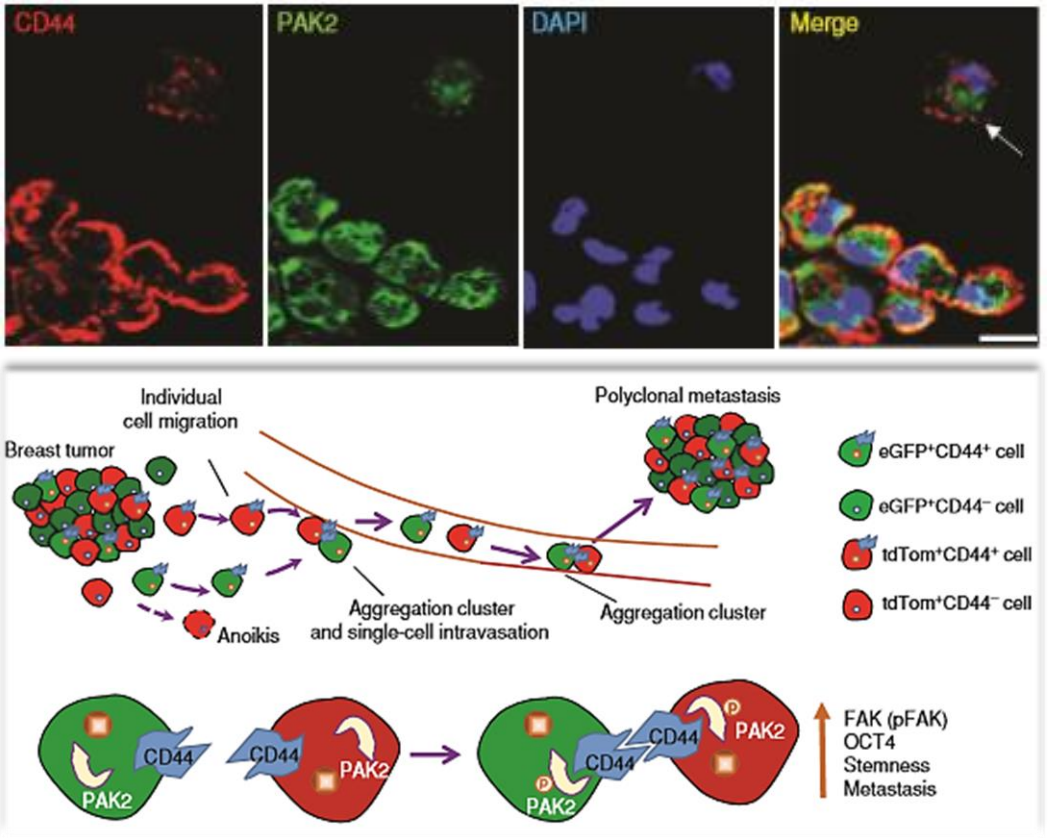
ACACR NewsLetter

Association of Chinese Americans in Cancer Research

Volume 2
Issue 6
February 2020



Liu et.al., Cancer Discovery, 2019

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ACACR 2020 annual meeting announcement

ACACR annual meeting 2020 will be held on April 25, 2020 at UCSD, San Diego, California

Member Introduction

Dr. Zhenkun Lou is a Professor and Co-Leader of Experimental Therapeutic Program at Mayo Clinic. He is an expert on DNA repair mechanisms and DNA damage signaling pathways.

Dr. Lou received his B. S. degree from East China University of Science and Technology and his Ph.D. degree from Mayo Clinic College of Medicine in 2001. He received his postdoctoral training under Dr. Junjie Chen at Department of Oncology, Mayo Clinic when he started working on DNA damage signaling pathways. He cloned MDC1 in Dr. Chen's lab and showed it is an early DNA damage response factor that docks onto γH2AX following DNA damage and helps recruit BRCA1 and 53BP1. He established his own lab in 2006 as an Assistant Professor of Pharmacology at Mayo Clinic and rose to the rank of full Professor in 2013. Dr. Lou keeps working on DNA damage signaling pathway after he established his own lab. He focuses on identifying new DNA repair factors as well as posttranslational modifications (PTMs) of DDR factors, such as ubiquitination, sumoylation, phosphorylation, acetylation, and methylation. These studies revealed new mechanisms that regulate cell cycle progression, recruit DNA repair factors to the damage sites, and coordinate DNA repair. These studies also have clinical implications as they elucidated how cancer cells respond to anti-cancer therapies such as radiation and PARP inhibitor. Dr. Lou also studies the AMPK-SIRT1 pathway and revealed new mechanisms contributing to cellular energy metabolism, inflammation and healthy aging.

