ACACR NewsLetter

Association of Chinese Americans in Cancer Research

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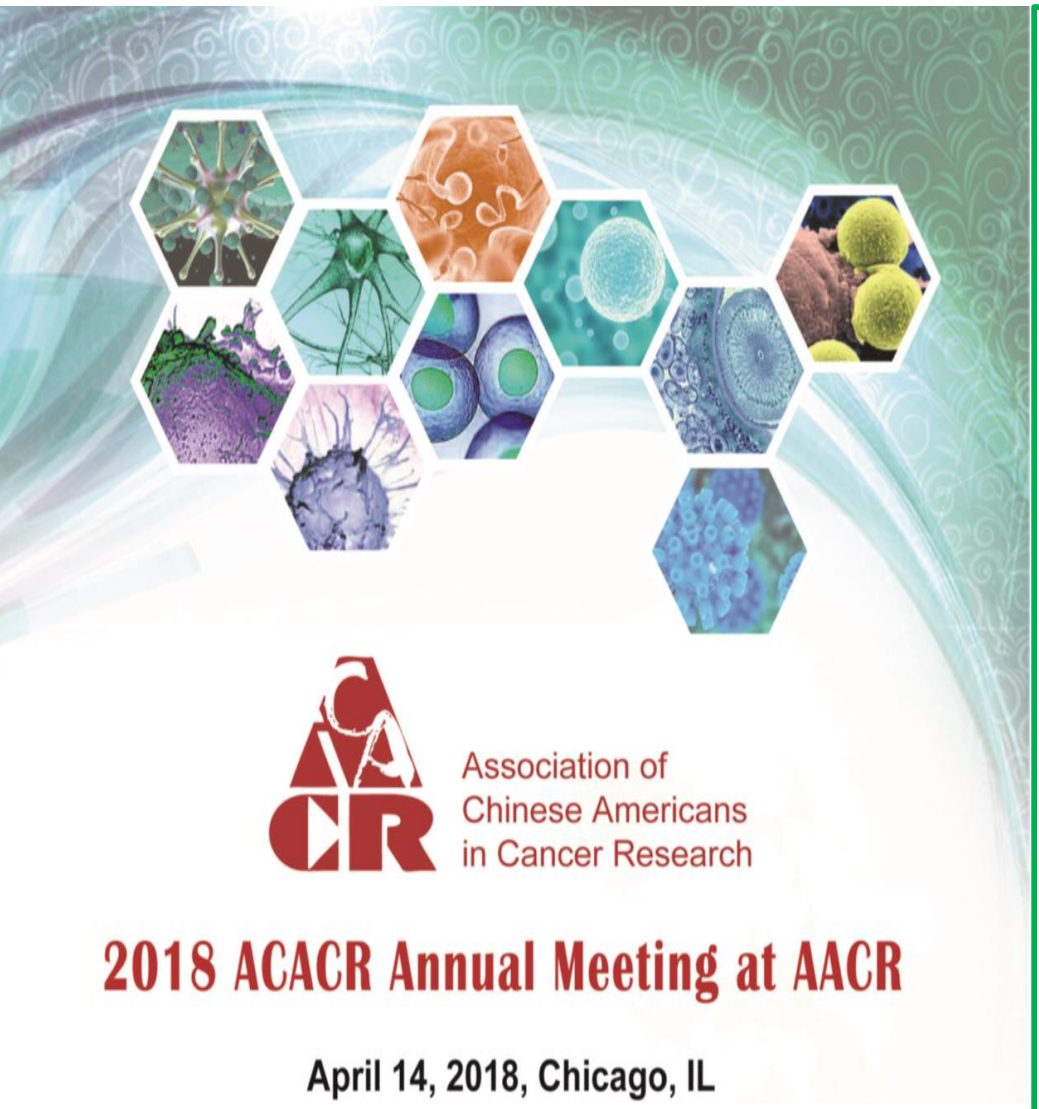
April 2018



