The Division of Surgical Research is a new division in the Michael E. DeBakey Department of Surgery established to meet the challenges posed by an increasingly competitive and rapidly advancing research environment. The division brings together department researchers to “compare notes,” share ideas, benefit from each other’s knowledge and experience, and lend support in grant and publication efforts. At the same time, the division aims to create a critical mass of well-recognized researchers with whom other investigators at BCM and elsewhere can readily collaborate. The mission of the division is to promote the development and growth of highly successful research and training programs by providing a supportive environment for investigators and trainees. Division members strive to achieve two major goals to meet their overall mission.

1). To enhance communication and collaboration among investigators inside and outside of the division:
   --- Establish an effective administrative structure and a strategic plan for the growth of the division
   --- Promote a spirit of teamwork and collaboration among investigators and staff
   --- Strengthen collaborations between laboratory investigators and clinical partners inside and outside of the department
   --- Provide research infrastructure and support systems to all investigators

2). To increase the quality and impact of our research and training programs:
   --- Optimize research areas or topics by considering clinical significance, high innovation, new technologies, and the existing strengths of investigators and resources
   --- Increase research productivity including grants, publications, and presentations
   --- Translate discoveries from laboratory research to clinical practice
   --- Provide mentoring and training opportunities to junior faculty, surgical residents, fellows, and students
   --- Enhance the department’s national and international reputation for excellence in surgical research

**Faculty members**

*Primary faculty (12):*

*Changyi (Johnny) Chen, MD, PhD, Professor of Surgery*

*Xinhua Feng, PhD, Professor of Surgery*

*Qizhi (Cathy) Yao, MD, PhD, Professor of Surgery*

*Austin J. Cooney, PhD, Associate Professor of Surgery*

*Kaiyi (Kelly) Li, PhD, Associate Professor of Surgery*

*Xia Lin, PhD, Associate Professor of Surgery*

*Rita Serda, PhD, Associate Professor of Surgery*

*Megumi Mathison, MD, PhD, Associate Professor of Surgery*

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Lidong Liu, PhD, Assistant Professor of Surgery
Stuart Corr, PhD, Assistant professor of Surgery
Jian-Ming Lu, PhD, Assistant Professor of Surgery
Hu Ying Shen, MD, PhD, Assistant Professor of Surgery
Yulong Liang, PhD, Instructor in Surgery

Joint faculty (15):
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William E. Fisher, MD, Professor of Surgery
Ronald H. Kerman, PhD, Professor of Surgery
Scott A. LeMaire, MD, Professor of Surgery
George P. Noon, MD, Professor of Surgery
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Sanjeev Vasudevan, MD, Assistant Professor of Surgery
Lalita Wadhwa, PhD, Assistant Professor of Surgery
Pawel Kolodziejski, MD, PhD, Instructor of Surgery
Xiaoying Shang, PhD, Instructor in Surgery
Research Interests

My laboratory is actively conducting several basic science and translational research projects that are highly relevant to clinical cardiovascular disease and pancreatic cancer.

Cardiovascular risk factors and their molecular mechanisms in cardiovascular disease
We are investigating the effects and the molecular mechanisms of several cardiovascular risk factors, including HIV protease inhibitors, the adipokine resistin, soluble CD40L, and uric acid, on biochemical pathways associated with endothelial cell functions. Some of the biochemical pathways under investigation are the endothelial nitric oxide synthase system, the oxidative stress system, and signal transduction pathways. We are carrying on these investigations using several experimental models, such as myographies, organ cultures, mouse models, human tissue samples, and different types of endothelial cells. Based on the molecular mechanisms we uncover, we develop effective therapeutic strategies to treat endothelial dysfunction and atherosclerosis.

Endothelial cell differentiation and angiogenesis
We are studying the role played by and the molecular mechanisms of hemodynamic factors and several novel molecules on endothelial cells differentiated from embryonic stem cells and from bone marrow-derived stem cells. We are identifying key regulatory genes that trigger endothelial cell differentiation and promote stable angiogenesis. These findings can potentially be applied to the design of novel therapeutic strategies to treat ischemic tissues using genetically engineered endothelial cells. In addition, these studies may provide useful information to genetically engineer novel tissues for vascular grafts.