Dr. Lou is the current president of Association of Chinese Americans in Cancer Research (ACACR). His work was/is supported by awards from Susan G Komen Foundation, Richard B. Schulze Family Foundation, DOD and NIH. Dr. Lou serves on several NIH grant review panels and AACR committees. He is on the Editorial Board of ***Journal of Biological Chemistry*** and ***Cancer Research***. He is also an Associate Editor of ***Genes & Diseases*** and ***Genome Instability & Disease*****.**

Member Introduction

Shi-Yuan Cheng, PhD
Professor of Neurology
Northwestern University, Chicago, USA

Dr. Cheng is currently a tenured Professor of Neurology at The Ken & Ruth Davee Department of Neurology, Lou & Jean Malnati Brain Tumor Institute, and the Lurie H. Robert Comprehensive Cancer Center, Northwestern University Feinberg School of Medicine, Chicago, IL USA. Dr. Cheng was the President of US Chinese Anti-Cancer Association (USCACA) in 2013-2017, a non-profit professional organization that facilitates collaborations among cancer researchers and physicians in US and China. Dr. Cheng was also the funding president of Association of Chinese American in Cancer Research (ACACR).

Dr. Cheng received his BS degree in biochemistry from Wuhan University in Wuhan, China in 1982 and his PhD degree in biochemistry from The Ohio State University in Columbus, Ohio, USA in 1992. From 1992 to 1999, Dr. Cheng received his postdoctoral trainings at UCSD and the Ludwig Institute for Cancer Research in La Jolla, California, USA. From 1999 to 2012, Dr. Cheng was appointed as an Assistant Professor then a tenured Associate Professor at University of Pittsburgh Cancer Institute (now Hillman Cancer Center) & Department of Pathology at University of Pittsburgh School of Medicine, Pittsburgh, PA. In 2012, Dr. Cheng joined faculty at Northwestern University as a tenured Professor and an Associate Scientific Director of Lou & Jean Malnati Brain Tumor Institute. Dr. Cheng was honored as a Zell Scholar by Zell Family Foundation at Northwestern University. He was also honored as a Kimmel Scholar by the Sidney Kimmel Foundation for Cancer Research, a V Scholar by the V Foundation for Cancer Research and Wang Kuan-Cheng Award for Outstanding Oversea Young Scientist, Chinese Academy of Sciences, China. Dr. Cheng is/was an editorial board member of Neuro-Oncology, Journal of Biological Chemistry, Journal of Neuro-oncology. Dr. Cheng has published ~90 peer-reviewed papers in top-ranking biomedical journals as first, senior or co-author including *Cancer Cell*, *Nat Cell Biol*., *JCI*, *Nat Commun., PNAS*, *Cell Reports, Cancer Res.* and ten invited review articles and book chapters. Dr. Cheng has been an expert reviewer including several terms of charter members for numerous study sections at NIH, Army Breast Cancer Research Program, Susan G. Komen Foundation, Juvenile Diabetes Research Foundation, Medical Research Council (England), Cancer Research UK, Canada Foundation for Innovation and Fund, National de la Researche, Luxemboug and others. Dr. Cheng's research has been continuously supported by grants from the US NIH, American Cancer Society, The US Amy Breast Cancer Research Program, and other agencies. He currently holds two active NIH R01s, a proved to be activated NIH R01, a project of NCI Specialized Programs of Research Excellence (SPORE) on Brain Cancer, and sponsors a NIH F31, and a K00 awardees. Dr. Cheng is a regular reviewer for >80 peer-review scientific journals. Dr. Cheng's research interests are to study dysregulated oncogenic signaling, non-coding RNAs, RNA splicing, autophagy, and epigenetics in brain gliomas and develop novel therapeutic approaches for treating brain tumors and other human cancers.

2019 ACACR Annual Meeting Report - Peter Zhou

The third annual meeting of the Association of Chinese Americans in Cancer Research (ACACR) was held in conjunction with the annual AACR meeting in the beautiful city of Atlanta, GA on April 1, 2019. More than 150 cancer researchers around the world attended this important meeting.

