to HuNoV infection in immunocompromised patients and propose human enterocytes as a physiologically relevant cell type for HuNoV replication. Successful detection of HuNoV antigens in formalin fixed biopsies can potentially be used as a diagnostic assay to detect HuNoV infection in patients where biopsies are available.

Email: karandik@bcm.edu

Lalit Sehgal, Ph.D.



Department of Lymphoma/Myeloma, MD Anderson Cancer Center

Research Interest:

Dr. Sehgal research interests focus on the communication between Mantle cell Lymphoma cells and stromal cells. His recent finding revealed that communication between the tumor and stroma can modulate the expression of key oncogene, that can be further targeted for effective therapy in MCL Relapse. The findings have forwarded the hematology field by exploring new targets for therapy.

Abstract:

Introduction

Mantle cell lymphoma (MCL) represents an aggressive, incurable form of non-Hodgkin's lymphoma (NHL). The health complications associated with advanced age of MCL patients restrict treatment with intense chemotherapy. Translocation t(11;14), responsible for overexpression of cyclin-D1, is the hallmark of MCL. More detailed insight into MCL pathogenesis has been delayed until the recent development of a tissue culture system, using human mesenchymal stromal cells (hMSC), suitable for propagating primary MCL cells. We hypothesized that tumor-initiating cells are responsible for MCL relapse and chemoresistance and thus, identification of signals responsible for survival and maintenance of MCL-initiating cells (MCL-ICs) is essential for design of curative treatment strategies.

Methods

Isolates of primary MCL cells (n = 40) were co-cultured with human mesenchymal stem cells (hMSCs) and the content of MCL-ICs was analyzed by flow-cytometry based on marker expression profile; CD34-CD3-CD45+CD19-. Cytokine array was used to identify the soluble factors enriched in the co-cultures and the expression of these factors was confirmed by RT-PCR analysis. The signaling pathways employed by the newly-identified factors were blocked in 3 MCL cell lines (JVM2, Mino, Z138) to confirm their essential role in survival of MCL cells and, more importantly, for MCL-ICs.

Results

Co-cultures of primary MCL isolates with hMSCs supported the growth of MCL cells for over 4 weeks with continued presence of MCL-ICs (CD34-CD3-CD45+CD19-) representing about 1% of MCL cells. We found that IL-6 produced by hMSCs triggered an FGF/FGFR autocrine loop in MCL-ICs. The extent of FGFR expression correlated tightly with expression of SOX11, a pathology related negative prognostic marker in MCL. MCL cell survival and growth was regulated via the FGFR/mir101/ EZH2/ NF-κB/XIAP axis. Blocking of this signaling pathway with FGFR1 inhibitors consistently induced early reduction in IAP family member and subsequently MCL cell death.

Conclusion

We established that propagation of primary MCL in co-cultures with hMSCs depends on an FGF/FGFR autocrine loop that enhances XIAP protein expression and thus, supports survival of MCL cells. We identified the factors essential for survival of MCL and MCL-ICs that present new targets for improved MCL treatment strategies.

Email: LSehgal@mdanderson.org

Rasoul Pourebrahimabadi, Ph.D.

Department of Genetics,
MD Anderson Cancer Center

Research Interest:

I am interested in the molecular mechanism of mutant TP53 gain of function activities. The focus of my research has been a traceable mouse model for osteosarcoma where the p53 is mutated specifically in the osteoblasts. I also study the interaction of tumor with the microenvironment specifically the immune response to tumors with p53 mutation.

Abstract:

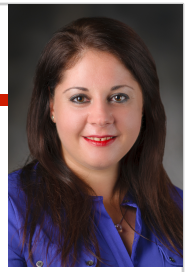
TP53 is the most frequently mutated gene in cancer. Many mutant p53 proteins exert oncogenic Gain-of-Function (GOF) properties that contribute to metastasis, but the mechanisms mediating these functions remain poorly defined *in vivo*. To elucidate how mutant p53 drives metastasis, we developed a traceable somatic osteosarcoma model that starts with either a single p53 mutation (R172H or R245W), or *p53* loss specifically in osteoblasts. To mark tumor cells, we utilized the mTmG allele which expresses membrane-targeted Tomato (mT) prior to Cre-mediated excision and membrane-targeted green fluorescent protein (mG) after excision so the tumor cells become GFP+ and the stromal cells remain RFP+. In this high penetrant, short latency mouse model, tumor micro metastasis can be detected by GFP expression in the context of a normal stroma and immune system (RFP+).