Pancreatic cancer
We have been heavily involved in pancreatic cancer research programs for many years. We have several projects focusing on the role and on the mechanisms of several genes, such as microRNA 196a (miR-196a), X-inactive specific transcript (XIST), and Jude-2 in pancreatic cancer. Our
comprehensive studies analyze human cancer specimens, clinical outcomes, established cell lines, a nude mouse model, and a genetically engineered mouse model of pancreatic cancer called the KPC model. We are developing PLGA [poly(lactic-co-glycolic acid)]-based nanotechnology for molecular imaging and for specific drug and gene delivery, which has great potential clinical applications, such as molecular diagnostics and targeted therapies.

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Selected Publications


**Key words (disease and expertise):**

- Angiogenesis
- Atherosclerosis
- Cardiovascular disease
- Endothelial dysfunction
- Endothelial nitric oxide synthase
- Hemodynamics
- Oxidative stress and antioxidant
- Pancreatic cancer
- PLGA-based nanotechnology
- Vascular tissue engineering
Xin-Hua Feng, PhD

Professor
Michael E. DeBakey Department of Surgery
Department of Molecular & Cellular Biology

Education
PhD: University of Maryland, College Park
Postdoctoral training: University of California, San Francisco

Research interests

Protein modifications and signaling networks in cell growth control, tumorigenesis, and development

My research aims to elucidate the underlying mechanisms and interplays among protein modifications, signaling pathways, and gene transcription as well as understanding their roles in cell proliferation, tissue differentiation, and pathogenesis of human diseases.

My current research projects include:

Phosphatome: genome-wide investigation of protein dephosphorylation
Signal transduction pathways are often regulated by the dynamic interplay between protein kinases and phosphatases. Using all the human protein serine/threonine phosphatases available, we systematically investigate the effect of dephosphorylation on key proteins involved in cell signaling and cell functions. We are currently genetically disrupting individual phosphatases to elucidate their in vivo functions during development.

SUMO, ubiquitin, and control of protein turnover and functions
We examine the effect of post-translational modifications, particularly ubiquitination and SUMOylation of transcription factors, in normal and cancer cells. We attempt to understand the molecular mechanisms by which environmental and developmental cues regulate the ubiquitination/proteasome and SUMOylation systems. Our studies will provide insights into the relationships between protein deregulation and human cancers or abnormal development.

TGF-β/BMP signal transduction
SMADs are evolutionarily conserved signal transducers and transcription factors controlling TGF-β/BMP functions. A large number of mutations that inactivate SMADs have been linked to human cancers and genetic diseases. We address the molecular interactions, requirements, and functionality of SMADs in TGF-β/BMP responses using cellular, genomic, and proteomic
approaches. We investigate how SMADs mediate transcription and how their actions are terminated. We also use in vitro and in vivo model systems to study how SMADs as tumor suppressors interplay with oncogenic pathways, in particular with those involved in lymphoma and in pancreatic and breast cancer.

**Genetic screens, BMP/TGF-ß signaling, and ES cells**

We are conducting genome-wide studies (e.g. genetic screens using lentiviral RNAi library) to identify novel TGF-ß signal modifiers or regulators involved in stem cell differentiation. Novel molecules that control TGF-ß/BMP signaling or participate in human ES cell self-renewal and differentiation will be further studied and in model organisms to define the molecules’ physiological roles in tissue differentiation and organ development.

**Immune suppression by TGF-ß**

TGF-ß is a major inflammatory and immune-regulatory cytokine, but the mechanisms by which TGF-ß exerts its actions are unclear. We are interested in investigating the signaling interactions between the TGF-ß pathway and other cytokine pathways (such as TNF-alpha, IL-1, and IL-6 pathways) in immune responses. This area of research may lead to the discovery of drugs to treat cancer and inflammatory diseases.