After a brief welcome and introduction, the meeting was kicked off with an exciting scientific presentation from Dr. Haian Fu, endowed professor and Chair of Department of Pharmacology and Chemical Biology at Emory University School of Medicine. Dr. Fu and his team have developed a unique platform to identify global protein-protein interactions in various tumor cell lines. In particularly, he and his colleagues identified key protein interaction networks of many oncogenes and tumor suppressor genes. These protein interaction networks lay a solid foundation for screening small molecules that can disrupt these key node interactions in suppressing tumor growth.

Dr. Jean Zhao, professor of Biological Chemistry and Molecular Pharmacology at Harvard Medical School and Dana-Farber Cancer Institute, gave the third presentation. Dr. Zhao and her group identified key development in integrating targeted therapy with immune check-point therapy in treating breast cancer.

Dr. Wei Zhang, professor and director of Center for Genomic and Precision Oncology in Wake Forest University Comprehensive Cancer Center, delivered the second scientific presentation. Dr. Zhang provided a comprehensive overview and most recent updates on cancer genetic mutations and their implications on precision oncology.

Dr. Zhenghe Wang reviewed the most updates of ACACR Newsletter and the acquisition of Genes & Diseases as the first ACACR society journal from February 2019. Many ACACR members have served as editor-in-chief (Dr. Tong-Chuan He), senior editors (Drs. Zhenghe Wang, Lin Zhang, Gloria Su and Peter Zhou), and editorial board members for Genes & Diseases, which will likely have an impact factor on 2020. Dr. Wang encouraged all members to submit their exciting research studies for publications in Genes & Diseases. Dr. Yong Li briefly discussed the annual financial report. Dr. Zhenkun Lou, past president of ACACR, provided a closing remark and announced that Dr. Lin Zhang will be the president-elected of ACACR for Year 2019-2020. Finally, all rejoiced during the social event with a variety of delicious Chinese foods and group photos. This annual meeting was organized by Drs. T.-C He, Shuli Xia, Gloria Su and Peter Zhou with great help from several faculty members from Georgia State University and Emory University, including Drs. Jenny Yang, Haian Fu, and Shi-Yong Sun. Tannon and MedChemExpress graciously sponsored this annual meeting.

2019-2020

RESEARCH HIGHLIGHTS

Zhenkun Lou lab at Mayo Clinic reported that ZFP161 regulates replication fork stability and maintenance of genomic stability by recruiting the ATR/ATRIP complex. Their findings suggest that ZFP161 coordinates ATR/Chk1 pathway activation and helps maintain genomic stability. https://www.nature.com/articles/s41467-019-13321-z.pdf. In another recent paper, his lab discovered that the AMPK−Parkin axis negatively regulates necroptosis by inhibiting RIPK1−RIPK3 complex formation; this regulation may serve as an important mechanism to fine-tune necroptosis and inflammation. https://www.nature.com/articles/s41556-019-0356-8.pdf. Moreover, his lab found that a novel UCHL3 inhibitor, perifosine, enhances PARP inhibitor cytotoxicity through inhibition of homologous recombination-mediated DNA double strand break repair. Their finding provides a novel therapeutic approach to optimize PARP inhibitor treatment efficiency. https://www.nature.com/articles/s41419-019-1628-8.pdf. Furthermore, his lab claimed that PD-L1 (B7-H1) competes with the RNA exosome to regulate the DNA damage response and can be targeted to sensitize to radiation or chemotherapy. https://www.ncbi.nlm.nih.gov/pubmed/31053471. Finally, his lab discovered that UFL1 promotes histone H4 ufmylation and ATM activation. Their findings reveal that ufmylation of histone H4 by UFL1 is an important step for amplification of ATM activation and maintenance of genomic integrity. https://www.nature.com/articles/s41467-019-09175-0.pdf.

Yong Li lab at Cleveland Clinic discovered that tissue-specific miRNA expression alters cancer susceptibility conferred by a TP53 noncoding variant. Their findings elucidate an underlying mechanism of cancer susceptibility that is conferred by genetic variation and yet altered by microRNA expression. https://www.nature.com/articles/s41467-019-13002-x.pdf. Also, Li lab reported that glyphosate induces benign monoclonal gammopathy and promotes multiple myeloma progression in mice, implicating a mechanism underlying the B cell-specificity of glyphosate-induced carcinogenesis observed epi-demiologically. https://link.springer.com/article/10.1186/s13045-019-0767-9.

