# ACACR NewsLetter

Association of Chinese Americans in Cancer Research

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## In This Issue

# New Year Greetings from the President of ACACR

Dear Colleagues and Friends,

Time flies fast. The year of 2017 has been a great and memorable one to all of us. At the 108th annual meeting of American Association for Cancer Research (AACR) in Washington DC, we successfully inaugurated our organization, Association of Chinese American on Cancer Research (ACACR) with approximately 200 attendees on April 3, 2017. Our first step to promote ACACR was to communicate with AACR in order to participate in the organization and scientific activities of AACR. In July, we nominated and recommended a total of 18 colleagues as candidates for various AACR committees and the of boards of AACR journals. In July, we were also asked to nominate a US co-chair for the AACR-CSCO joint session on Microenvironment and Cancer Immunotherapy at the CSCO's annual meeting in September 27, 2017 in Xiamen, China. Recently three of our colleagues were invited to serve as members of three different AACR subcommittees. These events signify our first success! However, we have much more to work on and a long journey to reach our goals in the coming new year. First, we need to recruit more colleagues as the members of ACACR, as the number of our registered members are still limited. Next, we will plan our second meeting that will be held at the 109th AACR annual meeting in Chicago. In addition, we should continue to communicate with AACR about the status of our nominations and seek more effective and efficient approaches to directly contact key officials and committees and journal editors. Furthermore, we will need more volunteers to be actively involved in organization of the ACACR activities. Lastly, we will continue our hard working toward our ultimate goals: to promote intellectual interactions and scientific collaborations and to bridge basic cancer research with translational research and expedite pharmaceutical drug discovery/development for innovative and personalized medicine/oncology for cancer prevention, prognosis and treatments. Let us work together, as a fist is more powerful than a finger. Looking forward to another fruitful, thrilling, and challenging year.

Happy New Year to all of you!

# RESEARCH HIGHLIGHTS

- **Wenwei Hu** and **Zhaohui Feng** labs at Rutgers Cancer Institute of New Jersey recently discovered that small GPTase Rac1 specifically interacts with mutant p53 that contributes to gain-of-function of p53 mutant protein, thereby promoting tumorigenesis. http://genesdev.cshlp.org/content/31/16/1641. Moreover, **Zhaohui Feng** and **Wenwei Hu** labs also showed that parkin targets HIF-1α for ubiquitination and degradation to inhibit breast tumor progression. https://www.nature.com/articles/s41467-017-01947-w

- **Zhenkun Lou** lab at Mayo Clinic in collaboration with **Liewei Wang** lab at Mayo Clinic showed that protein deacetylase SIRT7 regulates Akt activity through an adaptor protein FKBP51. http://www.cell.com/cell-reports/fulltext/S2211-1247(17)30027-X. Moreover, collaborating with **Jian Yuan** lab at Mayo Clinic , they further showed that the ubiquitin protease USP13 regulates the recruitment of BRCA1 through an adaptor protein Rap80. https://www.nature.com/articles/ncomms15752

- **Qing-Bai She** lab at University of Kentucky showed that snail determines the therapeutic response to mTOR kinase inhibitors by transcriptional repression of 4E-BP1. https://www.nature.com/articles/s41467-017-02243-3

- **Xiongbing Lu** lab at The University of Texas MD Anderson Cancer Center showed that amplification of USP13 drives ovarian cancer metabolism. https://www.nature.com/articles/ncomms13525

# RESEARCH HIGHLIGHTS

- **Rutao Cui** lab at Boston University demonstrated a potential melanocortin-1 receptor (mc1r)-targeted intervention strategy in mice to rescue loss-of-function mc1r in mc1r rhc variants for therapeutic benefit by activating mc1r protein palmitoylation. form this literature, individuals carrying mc1r variants, especially those associated with red hair color, fair skin and poor tanning ability (denoted as rhc variants), are associated with higher risk of melanoma. https://www.nature.com/articles/nature23887