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**Selected publications**

1. Dai F, Lin X, Chang C, **Feng XH**. (2009), Nuclear export of Smad2 and Smad3 by RanBP3 facilitates termination of TGF-ß signaling. *Dev Cell*, 16(3):345-357.


Keywords (disease and expertise):

- Embryonic stem cells
- Serine/threonine phosphatases available
- SMADs
- SUMOylation
- TGF-ß/BMP
- Ubiquitination
Qizhi Cathy Yao, MD, PhD

Professor
Michael E. DeBakey Department of Surgery
Department of Molecular Virology and Microbiology
Department of Pathology and Immunology

Education
MD: Southeast University School of Medicine, China
PhD: Emory University School of Medicine, Atlanta, GA
Postdoctoral training: Emory University School of Medicine, Atlanta, GA

Research Interests

My research programs include HIV vaccine development, pancreatic cancer pathogenesis, and therapy. Specifically:

- Developing chimeric virus-like particle HIV vaccines
- Understanding the functional roles of mesothelin in pancreatic cancer pathogenesis
- Understanding the functional roles of miR-198 in pancreatic cancer pathogenesis
- Understanding the functional roles of axon guidance gene Semaphorin 3E in pancreatic cancer pathogenesis
- Developing targeted nanoparticle therapy in pancreatic cancer
- Developing immunotherapy for pancreatic cancer

HIV Vaccines

My lab is interested in developing non-infectious HIV virus-like particles (VLPs) as candidate HIV mucosal vaccines for both preventive and therapeutic purposes. In preclinical studies, VLPs formed by structural proteins are highly immunogenic and capable of inducing protective immunity against various viral infections. We have modified vaccine immunogens into chimeric HIV VLPs which contain influenza viral surface glycoprotein HA or other immunologically functional molecules. We have shown that the chimeric HIV VLPs can induce enhanced humoral and cellular immune responses against HIV in a mouse model.

We have also studied the basic mechanisms of VLP-induced humoral and cellular immune responses, and other factors that affect these responses. For example, we found that VLP vaccines activate conventional B2 cells and promote B cell differentiation to IgG2a producing plasma cells; that VLP vaccines travel to the lymph nodes upon immunization and can be directly visualized by optical imaging techniques; and that intradermal immunization generates improved responses and might be a preferable delivery route for viral and cancer immunotherapeutic studies involving VLPs.
Since dendritic cells (DCs) have long been known to be pivotal in initiating immune responses, we are also interested in how VLPs modulate DC functions and will evaluate the efficacy of VLP-pulsed DC vaccines. In addition, we are interested in testing the efficacy of modified chimeric VLP oral-mucosal immunization in non-human primates.

**Pancreatic cancer pathogenesis and therapy**

Pancreatic cancer has one of the highest mortality rates and ranks as the fourth leading cause of cancer death in North America. Survival is poor because there are no reliable tests for early diagnosis and no effective therapies to treat metastatic disease. There is a need to better understand the molecular mechanisms of pancreatic cancer tumorigenesis and to develop effective treatments. My lab currently focuses on the study of key molecules in pancreatic cancer, including mesothelin (MSLN), trop2, and semaphorin 3E, and in their mechanisms of regulation. I am also interested in the involvement of microRNAs (miR-198) in pancreatic cancer, and how their dysregulation leads to pathogenesis. We are also currently exploring tumor-associated molecule targeted therapies and RNA interference delivery by liposomes and PLGA nanoparticles *in vivo*. Our group has shown that vaccinating mice with chimeric virus-like particles containing MSLN significantly inhibited tumor progression, suggesting a new therapeutic vaccine strategy whereby MSLN is targeted to attempt to control pancreatic cancer progression. We are also employing a K-ras mutation spontaneous pancreatic cancer mouse model to study prevention and the potential of our therapeutic regimens.