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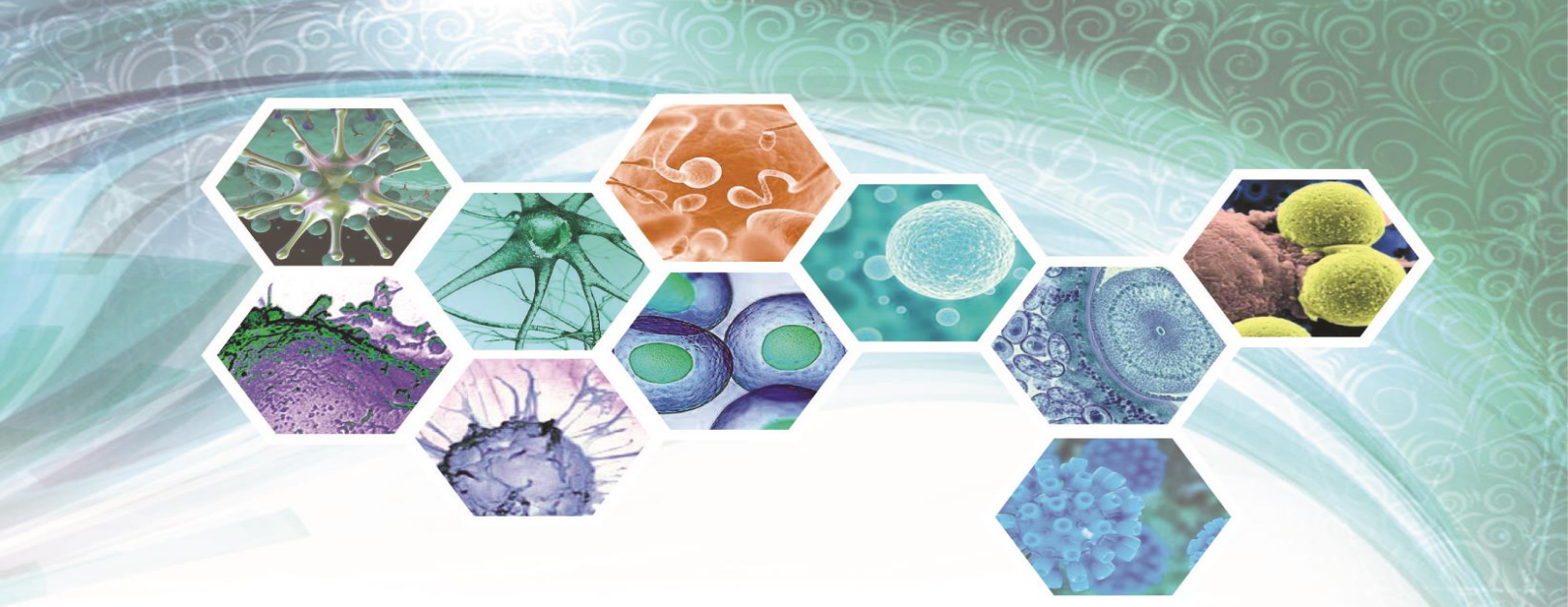
In This Issue



Association of Chinese Americans in Cancer Research

2018 ACACR Annual Meeting at AACR

April 14, 2018, Chicago, IL

Association of Chinese Americans in Cancer Research (ACACR)

Annual Meeting 2018

www.acacr.org

When: 2:00 p.m.– 4:30 p.m., April 14, 2018
Where: Daniel Hale Williams Auditorium
240 E. Huron Street, Chicago, IL 60611

Followed by Social/Dinner Activities

When: 5:00 p.m. – 7:00 p.m., April 14, 2018
Where: West Side-Ryan Family Atrium
303 E. Superior Street, Chicago, IL 60611

2018 ACACR Annual Meeting Agenda

When: April 14, 2018
Where: Daniel Hale Williams Auditorium
240 E. Huron Street, Chicago, IL 60611

2:00 p.m. **Welcome and introduction**. T.-C. He/Peter Zhou

2:05 p.m. **Overview/update of ACACR activities.** Shiyuan Cheng

2:15 p.m. **"RNA Methylation in Cancer".** Chuan He, The University of Chicago

2:45 p.m. **"CRISPR Screens and AI on Precision Cancer Medicine".** Shirley Liu, Dana-Farber Cancer Institute and Harvard T.H. Chan School of Public Health

3:15 p.m. **"Master of Small Molecules".** Zhinong Gao, MedChemExpress

3:25 p.m. **"Dual translational biomarker strategies to stratify patients for personalized cancer immunotherapy".** Jianda Yuan, Merck