- **Jean Zhao** lab at Dana-Farber Cancer Institute used mouse models of breast carcinoma and other solid tumors to show that selective cyclin-dependent kinases 4 and 6 (CDK4/6) inhibitors not only induce tumor cell cycle arrest, but also promote anti-tumor immunity. Their findings indicate that CDK4/6 inhibitors increase tumor immunogenicity and provide a rationale for new combination regimens comprising CDK4/6 inhibitors and immunotherapies as anti-cancer treatment. https://www.nature.com/articles/nature23465

- **Qiufu Ma** lab at Dana-Farber Cancer Institute identified that spinal circuits are involved in touch-evoked dynamic mechanical pain. https://www.nature.com/articles/nn.4549

- **Zigang Dong** lab at The Hormel Institute, University of Minnesota demonstrated that a deficiency of ribosomal S6 kinase 2 (RSK2) can result in dramatically decreased IFNγ secretion through an inappropriate phosphorylation status of T-bet, a modulator of IFNγ expression. Their results indicate that phosphorylation of T-bet is required for the inhibition of colon cancer metastasis and growth through a positive regulation of RSK2/T-bet/IFNγ signaling. http://www.pnas.org/content/114/48/12791.full.pdf

# RESEARCH HIGHLIGHTS

- **Jianjun Chen** and **Chuan He** labs at University of Chicago reported that FTO, highly expressed in subtypes of Acute Myeloid Leukemia (AML), plays an oncogenic role in AML as a N6-Methyladenosine RNA demethylase. http://www.cell.com/cancer-cell/fulltext/S1535-6108(16)30560-8
  Dr. **Chen** now is Professor and Vice Chair, Department of Systems Biology at City of Hope, Duarte, CA.

- **Shi-Yuan Cheng** lab at Northwestern University recently reported that radiation induces MST4 expression and that MST4 phosphorylates ATG4B at serine 383, which increases ATG4B activity and autophagic flux. He and his team found that inhibition of ATG4B reduces autophagy and tumorigenicity of glioblastoma (GBM) cells and improves the impact of radiotherapy on GBM growth in mice. http://www.cell.com/cancer-cell/pdf/S1535-6108(17)30508-1.pdf. Moreover, he collaborated with a China group and found together that TRIM24 is an oncogenic transcriptional co-activator of STAT3 in GBM.
  https://www.nature.com/articles/s41467-017-01731-w

- **Zhimin Lu** lab at The University of Texas MD Anderson Cancer Center and **Yizhi Jane Tao** lab at Rice University recently reported KAT2A coupled with the α-KGDH complex acts as a histone H3 succinyltransferase and preventing the α-KGDH complex from entering the nucleus, or expression of KAT2A(Tyr645Ala), reduces gene expression and inhibits tumor cell proliferation and tumor growth.
  https://www.nature.com/articles/nature25003. Moreover, **Zhimin Lu** lab reveals that stabilization of phosphofructokinase 1 platelet isoform by AKT promotes tumorigenesis in human glioblastoma development.
  https://www.nature.com/articles/s41467-017-00906-9.pdf

# RESEARCH HIGHLIGHTS

- **Ping Chi and Yu Chen** labs at Memorial Sloan Kettering Cancer Center identified an aberrantly expressed gastrointestinal-lineage transcriptome governed by HNF4G and HNF1A in 30% of castration-resistant prostate cancer. Dr. Chen and his co-workers found that HNF4G is a pioneer factor for this transcriptional program and its ectopic expression at physiologic levels reduces sensitivity to hormone deprivation. http://www.cell.com/cancer-cell/fulltext/S1535-6108(17)30461-0

- **Hao Zhu** lab at University of Texas Southwestern Medical Center uncover context-specific roles for the SWI/SNF component Arid1a in liver cancer, where elevated Arid1a promotes tumor initiation through CYP450-mediated oxidative stress, whereas reduced Arid1a in established tumors increases metastasis due to reduced expression of inhibitory factors. http://www.cell.com/cancer-cell/pdf/S1535-6108(17)30460-9.pdf

- **Xin Wei Wang** lab at National Cancer Institute showed that a large numbers of RNA binding proteins (RBPs) are dysregulated in hepatocellular carcinoma (HCC) and that NELFE, an RBP, enhances MYC-induced HCC development by regulating the binding of MYC to target promoters and the mRNA stability of several MYC-regulated genes. http://www.cell.com/cancer-cell/fulltext/S1535-6108(17)30252-0