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**Selected Publications**


**Key words (disease and expertise):**

- Breast cancer
- HIV
- Immunotherapy
- Mesothelin
- MicroRNA
- Nanoparticle targeted delivery
- Pancreatic cancer
- Vaccine
Austin J. Cooney, PhD

Associate Professor
Michael E. DeBakey Department of Surgery
Department of Molecular and Cellular Biology

Education
PhD: National University of Ireland, Galway
Postdoctoral training: Baylor College of Medicine

Research Interests
My laboratory is actively conducting several basic science and translational research projects that are highly relevant to clinical cardiovascular disease and regenerative medicine. The focus of my research group over the last decade and a half has been the transcriptional regulation of the pluripotent state in embryonic stem (ES) cells and their differentiation. Coming from a nuclear receptor background, I naturally focused on this family of ligand-activated transcription factors, which I found to play key roles in regulating ES cell differentiation. My research group has four broad focuses: (1) the maintenance of pluripotency through the nuclear receptor liver receptor homolog-1 (LRH-1), which interacts with Wnt/β-Catenin signaling; (2) silencing of pluripotency gene expression via the nuclear receptor germ cell nuclear factor (GCNF); (3) generation of induced pluripotent stem (iPS) cells focusing on nuclear receptors (NRs); and (4) ES cell differentiation into cardiomyocytes.

Differentiation of ES and iPS cells into cardiomyocytes
In terms of differentiation into cardiomyocytes, we have several projects in progress. For the purpose of modeling regenerative medicine, we generated mouse iPS cells and efficiently differentiated them into functional cardiomyocytes to study the regulation of this process. With Robert Schwartz, we collaborate on defining the roles of early lineage determinants in cardiac development. We have focused on the transcription factor Mesp1 and on Wnt signaling. However, it is our work with LRH-1 in ES cells that has pushed us further into understanding cardiac differentiation. We made the novel observation that over-expression of LRH-1 in ES cells leads to a dramatic increase in the number of beating colonies after differentiation. We have shown that the nodal coreceptor Cripto is an LRH-1 target gene. Cripto is highly expressed in ES cells, is rapidly down-regulated upon differentiation, and its expression is specific to the cardiac crescent. Using various Cre drivers developed in the Schwartz lab for early cardiac development, we will study the yin-yang roles of LRH-1 and GCNF in cardiac development using Cre/Lox approaches. In parallel with these genetic approaches, we will test the effects of LRH-1 and GCNF ligands on improving iPS generation and cardiomyocyte differentiation. Our goal is to translate these novel findings to human ES and iPS cells.
**In vitro and in vivo cardiac regeneration**

In collaboration with Dr. Todd K. Rosengart, we are developing virus-based strategies to treat cardiovascular diseases, such as infarction *in situ*. The goal is using viral vectors to induce transdifferentiation of cardiac fibroblasts and myofibroblasts into functional cardiomyocytes *in situ* in a patient's heart. We are modeling and developing the processes in rats, pigs, and in human cardiac fibroblasts.

**Silencing of pluripotency gene transcription**

Lrh-1 and GCNF, which are orphan members of the steroid receptor gene family of ligand-activated transcription factors, play yin/yang roles in regulating pluripotent gene expression. We showed that Lrh-1 maintains pluripotent gene expression in response to canonical Wnt signaling through βCatenin. GCNF is the major transcriptional repressor of pluripotency gene expression during the exit from this distinct phase in development, which is initiated by differentiation or gastrulation. GCNF silences pluripotency gene expression by the recruitment of the DNA methylation machinery. We have established GCNF knockout (KO) ES cells as a genetic model. We use proteomic and genomic strategies to dissect the role of GCNF in the regulation of DNA methylation, which is of fundamental importance. We are also testing GCNF ligands, which act as antagonists on ES cell differentiation.

**Generation of patient-specific iPS cell lines**

We are using genetic and pharmaceutical approaches to study the roles of Lrh-1 and GCNF during iPS formation. Using Cre/lox approaches, we will analyze the roles of LRH-1 and GCNF in iPS formation to generate KO fibroblasts for each factor. We will also test the ligands for LRH-1 and GCNF to determine if they promote iPS formation and quality.