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RESEARCH HIGHLIGHTS

Shi-Yuan Cheng lab at Northwestern University Feinberg School of Medicine discovered that LY6K promotes glioblastoma tumorigenicity via CAV-1-mediated ERK1/2 signaling enhancement independent of tyrosine receptor kinase signaling. Their study highlights the importance of the contribution of LY6K to glioblastoma tumor biology and suggests LY6K as a potential membrane target for treating glioblastoma. https://pubmed.ncbi.nlm.nih.gov/32055849-ly6k-promotes-glioblastoma-tumorigenicity-via-cav-1-mediated-erk12-signaling-enhancement/. In addition, his lab reported that SRSF3-regulated RNA alternative splicing promotes glioblastoma tumorigenicity by affecting multiple cellular processes. This study indicates SRSF3 as a significant regulator of glioma-associated alternative splicing program, implicating SRSF3 as an oncogenic factor that contributes to the tumor biology of glioblastoma. https://cancerres.aacrjournals.org/content/79/20/5288.full-text.pdf. Moreover. Cheng lab found that microRNA -93 regulates tumorigenicity and therapy response of glioblastoma by modulating autophagy. These findings reveal a key role for MIR93 in the regulation of autophagy and suggest a combination treatment strategy involving the inhibition of autophagy while administering cytotoxic therapy. https://www.tandfonline.com/doi/pdf/10.1080/15548627.2019.1569947?needAccess=true.

Pan Zheng lab at University of Maryland Baltimore School of Medicine found hijacking antibody-induced CTLA-4 lysosomal degradation for safer and more effective cancer immunotherapy. Their work provides a new paradigm in the field on how to target CTLA-4 effectively for cancer immunotherapy. https://www.nature.com/articles/s41422-019-0184-1.pdf.

Li Ma lab at the University of Texas MD Anderson Cancer Center revealed that aberrant activation of β-Catenin signaling drives glioma tumorigenesis via USP1-mediated stabilization of EZH2. https://cancerres.aacrjournals.org/content/79/1/72.full-text.pdf

2019-2020
RESEARCH HIGHLIGHTS

Huiping Liu lab at Northwestern University found that homophilic CD44 interactions mediate tumor cell aggregation and polyclonal metastasis in patient-derived breast cancer models. These findings will lead to innovative biomarker applications to predict prognosis, facilitate development of new targeting strategies to block polyclonal metastasis, and improve clinical outcomes. https://cancerdiscovery.aacrjournals.org/content/9/1/96.full-text.pdf.

Xiangsheng Zuo lab at the University of Texas MD Anderson Cancer Center found that peroxisome proliferator-activated receptor delta (PPARD) overexpression in villin-expressing gastric progenitor (VPGCs) to result in inflammation, dysplasia, and tumor formation. These findings suggest that PPARD and VGPCs might be therapeutic targets for stomach cancer. https://www.clinicalkey.com/service/content/pdf/watermarked/1-s2.0-S0016508519335723.pdf?locale=en_US&searchIndex=.
Zuo lab also discovered that pleiotropic effects of PPARD accelerate colorectal tumorigenesis, progression, and invasion. The findings address long-standing, important, and unresolved questions related to the potential role of PPARD in APC mutation-dependent colorectal tumorigenesis by showing PPARD activation enhances APC mutation-dependent tumorigenesis. https://cancerres.aacrjournals.org/content/79/5/954.full-text.pdf.

Erxi Wu lab at Baylor Scott and White Health and Texas A&M University discovered that nucleolin as an essential functional binding protein of salinomycin, is likely responsible for salinomycin's anticancer and anti-cancer stem cells (CSCs) activities. The suppressive effects of salinomycin on neuroblastoma CSCs occur via suppression of CD34 expression and the disruption of the interaction between nucleolin and the CD34 promoter. https://pubs.acs.org/doi/pdf/10.1021/jacs.8b12872.