- **Hua Lu** lab at Tulane University School of Medicine reported that mutant p53 gains its function via c-Myc activation upon CDK4 phosphorylation at serine 249 and consequent PIN1 binding. http://www.cell.com/molecular-cell/pdf/S1097-2765(17)30841-9.pdf

# RESEARCH HIGHLIGHTS

- **Wendong Huang** lab at Beckman Research Institute showed that show that CAMKIIg stabilizes c-Myc by phosphorylating it at Ser62, that the CAMKIIg level positively correlates with the c-Myc level in patient TCL, and that inhibition of CAMKIIg reduces TCL burden in mice. http://www.cell.com/cancer-cell/pdf/S1535-6108(17)30251-9.pdf

- **Jianjun Zhang** lab at The University of Texas MD Anderson Cancer Center performed sequencing of the T-cell receptor (TCR) in 45 tumor regions from 11 localized lung adenocarcinomas and observed substantial intratumor differences in T-cell density and clonality with the majority of T-cell clones restricted to individual tumor regions. They found that TCR ITH positively correlated with predicted neoantigen ITH and a higher degree of TCR ITH was associated with an increased risk of postsurgical relapse and shorter disease-free survival. http://cancerdiscovery.aacrjournals.org/content/early/2017/07/21/2159-8290.CD-17-0256

- **Jing Chen** lab at Emory University demonstrated that a high-fat diet increases circulating acetoacetate that enhances tumor growth potential of BRAF V600E melanoma cells in mice, which informs the design of a "precision diet" to lower cancer risk and development of metabolism-targeted therapies for cancer treatment. http://www.cell.com/cell-metabolism/pdf/S1550-4131(16)30643-X.pdf

- **Jianrong Lu** lab at University of Florida College of Medicine demonstrated that epithelial-to-mesenchymal transition confers pericyte properties on cancer cells. https://www.jci.org/articles/view/86623

# RESEARCH HIGHLIGHTS

- **Wenyi Wei** lab at Harvard Medical School reported that PD-L1 protein abundance is regulated by cyclin D-CDK4 and the Cullin 3SPOP E3 ligase via proteasome-mediated degradation. They found that cyclin D-CDK4 kinase destabilizes PD-L1 via Cul3SPOP to control cancer immune surveillance. https://www.nature.com/articles/nature25015

- **Catherine J. Wu** lab at Dana-Farber Cancer Institute demonstrated the feasibility, safety, and immunogenicity of a vaccine that targets up to 20 predicted personal tumor neoantigens. She and her co-workers have provided a strong rationale for further development of this approach, alone and in combination with checkpoint blockade or other immunotherapies. https://www.nature.com/articles/nature22991. Moreover, she and her team showed the evolutionary landscape of chronic lymphocytic leukemia treated with ibrutinib targeted therapy. https://www.nature.com/articles/s41467-017-02329-y

- **Hui-Kuan Lin** lab at Wake Forest School of Medicine showed that atad3a suppresses Pink1-dependent mitophagy to maintain homeostasis of hematopoietic progenitor cells. https://www.nature.com/articles/s41590-017-0002-1

- **Jianwen Que** at Columbia University used multiple models and lineage tracing strategies to show that the squamous–columnar junction basal cell population serves as a source of progenitors for the transitional epithelium. https://www.nature.com/articles/nature24269

# RESEARCH HIGHLIGHTS

- **Yiwen Chen** lab at The University of Texas MD Anderson Cancer Center developed a comprehensive toolkit named Ribo-TISH, which allows for detecting and quantitatively comparing TIs across conditions from TI-seq data. https://www.nature.com/articles/s41467-017-01981-8

- **Suming Kang** lab at Emory University demonstrated a mechanism by which GDH1 provides anti-anoikis and pro-metastatic signals through activating CamKK2 and AMPK that promotes tumor metastasis in LKB1-deficient lung cancer. http://www.cell.com/molecular-cell/pdf/S1097-2765(17)30884-5.pdf

- **Yibin Kang** at Princeton University established that human monoclonal antibody against Jagged 1(Clone 15D11) as a potential therapeutic agent for the prevention or treatment of bone metastasis for breast cancer. http://www.cell.com/cancer-cell/pdf/S1535-6108(17)30472-5.pdf