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**Selected Publications**


**Key words (disease and expertise):**
- Cardiac differentiation
- Embryonic stem cells
- Gene regulation
- Generation of iPS cells
- Hemodynamics
- Nuclear receptors
- Oxidative stress and antioxidant
- Pluripotent stem cells
- TGA-based nanotechnology
- Vascular tissue engineering
Kaiyi (Kelly) Li, PhD

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Education
PhD: The University of Texas M.D. Anderson Cancer Center, Houston
Postdoctoral training: Baylor College of Medicine, Houston

Research Interests

My research goal is to develop novel cancer therapies by identifying new key pathways for cancer development and progression.

There are three major areas of investigation in my laboratory:

Characterization of the function of DNA-repair proteins in tumor suppression using both knockout mouse models and clinical specimens
BRIT1/MCPH1 knockout mice have been generated in the lab and BRIT1’s role in the suppression of breast, liver, and pancreatic cancer is studied extensively using the unique knockout mouse model, as well as clinical specimens.

Development of cancer-specific therapies by targeting DNA repair deficiency in cancer
We use a synthetic lethality approach and combination therapy to develop more effective treatments for breast and liver cancer.

Identification of novel genes that drive breast and liver cancer development
Using a bio-informatics approach, we select candidate genes analyzing The Cancer Genome Atlas (TCGA) data and we characterize the genuine functions of these candidate genes in vitro and in animal models.

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Selected Publications


Key words (disease and expertise):

- Breast cancer
- DNA damage response pathways
- DNA repair
- Knockout mouse model
- Liver cancer
- Pancreatic cancer
- Synthetic lethality
- Targeted cancer therapy
- Tumor Suppressor
Xia Lin, PhD

Associate Professor
Department of Surgery

Education
Ph.D.: University of Maryland at College Park, College Park, MD
Postdoctoral training: University of California at San Francisco, San Francisco, CA

Research Interests

My research interest is on cell functions under physiological and pathological conditions. Currently, we are investigating several cell functions such as cell proliferation, differentiation, and metabolism by focusing on protein phosphatase. Specifically, we are trying to identify protein phosphatases that regulate critical signal transduction pathways such as BMP, TGF-β, insulin pathways, and gluconeogenesis. By doing this, we hope to understand better the signaling pathways that regulate normal cellular functions, and the deregulation of them leads to human diseases such as cancer, which is our main focus, bone disease, and diabetes. Eventually, we hope to provide the rationale for protein phosphatases as potential therapeutic targets.

Another major focus of my research is on the functions and regulation of TGF-β signal transduction pathway. We also investigate the crosstalk of TGF-β signal with other signaling pathways such as oncogenic pathway and hormone receptor pathway, and the role of protein posttranslational modifications (e.g. phosphorylation, ubiquitination and sumoylation) in TGF-β functions. By using cell-based assays and animal models, we seek to determine the role of TGF-β in normal cellular functions, cancer initiation, and cancer progression. Ultimately, our studies will advance our knowledge on understanding the molecular mechanisms of cancer initiation and progression, and on the identification of potential targets for cancer therapy.

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Selected Publications


Key words (disease and expertise):
- Cancer
- Cell cycle regulation
- Diabetes
- Mouse development
- TGF-β signaling
Dr. Rita Serda earned her PhD in Biomedical Sciences in 2006. She held faculty appointments at the University of Texas School of Medicine and the University of Texas Graduate School of Biomedical Sciences in Houston, before becoming an Assistant Member of The Methodist Hospital Research Institute in 2010. At Methodist, Dr. Serda was Interim Co-Chair for the Department of Nanomedicine, Director of the Scanning Electron and Atomic Force Microscopy Core, and Faculty Director of The Methodist Hospital Research Institute Academy for Medical Science and Technology. Dr. Serda joined Baylor College of Medicine in January 2014.