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Shuli Xia lab at Johns Hopkins School of Medicine found that extracellular matrix protein tenascin C increases phagocytosis mediated by CD47 loss of function in glioblastoma. The findings will facilitate the development of novel innate immune system–based therapies for brain tumors. https://cancerres.aacrjournals.org/content/79/10/2697.full-text.pdf.

Jian Jian Li lab at University of California, Davis reported that, whereas consumption of high-level palmitate (the major saturated fatty acid in the human diet) induced lipotoxic effects, consumption of low-level palmitate enhanced mitochondrial metabolism and compromised both high-level palmitateinduced lipotoxicity and CCl4-generated hepatotoxicity via a CDK1-SIRT3-CPT2 cascade. https://www.sciencedirect.com/science/article/abs/pii/S1534580719309438?via%3Dihub. In addition, **Li** lab discovered that dual blockage of STAT3 and ERK1/2 eliminates radioresistant glioblastoma multiforme cells. These findings demonstrate a previously unknown feature of STAT3-mediated ERK1/2 regulation and an effective combination of two targets in resensitizing glioblastoma to radiotherapy. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6463934/pdf/main.pdf.

Zhongsheng You lab at Washington University School of Medicine identified a Ca2+-CaMKK2-AMPK-Exo1 signaling pathway that safeguards chromosome stability by preventing abnormal processing of fork DNA. These findings reveal a link between [Ca2+]i and the replication stress response as well as a function of the Ca2+-CaMKK2-AMPK signaling axis in safeguarding fork structure to maintain genome stability. https://www.sciencedirect.com/science/article/abs/pii/S1097276519302734?via%3Dihub.

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RESEARCH HIGHLIGHTS

Lin Zhang lab at University of Pittsburgh School of Medicine demonstrated that RIP1/JNK-dependent p53 up-regulated modulator of apoptosis (PUMA) induction mediates acetaminophen induced liver injury by promoting hepatocyte mitochondrial dysfunction and necrosis, and suggest that PUMA inhibition is useful for alleviating acute hepatotoxicity attributed to acetaminophen overdose. https://aasldpubs.onlinelibrary.wiley.com/doi/pdf/10.1002/hep.30422. In addition, his lab discovered that BET inhibitors potentiate chemotherapy and killing of SPOP-mutant colon cancer cells via induction of DR5. Their findings may provide a new molecular marker for improving colon cancer therapies. https://cancerres.aacrjournals.org/content/79/6/1191.full-text.pdf.

Zhao-Hui Wu lab at University of Tennessee Health Science Center reported that upregulation of PD-L1 via HMGB1-activated IRF3 and NF-κB contributes to UV radiation-induced immune suppression. The findings identify PD-L1 as a critical component of UV-induced immune suppression in the skin, which facilitates immunoevasion of oncogenic melanocytes and development of melanoma. https://cancerres.aacrjournals.org/content/79/11/2909.full-text.pdf. In addition, Wu lab found that LINC02273 drives breast cancer metastasis by epigenetically increasing AGR2 transcription. Their findings uncover a key role of LINC02273-hnRNPL-AGR2 axis in breast cancer metastasis and provide potential novel therapeutic targets for metastatic breast cancer intervention. https://molecular-cancer.biomedcentral.com/articles/10.1186/s12943-019-1115-y.

Qi-En Wang lab at The Ohio State University discovered that inhibition of miR-328 is a novel strategy for efficient elimination of cancer stem cells to prevent tumor metastasis and recurrence in patients with epithelial ovarian cancer as inhibition of miR-328–3p impairs cancer stem cell function and prevents metastasis in ovarian cancer. https://cancerres.aacrjournals.org/content/79/9/2314.full-text.pdf,

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RESEARCH HIGHLIGHTS

Yi-Chieh Nancy Du lab at Cornell University discovered function and clinical relevance of the receptor for hyaluronic acid-mediated motility (RHAMM) isoforms in pancreatic tumor progression and concluded that $RHAMM^B$ but not $RHAMM^A$ can serve as a prognostic factor for pancreatic cancer. https://www.ncbi.nlm.nih.gov/pubmed/31072393. Her lab also found that orthotopic mouse models of liver metastasis by intrasplenically injecting the pancreatic tumor cells are useful in studying the molecular mechanisms of metastasis and evaluating therapeutic regimens. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6800204/.