- **Zhongsheng You** lab at Washington University School of Medicine shed new light on DNA damage responses. First, his group found that nonsense-mediated RNA decay (NMD) pathway is repressed in response to persistent DNA damage in nonproliferating cells in a p38 MAPK-dependent manner; http://www.jbc.org/content/292/37/15266.long. Second, they also revealed that Dna2 initiates resection at clean DNA double-strand breaks; https://academic.oup.com/nar/article/45/20/11766/4157875


# MEMBERS' ACHIEVEMENTS

**Congratulations to Dr. Haian Fu who is recently appointed as Chair of the Department of Pharmacology at Emory University School of Medicine, Atlanta, GA.**

**Congratulations to Dr. Zhenkun Lou being invited to serve the AACR NextGen Grants for Transformative Cancer Research Scientific Review Committee. Dr. Lou is Professor at the Department of Oncology, Mayo Clinic, Rochester, MN. He is also the current President-Elect of ACACR.**

**Congratulations to Dr. Lin Zhang being invited to serve the AACR annual meeting committee. Dr. Zhang is Professor at the Department of Pharmacology and Chemical Biology, University of Pittsburgh School of Medicine, Pittsburgh, PA. He also serves as the Chair of Public Relationship Committee of ACACR.**

**Also Congratulations to Dr. Xiaoting Zhang, who has been invited to serve as a member of the AACR Breast Cancer Research Grants Scientific Review Committee. Dr. Zhang is Associate Professor at Department of Cancer Biology, University of Cincinnati, Cincinnati, OH.**

We would like to thank Dr. Lin Zhang and all other members of the ACACR peer-review group for their hard work to put a 346 page recommendation material together for AACR to consider. We hope to see more members from our association to contribute their time and energy to serve the cancer research community.

# 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. 银行直接转帐. Routing No: 044000037, Account No: 121901257.

3. 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。

希望更多肿瘤研究者加入我们协会，为建立“华人肿瘤研究学者之家”贡献力量。

# POSTDOCTORAL FELLOW

A postdoctoral position is immediately available in the laboratory of Dr. Zheng Fu in the Department of Human and Molecular Genetics and the VCU Institute of Molecular Medicine at Virginia Commonwealth University, School of Medicine. The main research focus in Dr. Fu's laboratory is to elucidate the molecular mechanisms of cell cycle control and genomic stability maintenance. The aim is to understand how deregulation of these physiological processes contributes to carcinogenesis and ultimately apply the findings to the development of novel, improved, and targeted cancer therapies (eLife. 5:e10734, 2016; Nat Cell Biol. 10:1076, 2008; J Clin Invest. 119:2714, 2009; Cell Reports. 21(8): 2147, 2017).

Dr. Fu's laboratory is seeking highly qualified, dedicated individuals with a commitment to research excellence. The successful candidate should have a PhD or MD degree with a strong background in molecular biology, cell biology, and/or biochemistry. Research experience in animal models would be an advantage. The candidates must also have strong organizational, written, and verbal communication skills in English.

Candidates interested in the position should send a cover letter with a brief summary of research experience and interests, an updated CV, and contact information for three references to:

Zheng Fu, PhD

Associate Professor

Department of Human & Molecular Genetics

Virginia Commonwealth University

401 College Street

Richmond, VA 23298

Email: zheng.fu@vcuhealth.org

# ACACR leadership

**President**

**Shi-Yuan Cheng**

**President-Elect**

**Zhenkun Lou**

**Board of Directors**

**Hua Luo, Wei Gu, Jinsong Liu, Shaomeng Wang**

**General Secretary**

**Shuli Xia**

**Treasurer**

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**Committee Chair/Co-Chair**

**Advisory**: Yibin Kang
**Annual meeting**: Tongchuan He; Gloria Su; Binhua Zhou
**Audit**: Zhaohui Feng
**Fundraising**: Xin Fan
**IT-support:** Wenwei Hu; Mushui Dai
**Membership**: Yaguang Xi
**Public relationship**: Lin Zhang

Association of Chinese Americans in Cancer Research

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