Thus far, 144 mice have developed osteosarcoma with complete penetrance and an average survival to 304± 65 day. We observed metastasis in 55% of p53 mutant osteosarcomas which was significantly higher than 26% metastasis in p53 null tumors ($p<0.01$). In mutant osteosarcomas, the metastasis rate was found significantly associated with the *p53* copy number. Spectral karyotyping (SKY) on early passage cells derived from somatic p53 mutant tumors exhibited marked aneuploidy with modal chromosome numbers near tetraploid. By analyzing tumor cells with live image microscopy and FACS, we observed that a population of tumor cells exhibited both GFP and RFP fluorescence suggesting a cell fusion event between a tumor and a stromal cell. The same population of cells with double fluorescence were detected in the blood of mice with metastasized tumors. These cells were highly positive (85%) for the monocyte lineage markers CD45 and CD11b suggesting that the tumor cells are fused with the macrophages. Fluorescent microscopy of fresh sections at the site of metastasis further showed that a part of metastatic site expresses both RFP and GFP. We are currently analyzing the role of mutant p53 in promoting metastasis via cell fusion.

These findings contribute to our understanding of the role of mutant p53 GOF in metastasis. Our long term goal is to study how tumor-stromal interactions affect tumor development and metastasis. This understanding will have broad translational significance in diagnosis and treatment of tumors with mutant p53.

Email: RPourebrahimabad@mdanderson.org

Emanuela Gentile, Ph.D.



Department of Thoracic and Cardiovascular Surgery, MD Anderson Cancer Center

Research Interest:

RNAi- based therapeutics offer a promising and powerful approach to treat multiple cancers. The key to therapeutic achievement using RNAi is to optimize the delivery using no viral vectors. My goal is to develop a novel RNAi Delivery System for the Lung Cancer therapy.

Abstract:

Small interference RNA (RNAi)-based therapeutics have been used to silence expression of targeted pathological genes. However, short half-life, poor cellular uptake, and non-specific distribution of small RNAs call for the development of novel delivery systems to facilitate the therapeutic use. We developed a novel cationic liquid crystalline nanoparticle (CLCN) for efficiently delivering synthetic RNAi including siRNAs and micro-RNA mimics. CLCNs were prepared by mixing under high speed homogenization a lipophilic phase with a hydrophilic phase containing an emulsifier/stabilizer such as poloxamer. CLCNs were assembled with synthetic small siRNA molecules in nuclease free water to create CLCN/siRNA complexes. The homogenous and stable CLCNs and CLCN-siRNA complexes displayed sizes under 100 nm and positive charges between 25-35 mV on the CLCN surface. No cytotoxicity was detected in both lung cancer and normal cells treated with various concentrations of CLCNs (from 0.01 to 100 mM) by in vitro cell proliferation assay. The CLCNs were taken up by human cells though endocytosis after binding with the cell membrane and traveling from early endosomes to the lysosome after 24 h treatment as shown by intracellular trafficking analysis with transmission electron microscopy (TEM). The presence of the fluorescent CLCN/siRNA complexes in the cytoplasm was observed as early as 2 h post treatment by confocal fluorescence imaging analysis. A significant inhibition of gene expression was detected in EGFP lung cancer H1299 cells treated with CLCNs/siEGFP complexes 24 hour after transfection compared to the untreated and non-specific CLCN-siRNA controls. Biodistribution analysis showed that the CLCNs were successfully delivered to various organs including liver and lung and into the subcutaneous human lung cancer H1299 tumor xenografts in mice 24 h after systemic administration by tail vein. CLCNs are a unique and advanced delivery system capable of protecting siRNA from degradation and efficiently delivering siRNA to the cytoplasm where effective gene silencing is achieved. (This study is partially supported by NIH/NCI grants Lung SPORE 5P50CA070907 and R01CA176568, a CPRIT Grant and a MDACC Moonshot Program Grant).

Email: EGentile@mdanderson.org

Maria Neus Bota Rabassedas, Ph.D.