Chengfeng Yang lab at University of Kentucky found that in vivo β-catenin attenuation by the integrin α5-targeting nano-delivery strategy suppresses triple negative breast cancer (TNBC) stemness and metastasis. These findings suggest that integrin α5-targeting nanoparticles may provide a facil and unique strategy of specially attenuating β-catenin in vivo for treating metastatic TNBC. https://www.ncbi.nlm.nih.gov/pubmed/31504995.

Qing Zhang Lab at University of North Carolina and UT Southwestern Medical Center identified PBRM1 acts as a lysine 382 acetylation through its bromodomain 4 to suppress renal tumor growth. https://www.nature.com/articles/s41467-019-13608-1. Moreover, Zhang lab also identified ADSL is an oncogenic driver in triple negative breast cancer via activating cMYC pathway. https://www.nature.com/articles/s41467-019-13168-4. Zhang lab also shared a protocol of how to use seahorse machine to measure OCR and ECAR in cancer cells. https://link.springer.com/protocol/10.1007%2F978-1-4939-9027-6_18

Yu-Ying He lab at University of Chicago discovered that m^6A mRNA demethylase FTO regulates melanoma tumorigenicity and response to anti-PD-1 blockade. The findings suggest that the combination of FTO inhibition with anti-PD-1 blockade may reduce the resistance to immunotherapy in melanoma. https://www.nature.com/articles/s41467-019-10669-0.pdf.

Congratulations!

Genes & Diseases is officially accepted as an SCI journal!

https://www.sciencedirect.com/journal/genes-and-diseases

Postdoctoral Research Associate, St. Jude Children's Research Hospital

Overview

The laboratory of Dr. Taosheng Chen studies the roles of nuclear xenobiotic receptors (PXR, CAR) and drug–metabolizing enzymes (CYP3A) in tumorigenesis and cancer drug resistance. We use multidisciplinary approaches to investigate signaling pathways, identify and validate targets, and develop chemical probes to interrogate the function of PXR, CAR and CYP3A in order to overcome drug resistance and tumorigenesis in cellular and animal models (*Lin et al, **Nat Commun**. 8:741, 2017*).

Responsibilities

The postdoctoral fellow will work on one or more of the following projects:

- Transcriptional and splicing regulation of PXR and CYP3A in human cancers;
- Mechanism of action of chemical probes, including bi-functional compounds such as PROTACs;
- Co-crystal structural analysis and computational modeling of small molecule – protein interactions;
- Signal cross-talk between drug metabolism and energy metabolism pathways;
- Target discovery by data mining approaches followed by experimental validation.

The Chen Lab provides a unique training environment: in addition to basic research on transcriptional/ post-transcriptional regulation, the postdoctoral fellow will learn and gain experience in small molecule drug discovery from target identification, compound screening to preclinical studies. Former Chen Lab postdocs have landed jobs as Assistant Professors in Universities or Senior Scientist in Pharmaceutical Companies. The successful candidate will work in a collaborative and multidisciplinary environment by collaborating with biologists, chemists and structural biologists (https://www.stjude.org/chen).

Minimum Education

Highly motivated individuals with a strong publication record are encouraged to apply. A strong interest in studying transcriptional/post-transcriptional regulation, together with experience either in cell and molecular biology, protein biochemistry and structural biology, molecular pharmacology, large-scale data mining or RNA biology, are desirable. Candidates must have (or soon receive) a PhD degree.

Contact Information

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THE UNIVERSITY OF CHICAGO

Postdoctoral Scholar in epitranscriptomics and cancer, The University of Chicago

Multiple postdoctoral positions are available in the laboratory of Yu-Ying He, PhD, at the University of Chicago. We seek to recruit several highly motivated postdoctoral fellows to investigate the regulatory and functional mechanisms of epitranscriptomics and RNA modifications in tumor development, progression, and therapeutic response (See our recent publication: https://www.nature.com/articles/s41467-019-10669-0). We are particularly interested in, but not limited to, the role of RNA modifications in genomic stability and inflammation in tumorigenesis and therapeutic response in cells and animal models.

Applicants must have a recent Ph.D. or equivalent degree in the biological sciences. Demonstrated expertise in molecular, cellular, and biochemical techniques, is preferred. Experience with mammalian cell culture, microscopy, immunoassays, molecular biology, animal models, and/or systems analysis of molecular interactions is desirable. Highly motivated individuals with excellent communication skills and the ability to work effectively within a research team are encouraged to apply.