3:50 p.m. **ACACR Annual Financial Report.** Yong Li

4:00 p.m. **ACACR Publications/Newsletters.** Zhenghe Wang

4:10 p.m. **Closing Remarks/Future Plans.** Zhenkun Lou

4:30 p.m. **Networking/Social**

5:00 p.m. **Dinner/Social.** West side-Ryan Atrium, Lurie Biomedical Research Building 303 E. Superior Street, Chicago, IL 6061)

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RESEARCH HIGHLIGHTS

Jian Yu and **Lin Zhang Labs** at University of Pittsburgh showed that targeting the BH3-only Bcl-2 family protein PUMA suppresses chemotherapy-induced gastrointestinal injury by protecting intestinal stem cells. http://stm.sciencemag.org/content/10/427/eaam7610.full.

Moreover, they found that PUMA has a novel function of amplifying necroptosis signaling, in addition to its function in apoptosis. http://www.pnas.org/content/early/2018/03/21/1717190115.

Pan Zheng and **Yang Liu Labs** at Children's National Health System found that the antitumor effect of anti-CTLA-4 is dependent on the local depletion of Tregs via interactions with the Fc receptor on other host cells and the subsequent antibody-dependent cellular cytotoxicity. https://www.nature.com/articles/s41422-018-0011-0. Moreover, they developed human CTLA-4 knock-in homo- and heterozygous mouse models, which could be useful for identifying safer anti-CTLA-4 therapies. https://www.nature.com/articles/s41422-018-0012-z. In addition, **Yang Liu Lab** showed that CD24–p53 axis suppresses diethylnitrosamine-induced hepatocellular carcinogenesis by sustaining intrahepatic macrophages. https://www.nature.com/articles/s41421-017-0007-9.

RESEARCH HIGHLIGHTS

Hua Lu Lab at Tulane University discovered SPIN1 as a nucleolar negative regulator of the ribosomal stress-MDM2-p53 pathway. The biological significance of this study lies in that SPIN1 is highly expressed in human cancers that are associated with the down regulation of the p53 signature. https://elifesciences.org/articles/31275.

Zhenkun Lou Lab at Mayo Clinic discovered that L3MBTL2 is mutated in T cell prolymphocytic leukemia and that L3MBTl2 is a missing link that coordinates key ubiquitin signaling events to induce DNA repair and checkpoint activation. https://www.nature.com/articles/s41556-018-0071-x. Moreover, **Zhenkun Lou** and **Jian Yuan Labs** at Mayo Clinic found that the deubiquitinase USP9X promotes tumor cell survival and confers chemoresistance through YAP1 stabilization. https://www.nature.com/articles/s41388-018-0134-2.

Shuli Xia Lab at Johns Hopkins School of Medicine discovered that Krüppel-like factor 4 (KLF4) upregulates expression of UDP-α-D-glucose 6-dehydrogenase (UGDH) and that targeting UGDH inhibits glioblastoma growth and migration. https://www.nature.com/articles/s41388-018-0138-y. Moreover, the lab found that KLF4 induces mitochondrial fusion. http://www.jbc.org/cgi/doi/10.1074/jbc.RA117.001323.

RESEARCH HIGHLIGHTS

Lizi Wu Lab at University of Florida discovered that CRTC1-MAML2 fusion-induced lncRNA LINC00473 expression maintains the growth and survival of human mucoepidermoid carcinoma cells and that LINC00473 acts as a promising biomarker and therapeutic target for human CRTC1-MAML2-positive mucoepidermoid carcinomas. https://www.nature.com/articles/s41388-017-0104-0.

Qing Lu Lab at Cincinnati Children's Hospital Medical Center found that a histone deacetylase 3-dependent pathway delimits peripheral myelin growth and functional regeneration. They identified the HDAC3-TEAD4 network as a dual-function switch of cell-intrinsic inhibitory machinery that counters myelinogenic signals and maintains peripheral myelin homeostasis, highlighting the therapeutic potential of transient HDAC3 inhibition for improving peripheral myelin repair. https://www.nature.com/articles/nm.4483.

Guo-Cheng Yuan Lab at Dana-Farber Cancer Institute discovered that hub enhancers are the major constituents responsible for super-enhancer functional and structural organization. https://www.nature.com/articles/s41467-018-03279-9.