Hyperthermia-driven immunotherapy.
Dr. Serda’s research interests center on using nanotechnology and radio waves to drive accumulation of therapeutics at sites of pathology with a major goal of stimulating anti-cancer immune responses. Using a portable radiofrequency (RF) device integrated with an intravital microscope, our research team has demonstrated that RF-induced hyperthermia stimulates an increase in intratumoral vascular flow and accumulation of macromolecules and nanoparticles (NPs). RF-induced hyperthermia further stimulates the production of immunogens, increases vascular permeability, and increases susceptibility to additional adjuvant therapies. My research exploits these traits to achieve specific localization of immune modulating NPs within the tumor. As an example, we use adeno-associated viral (AAV)-mediated gene therapy for immune stimulation and blockade of immune suppression.

Mass Transport of Nanotherapeutics.
Barriers to the transport of therapeutics include the vascular endothelium, interstitial and stromal components, cellular membranes, and intracellular organelles. Sequential targeting and delivery of agents is achieved by presentation of agents in carrier particles, termed Logic-Embedded Vectors (LEVs). LEVs integrate micro- and nano-particles into multi-dimensional functional entities that have the ability to act at multiple levels to bypass biological barriers and deliver therapeutic payloads to desired cell populations and intracellular organelles. My research utilizes intravital multiphoton, tissue scanning and transmission electron microscopy to image in vivo transport phenomena.

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**Selected Publications**


**Key words (disease and expertise):**

- Cancer nano-therapeutics
- Immunotherapy
- Therapeutic hyperthermia
Dr. D. Srivastava and his colleagues reported that the combination of three transcription factors, Gata4, Mef2c, and Tbx5, reprogrammed postnatal murine cardiac fibroblasts directly into differentiated cardiomyocyte-like cells \textit{in vitro}.

Furthermore, Dr. Srivastava and others, including us, recently reported that resident cardiac fibroblasts were reprogrammed into cardiomyocyte-like cells in the murine heart by direct injection of Gata4, Mef2c, and Tbx5 into the myocardium after coronary ligation. These results raise the possibility that we can generate new cardiomyocytes from scar tissue after myocardial infarction in humans.
Selected Publications


Key words (disease and expertise):

• Cardiovascular disease
• Cardiac regeneration
Narasimhaswamy S. Belaguli, PhD

Assistant Professor
Michael E. DeBakey Department of Surgery
Michael E. DeBakey Veterans Affairs Medical Center

Education
BVSc: Bangalore Veterinary College, Karnataka, India
MVSc: Bangalore Veterinary College, Karnataka, India
PhD: Memorial University, Newfoundland, Canada
Post-doctoral training: Baylor College of Medicine, Houston, Texas

Research Interests

My lab is interested in understanding the transcriptional regulatory mechanisms involved in the induction and maintenance of differentiation programs in various cell types such as myogenic cells, pancreatic beta cells, gastrointestinal epithelial cells, and colorectal cancer cells.

Myogenic cells

In depth studies over the last three decades have helped understand the mechanisms by which cells commit to a particular cell lineage, undergo differentiation, and maintain the differentiated phenotype. The differentiation program unique to a cell type can be switched and cells are made to assume an alternate differentiation program without transitioning through an intermediate pluripotent “stem cell” phase by a process called “transdifferentiation.” Previously, I have determined that fibroblasts can be transdifferentiated into vascular smooth muscle cells by overexpressing three transcriptional regulators, SRF, GATA6, and CRP2, which are highly enriched in vascular smooth muscle cells. More recent studies from several labs have shown that fibroblasts can also be transdifferentiated into cardiac muscle cells by overexpressing a defined set of cardiac muscle-enriched transcriptional regulators. The mechanisms and signaling events involved in transdifferentiation of fibroblasts in to cardiac myocytes is an area of active research in my laboratory.