Department of Thoracic/Head & Neck Medical Oncology, MD Anderson Cancer Center

Research Interest:

Understanding how cells become what they are and how they maintain their state, changing their surroundings to create the perfect environment for them, amazes me. A nice model to study these behavior is cancer: we have a group of cancer cells changing their surroundings and signaling to other cell types within it, transforming everything to their convenience. Cancer associated fibroblasts (CAFs) are found within the tumor and around the tumor periphery, signaling back and forth with tumor cells and enhancing invasion and metastasis. My current goal is to understand the interactions between lung tumor cells and CAFs and how by disrupting this communication could stop metastasis. To this end, we have developed a plethora of methods in the lab, from in *vitro* 3D matrices that mimic the tumor extracellular matrix to *in vivo* mice models.

Abstract:

Metastasis is the primary cause of death from epithelial tumors. It has become increasingly clear that metastasis is driven by complex cell-cell interactions within the tumor stroma. In human lung cancer, cancer-associated fibroblasts (CAFs) are found within the tumor parenchyma and around the tumor periphery, leading us to ask whether CAFs in these two locations play distinct biological roles in metastasis. To address this question we developed a 3D co-culture model that allows high resolution imaging and employs a microwell-based approach to create 50-cell aggregates of lung adenocarcinoma cells and primary CAFs (49:1) derived from mice that develop lung adenocarcinoma owing to expression of K-ras^{G12D}. This approach allows tight control of aggregate size and shape and spatial orientation of the CAFs, recapitulating their localization within or on the periphery of the tumor mass. In collagen gels, the aggregates develop multi-cellular invasive projections of tumor cells, and CAFs emerge from the aggregates and develop filamentous projections that make contact with invasive and non-invasive areas of the aggregates. CAFs originating from these 2 locations appear to exert distinct pro-invasive effects on tumor cells. Our goal is to elucidate how CAFs interact with tumor cells at the molecular level and whether these interactions are relevant to the pro-metastatic effects of CAFs in human lung cancer, which could be translated into therapeutic approaches to prevent metastasis in patients.

Email: MBota@mdanderson.org

Junji Xing, Ph.D.

Immunobiology and Transplantation Research, Houston Methodist Research Institute

Research Interest:

As a postdoc in Houston Methodist Research Institute affiliated with Weill Cornell Medicine of Cornell University, my research is now involved in characterizing the roles of TRIM family protein in host innate immunity regulation and revealing the unknown host defense mechanisms, autoimmune mechanisms, and ubiquitin-signaling pathways.

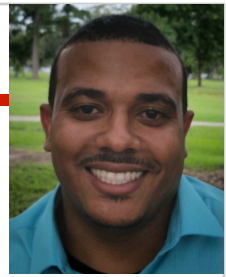
Abstract:

The respiratory tract is heavily populated with innate immune cells and represents a unique compartment in pathogen entry and protective immunity, but the mechanisms that control the local innate immunity are poorly defined. Here we report that the E3 ubiquitin ligase TRIM29 was selectively expressed by alveolar macrophages and that TRIM29 acted as a critical regulator of macrophage activation and type I interferon (IFN) as well as proinflammatory cytokine production. We found that challenge of wildtype mice with influenza A virus led to lethal pulmonary inflammation, whereas *Trim29*$^{-/-}$ mice were protected from influenza A infection duo to enhanced type I IFN production and strong anti-viral immunity. Histologically, the lung of *Trim29*$^{-/-}$ mice exhibited attenuated viral loads, reduced edema, alveolar hemorrhage, alveolar wall thickness and neutrophil infiltration. Furthermore, in response to low dose intranasal LPS challenge or *Haemophilus influenzae* infection, TRIM29 deficient mice showed markedly enhanced production of proinflammatory cytokines (up to 10 folds) and died of acute pulmonary inflammation, which contrasted to wildtype mice where most survived the LPS challenge or *Haemophilus influenzae* infection. Mechanistically, we demonstrated that TRIM29 induced Lys48 (K48)-linked ubiquitination and degradation of NEMO, a key adaptor protein in interferon regulatory factors (IRFs) signaling and NF-κB signaling, and therefore suppressing cytokine production. We also showed that TRIM29 used the OmpH-OmpH domains to bind to NEMO in the lysosome, followed by its ubiquitination at the Lys183 site and proteolytic degradation. These data demonstrate that TRIM29 is a key negative regulator of alveolar macrophage activation by degrading NEMO, and this effect is especially important in mediating local immunity and immunopathology.