RESEARCH HIGHLIGHTS

Shirley Liu Lab with another lab at Dana-Farber Cancer Institute found that in many human cancers, expression of PBRM1 and ARID2 inversely correlated with expression of T cell cytotoxicity genes, and Pbrm1-deficient murine melanomas were more strongly infiltrated by cytotoxic T cells. http://science.sciencemag.org/content/early/2018/01/03/science.aao1710.

Hexin Chen Lab at University of South Carolina found that HER2 overexpression triggers an IL-1α pro-inflammatory circuit to drive tumorigenesis and promote chemotherapy resistance. https://www.ncbi.nlm.nih.gov/pubmed/29382706.

Zigang Dong Lab at University of Minnesota discovered that RSK2 is required for TRAF6 phosphorylation-mediated colon inflammation (https://www.nature.com/articles/s41388-018-0167-6) and found that veratramine modulates AP-1-dependent gene transcription by directly binding to programmable DNA (https://www.ncbi.nlm.nih.gov/pubmed/29237043).

Wenyi Wei Lab at Harvard Medical School found that phosphorylation of EZH2 by AMPK suppresses PRC2 methyltransferase activity and oncogenic function. https://www.ncbi.nlm.nih.gov/pubmed/29351847.

RESEARCH HIGHLIGHTS

Wenwei Hu Lab at The State University of New Jersey found that a polymorphism with either arginine (R72) or proline (P72) at codon 72 in the tumor suppressor p53 affects aging and longevity in mouse models. https://elifesciences.org/articles/34701.

Li Ding Lab at Washington University in St. Louis conducted a comprehensive analysis of oncogenic driver genes and mutations in >9,000 tumors across 33 cancer types and highlighted the prevalence of clinically actionable cancer driver events in TCGA tumor samples. http://www.cell.com/cell/pdf/S0092-8674(18)30237-X.pdf. Moreover, **Li Ding** and **Feng Chen Labs** at Washington University in St. Louis conducted a pan-cancer analysis to identify hundreds of predisposing germline variants (http://www.cell.com/cell/pdf/S0092-8674(18)30363-5.pdf) and systematic analysis of splice-site-creating mutations in cancer (https://www.ncbi.nlm.nih.gov/pubmed/29617666). In addition, **Li Ding Lab** described driver fusions and their implications in the development and treatment of human cancers. https://www.ncbi.nlm.nih.gov/pubmed/29617662.

RESEARCH HIGHLIGHTS

Jean Zhao Lab at Dana Farber Cancer Institute highlighted recent data from orthotopic brain metastasis models that implicate brain-specific drug resistance mechanisms in breast cancer brain metastases and suggest a translational research paradigm to guide drug development for treatment of breast cancer brain metastases. https://www.ncbi.nlm.nih.gov/pubmed/29437794.

Han Liang Lab at The University of Texas MD Anderson Cancer Center conducted a pan-cancer analysis of enhancer expression in nearly 9000 patient samples and found that global enhancer activation positively correlates with aneuploidy but not mutations and enhancers as key regulators of therapeutic targets, including PD-L1. http://www.cell.com/cell/pdf/S0092-8674(18)30307-6.pdf. Furthermore, **Han Liang Lab** with other labs conducted systematic functional annotation of somatic mutations in cancer. http://www.cell.com/cancer-cell/pdf/S1535-6108(18)30021-7.pdf. Moreover, **Han Liang Lab** with another lab described that molecular characterization and clinical relevance of metabolic expression subtypes in human cancers. https://www.ncbi.nlm.nih.gov/pubmed/29617665.

RESEARCH HIGHLIGHTS

Hai Hu Lab at Chan Soon-Shiong Institute of Molecular Medicine conducted an analysis of clinicopathologic annotations for over 11,000 cancer patients in the TCGA program that leads to the generation of TCGA Clinical Data Resource, which provides recommendations of clinical outcome endpoint usage for 33 cancer types. http://www.cell.com/cell/pdf/S0092-8674(18)30229-0.pdf.

Hui-Kuan Lin Lab at Wake Forest School of Medicine found that Atad3a suppresses Pink1-dependent mitophagy to maintain homeostasis of hematopoietic progenitor cells. https://www.nature.com/articles/s41590-017-0002-1.

Xiaoqi Liu Lab at Purdue University found that Plk1-mediated phosphorylation of TSC1 enhances the efficacy of rapamycin. https://www.ncbi.nlm.nih.gov/pubmed/29559472.