Pancreatic beta cells

Pancreatic beta cells secrete insulin, a vital hormone that regulates blood glucose levels. Insulin deficiency and/or inefficient utilization of insulin cause diabetes. We have identified serum response factor (SRF) as a beta cell-enriched transcriptional regulator of insulin gene expression. My lab has been using genetically modified mice, genomic, transcriptomic, and biochemical approaches to investigate the mechanisms by which SRF and co-accessory factors regulate beta cell gene expression and glucose homeostasis.
**Gastrointestinal epithelial cells**

The mammalian intestine is one of the organs in which the epithelium is rapidly and perpetually turned over. Several growth factors, signaling molecules, and transcriptional regulators are involved in maintaining intestinal epithelial homeostasis. GATA factors are zinc-dependent transcriptional regulators important for proliferation and differentiation of intestinal epithelial cells. To identify proteins that cooperate with GATA factors to regulate intestinal gene expression I have employed yeast-two-hybrid screens, proteomics, and biochemical approaches.

**Colorectal cancer**

Colorectal cancer is the third most commonly diagnosed cancer. Development and metastasis of colorectal cancer is associated with increased expression of GATA6. I have been using transcriptional profiling of colorectal cancer cells, human cancer tissue arrays, and biochemical approaches to examine the mechanisms by which GATA6 and GATA6-interacting factors promote colorectal cancer development and metastasis.

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**Selected Publications**


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**Keywords (disease and expertise):**

- Colorectal cancer
- Gastrointestinal epithelial cells
- GATA6
- Myogenic cells,
- Pancreatic beta cells
- Serum response factor (SRF)
- Transdifferentiation
Lidong Liu, PhD
Assistant Professor
Michael E. DeBakey Department of Surgery

Education
BS and MS: Wuhan University, China
PhD: Kyoto University, Japan
Postdoctoral training:
Biomolecular Engineering Research Institute, Japan
University of Washington, Seattle

Research Interests
I am interested in understanding the signaling transduction mechanisms that regulate cancer initiation, progression, and metastasis.

My current research is focused on two areas:

TGF-β signaling in cancer
I am investigating the molecular mechanisms of TGF-β signaling involved in cancer progression and metastasis, with special emphasis on the roles Smads play as regulators and mediators of TGF-β signaling via cross-talk with other signaling pathways.

EMT in cancer
I am studying the molecular basis of epithelial-mesenchymal transition (EMT) and the roles of EMT and stem-like cells generated by EMT in cancer invasion and metastasis. The ultimate goal of my research is to reveal the molecular basis of malignancy and discover targets for cancer treatment.

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Selected Publications


Key words (disease and expertise):

- Cancer stem cell
- Epithelial-mesenchymal transition (EMT)
- Prostate cancer
- TGF-β
Dr. Corr obtained his BEng (hons) in Electronics with Music from The University of Glasgow. He went on to study an MEng in Electrical Systems majoring in Nano-electronics and Photonics at Dublin City University, Rep. of Ireland. During this period, he was selected for the Irish Government sponsored FÁS Science Challenge, which placed him in Dr. Lon J. Wilson's Nano-materials group at Rice University, Department of Chemistry where he worked on building a prototype to quantify cyclic magnetic field absorption by gadolinium loaded ultrashort carbon nanotubes (Gd3+ US-SWNTs), which are used as superlative MRI contrast agents. Having returned to Dublin to finish his masters he then completed his PhD studies, over a period of two years, in the field of silver nanoparticles and thin-films for surface enhanced raman spectroscopy of strained silicon. He subsequently spent a 3-month period at the International Space University, Strasbourg, France, as part of their MSc. in Space Studies program - a program which he is still affiliated with. With this knowledge and experience he was asked to return to Rice as a postdoctoral fellow, to synthesize and apply silver nanoparticles to non-invasive radio frequency (RF) hyperthermia, which was part of an active collaboration between Prof Wilson and Dr. Steven A. Curley, at MD Anderson Cancer Center. Since working on this project in 2009, Dr. Corr eventually transferred over to MDACC in 2011 and has since been involved in the research and development of nanoparticle-assisted non-invasive RF hyperthermia.