Email: jxing@houstonmethodist.org

Jason Carey, Ph.D.

Department of Experimental Radiation Oncology, MD Anderson Cancer Center



Research Interest:

My research focuses on understanding disease progression and relapse through cell cycle deregulation and alterations of DNA repair mechanisms. Within this parameter I have centered a portion of my research to focus on understanding inherited mechanisms of breast cancer disease progression, including but not limited to BRCA1/2 mutations and how these inherited traits influence disease outcome. Additionally, my research is committed to understanding the correlation between cell cycle deregulation during tumorigensis and manipulation of DNA repair pathways that influences overall survival in patients to radiation/chemotherapy treatment.

Abstract:

PARP inhibitors represent a novel class of cancer therapeutics that exploit a synthetic lethal mechanism specific to cancer cells with a defect within the Homologous Recombination (HR) DNA repair pathway. Therefore patients with a BRCA1/2 germ-line mutation have the highest response rate to PARP inhibitors. However, early clinical trial data has indicated that only ~30-40% BRCA mutant patients respond to PARP inhibitors. Coincidentally, the oncogene c-myc is the most frequently amplified gene in BRCA mutant tumors. (~40-60%) Therefore we hypothesized dual combination therapy targeting c-myc and PARP in BRCA mutant tumors is an effective therapeutic strategy in BRCA mutant tumors. We screened a panel of MYC expressing BRCA wild-type and mutant TNBC cell lines for dual combination with CDK inhibitor Dinaciclib and the PARPi, Niraparib. Dinaciclib + Niraparib lead to a synergistic growth inhibition in both BRCA wild-type and mutant c-myc expressing TNBC cell lines. Dinaciclib induced sensitivity to PARP inhibition correlated with a dose dependent down regulation of c-myc in TNBC cell lines. Combination therapy also lead to an increase in DNA damage gH2AX with a decrease in HR repair (BRCA1/RAD51). C-myc siRNA induced increased sensitivity to PARP inhibitor in BRCA wild-type (MB231) & BRCA mutant (SUM149) TNBC cell lines and down regulated HR repair. TNBC cell line mouse Xenografts demonstrated down regulation of c-myc and RAD51 in Dinaciclib + Niraparib treated tumors. Dual Dinaciclib + Niraparib therapy also induced synergistic growth inhibition in PARP inhibitor acquired resistant cell lines while demonstrating significant growth inhibition across a panel of c-myc overexpressing cancer cell lines (Ovarian, Prostate, Pancreatic & Lung). In conclusion, we demonstrated that dual CDK + PARP inhibition induces synergistic growth inhibition in both BRCA wild-type and mutant TNBC cell lines and is dependent upon down regulation of c-myc. This study supports c-myc as predictor of response to PARP inhibitor therapy and may also serve as a biomarker of response to Dinaciclib + PARPi therapy in BRCA mutant patients.

Email: JPCarey@mdanderson.org

Breakout Sessions

Making the Most of Your Postdoc

Topics Include:

How to promote yourself (for women postdocs only)
with the *TMC Women's Leadership Council*
"Do you freeze when you are recognized for your accomplishments…and credit your mentor or research team in response? Or say that you were "lucky"? Are you afraid that everyone will discover that you *really* don't know as much as they think you do? Many women have these same reactions, responses and fears that have been acquired through socialization and are actually getting in the way of their success. Self-promotion is an art, for sure, but knowing how and when to speak about your accomplishments is critical to your success. In this session you will explore what gets in YOUR way of promoting yourself and develop techniques that suit your style."

Keep it simple: Lessons learned from teaching science to kids
with *Dr. Ennio Tasciotti*
"All too often scientific presentations are a complex collection of experimental results obtained with cutting edge and sometimes relatively obscure techniques. This makes scientists sound like aliens to the rest of the community with devastating results in terms of public awareness of what happens in a research lab. What's gone missing in the process is the beauty of simplicity and clarity. Every scientific endeavor can be narrated so that the research is depicted like a child's bedtime story. Learning how to master storytelling will have unexpected and incredibly beneficial outcomes in the ability to convey a scientific message to any kind of audience."