Peixuan Guo Lab at The Ohio State University found that nanoparticle orientation controls RNA loading and ligand display on extracellular vesicles for cancer regression. https://www.nature.com/articles/s41565-017-0012-z.

RESEARCH HIGHLIGHTS

Hui-Wen Lo Lab at Wake Forest University School of Medicine found truncated glioma-associated oncogene homolog 1 (tGLI1) mediates mesenchymal glioblastoma via transcriptional activation of CD44 (http://cancerres.aacrjournals.org/content/early/2018/02/20/0008-5472.CAN-17-2933) and interaction between STAT3 and GLI1/tGLI1 oncogenic transcription factors promotes the aggressiveness of triple-negative breast cancers and HER2-enriched breast cancer (https://www.ncbi.nlm.nih.gov/pubmed/29449694).

Lihua Yu Lab at H3 Biomedicine report that 119 splicing factor genes carry putative driver mutations over 33 tumor types in TCGA. The most common mutations appear to be mutually exclusive and are associated with lineage-independent altered splicing. Samples with these mutations show deregulation of cell-autonomous pathways and immune infiltration. http://www.cell.com/cell-reports/pdf/S2211-1247(18)30152-9.pdf.

RESEARCH HIGHLIGHTS

Da Yang Lab at University of Pittsburgh characterized the epigenetic landscape of lncRNAs genes across a large number of human tumors and cancer cell lines and observe recurrent hypomethylation of lncRNA genes, including EPIC1. EPIC1 RNA promotes cell-cycle progression by interacting with MYC and enhancing its binding to target genes. http://www.cell.com/cancer-cell/pdf/S1535-6108(18)30110-7.pdf.

Xiaoming He Lab at The Ohio State University discovered that targeted production of reactive oxygen species in mitochondria can overcome cancer drug resistance. https://www.nature.com/articles/s41467-018-02915-8.

Tim H.-M. Huang Lab at University of Texas Health Science Center at San Antonio found that single-cell RNA-seq reveals a subpopulation of prostate cancer cells with enhanced cell-cycle-related transcription and attenuated androgen response. https://www.ncbi.nlm.nih.gov/pubmed/29233929.

RESEARCH HIGHLIGHTS

Jing Wang Lab at University of Nebraska Medical Center found that TGFβ and IGF1R signaling activate protein kinase A through differential regulation of ezrin phosphorylation in colon cancer cells. http://www.jbc.org/content/early/2018/03/29/jbc.RA117.001299.full.pdf.

Timothy Wang Lab at Columbia University show that catecholamines promote ADRB2-dependent pancreatic ductal adenocarcinoma development and secretion of neurotrophins (NT), which in turn promote tumor innervation leading to increased NE and tumor growth. Blockade of ADRB2 or NT receptors improves gemcitabine's therapeutic effect. http://www.cell.com/cancer-cell/fulltext/S1535-6108(17)30510-X.

Catherine J. Wu lab at Dana-Farber Cancer Institute reviewed the emerging field of personalized cancer vaccination and discussed recent developments and future directions for this promising treatment strategy. https://www.nature.com/articles/nri.2017.131.

POSTDOCTORAL POSITIONS

Two postdoctoral positions are immediately available in the research laboratory of Dr. Xiongbin Lu at Indiana University School of Medicine and Simon Cancer Center. The candidates will participate in funded translational research programs to identify drug targets in cancer immunotherapy and targeted therapy using bioinformatical and experimental approaches. They will have opportunities to collaborate with physician scientists in Indiana University Simon Cancer Center and Vera Bradley Breast Cancer Center. Candidates must have significant hands-on experience in cancer biology and mouse models (xenograft, GEMM models, etc). Experience in immunology is preferred. A competitive salary with an excellent compensation and benefits package will be provided. Please refer to recent publications from the laboratory for more information (*Nature*, 2015, PMID: 25901683; *Nature Communications*, 2016, PMID: 27892457).

Interested individuals should send letter of application, curriculum vitae, and the names and e-mail addresses of three references to:

Dr. Xiongbin Lu
Vera Bradley Foundation Chair in Breast Cancer Innovation
Department of Medical and Molecular Genetics
Indiana University School of Medicine.
E-mail: xiolu@iu.edu.

Applications will be received until the positions are filled.

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。

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Association of Chinese Americans in Cancer Research