Applicants should submit curriculum vitae, a cover letter stating research interests and career goals, and names and contact information (addresses, telephone numbers, email addresses) of three references to Yu-Ying He at yyhe@medicine.bsd.uchicago.edu.

THE OHIO STATE UNIVERSITY
WEXNER MEDICAL CENTER

Postdoctoral Researcher

Dr. Jenny Wang (https://medicine.osu.edu/cancer-biology-genetics/directory/faculty/wang-jenny-phd/pages/index.aspx), a professor at Department of Cancer Biology and Genetics, The Ohio State University Wexner Medical Center invites applications for full-time researchers at the level of Postdoctoral Researcher. We are looking for scientists with a doctoral degree, outstanding academic credentials and a record of scholarly productivity in the areas of tumor biology, immunology and genetics. Successful candidates will conduct research to determine molecular mechanisms of colon cancer immune evasion, metastasis, tumor dormancy and drug resistance, identify and validate novel targets using 2D- and 3D- cell cultures, mouse models and PDXs, and ultimately develop effective therapies to treat colon cancer patients. The Ohio State University offers a competitive salary and outstanding benefits.

The Ohio State University is one of the largest public universities with 200+ academic majors, significant physical and interdisciplinary interactions between the Colleges of Arts and Sciences and Medicine. This is complemented by an outstanding Comprehensive Cancer Center, which provides the infrastructure and resources for strong interdisciplinary interactions with a focus on translation, in a collegial and supportive research environment. The university is located in Columbus, a vibrant and rapidly developing city of almost one million, which is recognized by Money Magazine as one of the “6 best big cities” in the US.

The Ohio State University is an Equal Opportunity/Affirmative Action Employer. All qualified applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, disability status or protected veteran status.

Applications should include Curriculum Vitae, Research Summary, and contact information for three academic references and be sent to jing.wang@osumc.edu. Can also apply online at http://wexnermedical.osu.edu/careers. Enter keyword or job ID# 457401 to review the job posting for Post-Doctoral Researcher.

How to become a member of ACACR

如何成为ACACR 协会会员

感谢大家对ACACR 的关心和鼓励，更感谢许多志愿者们的付出。我们的财务李勇已把协会的银行帐户，PayPal 帐户开好；我们 IT 小组的戴木水已经将网上自动付款体系建成；我们会员小组的席亚光已将会员注册的表格等设计好。下面是如何成为我们协会成员了。

我们有两种会员制，普通会员 (regular member) 和 临时会员 (associate member)。普通会员又分终生会员 (lifetime membership) 以及年度会员， 前者会费 $500, 后者会费每两年$100。临时会员暂不收费，但以后可能会有所改变。

目前我们还是半自动化注册 (即有部分是手工)。请到我们网站 acacr.org 在“membership”栏下载注册表, 填好后电邮给表最后的邮件地址。

我们共有三种付会员费的方式：

1. 在我们网站上用Paypal或信用产卡付。tacacr@outlook.com

2. 支票. 请写明付给 "Association of Chinese Americans in Cancer Research, Inc." 需要邮寄支票的, 请与Shuli 联系, xia@kennedykrieger.org. 请在电邮上注明 ACACR member.

我们将在收到付款后五-七个工作日发出收据。

协会会员的益处:

协会普通会员和临时会员都可以参加WeChat 的讨论，信息交流，年会以及其他一些由ACACR 组织的活动。普通会员还有以下一些额外的福利。

（1）协会内部选举和被选举权;

（2）由ACACR 推荐去AACR 各种委员会和杂志编辑部;

（3）在我们协会网站上招人广告栏上发广告(微信群里的帖子会很快被淹没);

（4）在我们协会网站上贴一些会议通知;

（5）在我们协会每月一次的 Newsletter 上登广告 (非会员收费 $20);

（6）我们协会网站和 Newsletter “Research Highlights" 栏目中将优先选发协会会员刚发表的文章;

（7）今后ACACR 有小型奖励机会 (award opportunity), 将优先考虑我们的普通会员;

（8）今后购买ACACR 赞助商的物品时可能有折扣机会。

普通会员今后可能有的福利还包括会员学术交流活动 (annual retreat), 成员互助等。

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