Show off what you're really working with through a winning Cover Letter and CV
with *Dr. Toyin Babarinde*
"If skills equal possibilities, and all I have is research experience, then my possibilities seem ~~LIMITED~~ ENDLESS. Do you know by virtue of *successfully* completing your PhD, you've gained a tremendous amount of transferrable skills that many companies are looking for? Your only job now is to convince potential employers that you have these transferable skills and can effectively apply them on a daily basis to a your desired position. In this session you will learn how to take inventory of ALL you have to offer and incorporate those skills into a winning CV and Cover letter."

How to Give a Great Elevator Speech
with Dr. *Carrie Cameron*
"The quote "You don't really understand something until you can explain it to someone else" has been attributed to great thinkers throughout the ages. Creating and delivering an excellent scientific Elevator Speech not only gives you presentation skill and confidence, it also develops your critical thinking capacity. This workshop, suitable for all levels, disciplines, and language backgrounds, will guide you through writing a strong 90-second speech and practicing successful rehearsal and presentation skills."

Posters

Poster Presentations

Number	Name	Title
(Morning session)		
1	Engel, Brian	Development of caspase-3 sensors for molecular imaging of cell death.
2	Chiu, Gabriel	Intranasal Administration of Mesenchymal Stem Cells Promotes Recovery from Cognitive Deficits and Neurological Dysfunction Induced by Cisplatin
3	Audia, Alessandra	CD109 regulates tumor propagation of radio-resistant glioma stem cells
4	Huang, Chenfei	NOTCH1 activation inhibits head and neck squamous cell carcinoma growth by downregulating proto-oncogenes AXL kinase and α-Catulin.
5	Pitner, Mary Kathryn	Silencing of ERK2 reverses EMT and suppresses the CSC phenotype, inhibiting lung metastasis in triple-negative breast cancer
6	Basseres, Eugenie	Understanding the Mechanism of Action of ridinilazole, a Novel Treatment for Clostridium difficile
7	Da Silva Caetano, Mauricio	Gender –Specific Role of Epithelial STAT3 in K-ras Mutant Lung Cancer
8	Du, Yi	Blocking c-Met-mediated PARP1 phosphorylation enhances anti-tumor effects of PARP inhibitors
9	Lacourt, Tamara	Differential mechanisms of cancer- versus cancer therapy-related fatigue in patients with acute myeloid leukemia
10	Duma, Denise	De novo discovery of candidate gene sets for the avian hair cell regeneration
11	Maj, Magdalena	Inhibition of mitochondrial p53 accumulation prevents cisplatin-induced neuropathy.
12	Kawakami, Masanori	Antineoplastic activity of CDK2/9 inhibitor CCT68127 occurs via induced anaphase catastrophe and inhibition of PEA15 phosphorylation in lung cancer

Poster Presentations

Number	Name	Title
13	Lakshmi Veeranki, Omkara	Role of CDK9 inhibition as a sensitizer to radiation in esophageal adenocarcinoma: in vitro and in vivo efficacy study
14	Dondossola, Eleonora	Dissecting the foreign body response to biomaterials by non-linear intravital microscopy
15	Housten, Ashley	Limitations of The Short Test Of Functional Health Literacy In Adults (S-TOFHLA) as a Health Literacy Measure
16	Deweese, Menton	Affective Modulation of the Late Positive Potential Following Repeated Exposure to Cigarette Cues in Smokers and Never-smokers
17	Lu, Li	A mouse model of neurodegeneration
18	Jin, Yan	3-D Tract-Specific Functional Analysis Of White Matter Integrity In Alzheimer's Disease
19	Zhu, Bokai	A Cell-autonomous Mammalian 12-hour Clock Coordinates Metabolic and Stress Rhythms
20	Endres, Bradley	A novel method for imaging the pharmacological effects of antibiotic treatment on *Clostridium Difficile*