RF technology is gaining prominence as a powerful new surgical oncology tool in the fight against cancer. Recent work has shown synergy when combining RF therapy with systemic chemotherapy administration. His current studies have also shown enhanced delivery and retention of chemotherapeutics into tumors when exposed to low levels of RF fields. He is currently active in this field and we are currently seeking FDA approval for full human trials.

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Jian-Ming Lü, PhD
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Michael E. DeBakey Department of Surgery

Education
PhD: Lanzhou University, Lanzhou, China
Postdoctoral training:
University of Texas, Health Science Center at Houston
University of Houston
Leipzig University

Research Interests
My research is focused on several basic science and translational research projects that are highly relevant to clinical diseases and pancreatic cancer. I have a strong background and research experience in organic chemistry, medicinal and synthetic chemistry, and biochemistry, including enzyme activities and mechanisms.

In recent years, I have been studying the fields of translational medicine and medicinal chemistry, working with cell-free, well-established in vitro as well as in vivo models. The primary goal of my projects is to develop new, safe, and effective therapies using natural or naturally-derived substances. For example, I have been developing medicines for hyperuricemia-related diseases, such as gout, using natural substances and by modifying their structure to enhance their effects. Currently, I am also screening naturally-derived substances for inhibitors of enzymes such as myeloperoxidase, HIV protease, and arginase, key enzymes in the development of diseases.

Another focus of my research is the delivery of nanoparticle gene/drug complexes targeted to cancer cells as well as to vascular cells by using antibodies or other specific proteins conjugated to PLGA (poly(lactic-co-glycolic acid)-based nanoparticles. I am developing a new PLGA-based material for molecular imaging and specific drug and gene delivery, which has great potential clinical applications such as molecular diagnostics and targeted therapies.

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Selected Publications


Key words (disease and expertise):

- Cardiovascular disease
- Drug discovery and development
- Enzyme inhibitors, mechanisms
- Gout and hyperuricemia
- Natural substances and structure modification
- Organic synthesis, characterization
- Oxidative stress, free radicals, and antioxidants
- Pancreatic cancer
- Polymer nanoparticle drug/gene delivery
- Xanthine oxidase, HIV protease, cyclooxygenase, arginase
Ying H. Shen, MD, PhD

Assistant Professor
Michael E. DeBakey Department of Surgery
Director of Cardiothoracic Surgery Research Laboratory

Education
MD: Beijing Medical College, Beijing, China
PhD: University of New South Wales, Sydney, Australia

Research Interests
My broad research interest is on vascular diseases. One of my main interests is to study the molecular mechanisms of aortic aneurysms and dissections, highly lethal but poorly understood conditions. During the past few years, we have established mouse models of aortic aneurysms and dissections and developed various techniques to evaluate the aortic structure and functions. We have also developed several projects to study the regulation of aortic inflammation and destruction, as well as aortic repair and remodeling.

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Selected Publications


Key words (disease and expertise):

- Aortic aneurysms and dissections
- Diabetic vascular diseases
- Vascular biology and diseases
Yulong Liang, PhD
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Education
PhD: Fudan University Shanghai Medical College, China
Postdoctoral training: Baylor College of Medicine, Houston

Research Interests
My research focuses on elucidating the roles and the underlying mechanisms of DNA damage and repair pathways in tumor development, progression, and metastasis, as well as developing novel therapeutic methods to target cancer cells.

DNA damage response and genomic instability in cancer
DNA repair deficiency and genomic instability are important hallmarks of cancer. By elucidating the roles of BRIT1/MCPH1, an important protein involved in DNA damage and repair pathways, I will provide insights into the relationship of DNA repair deficiency with genomic instability, cancer initiation, progression, and/or metastasis.

Translational research and treatment of cancer
In this area, I will investigate how to target cancer cells with genomic instability, which may eventually lead to the discovery of drugs for cancer treatment.

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Selected Publications


**Key words (disease and expertise):**

- Breast cancer
- DNA damage response
- Double-strand breaks
- Genomic instability
- Homologous recombination
- Liver cancer
- Synthetic lethality model
• Targeting therapy of cancer
• Tumorigenesis