Poster Presentations

Number	Name	Title
(Afternoon Session)		
21	Puerta Martinez, Franscisco	Targeting immunotherapy to metastatic cancers enhancing oncolytic viruses with immune checkpoints modulation
22	Davogustto, Giovanni	mTORcise: Replicating Exercise Induced Remodeling of the Heart by targeted deletion of Tuberin
23	Li, Hai	*In vitro* Demonstration of the Channel Activity of an Anion Channelrhodopsin
24	Kang, Hong	A Novel Schema to Enhance Data Quality of Patient Safety Event Reports
25	Ma, Jiacheng	HDAC6 Inhibition Effectively reverses Chemotherapy-Induced Peripheral Neuropathy
26	Park, Jihyun	PEA-15 (phosphoprotein enriched in astrocytes) regulates epithelial-mesenchymal transition and invasive behavior through its phosphorylation in triple negative breast cancer
27	Zhang, Jingwen	HPRM: Hierarchical Principal Regression Model of Diffusion Tensor Bundle Statistics
28	Morucho Manchon, Jose	Doxorubicin induces DNA and synaptic damage in neurons
29	Filant, Justyna	Regulation of exosome secretion in ovarian cancer
30	Maneix, Laure	Nuclear proteolysis in the regulation of metabolic genes in multiple myeloma
31	Mustachio, Lisa Maria	Loss of the ISG15 protease USP18 mislocalizes and destabilizes KRAS in lung cancer
32	Sharma, Manvi	TBD

Poster Presentations

Number	Name	Title
33	Li, Na	ZMYND8 reads the dual histone mark H3K4me1-H3K14ac to antagonize the expression of metastasis-linked genes
34	Bae, Narkhyun	Evolving Spindlin1 Small Molecule Inhibitors Using Protein Microarrays
35	Archila-Suerte, Pilar	Neuroanatomical Differences in Speech Perception Ability in Bilingual Children
36	Dorniak, Piotr	Adrenergic signaling promotes cervical tumor growth and dissemination
37	Pudakalakatti, Shivanand	Unique molecular signatures to distinguish immunotherapy responding and resistant cell lines in melanoma
38	Li, Tengfei	Predicting the clinical outcomes using imaging covariates with missing responses
38	Rivera Molina, Yisel	Modified oncolytic adenovirus, Delta-24-RGDGREAT, as an imunotherapeutic agent for Glioblastoma
40	Liu, Yufeng	Spatial Large-Margin Angle-Based Classifier for Multi-Category Neuroimaging Data

MD Anderson Cancer Center Postdoctoral Association

All postdoctoral fellows in The University of Texas MD Anderson Cancer Center are automatically members of the Postdoctoral Association (PDA). The PDA Executive Committee (PDAEC) strives to improve, enhance, and enrich the postdoctoral fellowship experience at MD Anderson by planning monthly events, annual science and career symposia, and fostering interactions between members of the Texas Medical Center (TMC) through social and career development events.

Ways to contact the PDA and find information:

- http://www.mdanderson.org/postdoctoralassociation
- postdoctoralassociation@mdanderson.org
- http://www.linkedin.com/groups?gid=4420426
- http://www.facebook.com/#!/groups/83955564561/
- http://www.twitter.com/MDApostdoc

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We would also like to recognize the contributions of Faculty and Academic Development, especially Dr. Tracy Costello, Office of Postdoctoral Affairs and Development, and Veronica Paniagua. Additionally, we thank Martha Skender in Academic and VISA Administration for supporting the inception of APSS six years ago and the teams in Medical Graphics and Photography for creating professional symposium materials.

We are indebted to the faculty judges from the Faculty Academic Review Committee for their constructive evaluation and feedback for our participants.

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A very special thank you to Dr. Zeinab Abou Yehia, who chaired the organizing committee before leaving MD Anderson earlier this year. Without her initial work and commitment to the organization, this symposium would not have been possible.

Finally, a big thank you to all of the members of PDAEC and specially our outgoing chairs Dr. Tara Dobson and Dr. Haley Peters - this symposium was made possible by all your hard work, organization, and support.

2016 APSS Organizing Committee
Dr. Pedro Correa de Sampaio, Chair
Dr. Taghreed Hirz, Vice-chair
Dr. Rajan Chaudhari, Member
Dr. Monica Reyes, Member
Dr. Lalit Sehgal, Member
Dr. Eleonora Dondossola, Member
Dr. Emanuela Gentile, Member

The Mission of APSS

The Annual Postdoctoral Science Symposium (APSS) was initiated on August 4th 2011, by the MD Anderson Postdoctoral Association to provide a platform for talented postdoctoral fellows to present their work to a wider audience.

APSS organizes a scientific symposium that incorporates all Texas Medical Center affiliated institutions. Our goal is to provide postdocs across the entire Texas Medical Center a unique opportunity to learn, interact and foster new collaborations.