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Followed on the successes of the inaugural meeting of the Association of Chinese Americans in Cancer Research (ACACR) in Washington DC last year, the second annual ACACR meeting was held in conjunction with the annual AACR meeting in Chicago on April 14, 2018. Despite an atypical snow and windy weather in Chicago, more than 150 members gathered in a warm and roomy auditorium of Northwestern University Feinberg School of Medicine. The annual meeting started with the presidential address followed by four scientific presentations. Dr. Shi-Yuan Cheng, president of ACACR, reiterated the mission of ACACR is to foster collaborative and innovative research for the treatment and prevention of cancer. He also provided an overview of ACACR accomplishments last year.

After strong nominations from ACACR, more than 8 members were selected as editorial board members for a variety of AACR journals, and 1 member was invited as a panelist of AACR Breast Cancer Research Grants Scientific Review Committee.
In addition, the ACACR Newsletter was launched last year equipped with outstanding editors and editorial team members. Several Newsletters have been published and received enormous appreciation from the community.

The first scientific presentation was delivered by Dr. Chuan He, professor and HHMI investigator at the University of Chicago. Dr. He is a pioneer and leader in RNA biology and mRNA methylation. Of the more than 100 types of chemical modifications identified in cellular RNAs, N$^6$-methyladenosine (m$^6$A) is the most abundant mRNA modification, and plays essential roles in nearly every aspect of the mRNA life cycle, as well as in various cellular, developmental, and disease processes. Dr. He reviewed the breadth and depth of m$^6$A mRNA modification in drug resistance and metastasis of various tumors.
Dr. Shirley Liu, professor and center director for the Functional Cancer Epigenetics at Dana-Farber Cancer Institute, gave the second scientific presentation. Dr. Liu is a renowned scientist and has developed multiple computational platforms and mathematic models to identify key driver gene mutations in carcinogenesis. Through genome-wide transcription factor binding, chromatin dynamics, and gene expression profiles, her group have contributed significant mechanistic insights in tumor biology. She also applied CRISPR/Cas9 technology to screen key molecules responsible for drug resistance in cancers. Her presentation was fascinating and promoted great discussions among members.

Shirley Liu talks about ‘CRISPR Screens and AI on Precision Cancer Medicine’.
This annual meeting was partly sponsored by Medchem Express (MCE). Dr. Zhinong Gao, CEO of MCE, gave a third presentation. Dr. Gao started with a heart-touching personal story on fighting cancer; his wife suffered from metastatic breast cancer and she passed away after a 2-year painful battle with this disease. Motivated by the loss of his lovely wife, Dr. Gao decided to establish the MCE company to develop small molecule inhibitors. Currently, MCE has the larger inhibitor library than NCI; all of these inhibitors are full-characterized, highest purity, and available to all ACACR members at a discount price. His wish is that these efforts can contribute to members’ successes in the fight on cancer. The last presentation was provided by Dr. Jianda Yuan, senior director of the Translational Oncology at Merck. Dr. Yuan reviewed the most recent developments in immune checkpoint blockade. He also discussed dual translational biomarker strategies that may be used to stratify for personal immunotherapy. All of these presentations were on the forefront of cancer research; they were outstanding and received great appreciations from members.
Delicious dinner

All members’ picture after dinner
Research Highlights

Qing Zhang Lab at Lineberger Comprehensive Cancer Center, University of North Carolina School of Medicine found that von Hippel-Lindau (VHL) substrate transcription factor ZHX2 is an oncogenic driver in clear cell renal cell carcinoma.

http://science.sciencemag.org/content/361/6399/290.long

Felix Y. Feng Lab at UCSF showed a comprehensive view of how structural variations affect critical regulators in metastatic prostate cancer. https://www.cell.com/cell/fulltext/S0092-8674(18)30842-0

Howard Y. Chang Lab at Stanford University revealed that IncRNA PVT1 promoter is a tumor-suppressor DNA boundary element.

https://www.cell.com/cell/fulltext/S0092-8674(18)30400-8

Yujiang Geno Shi Lab at Harvard Medical School and Fudan University uncovered a pathway linking diabetes to cancer via glucose-regulated phosphorylation of TET2.

https://www.nature.com/articles/s41586-018-0350-5

Li Yang Lab at National Cancer Institute, USA reported that miR-130a and miR-145 reprogram Gr-1⁺CD11b⁺ myeloid cells and inhibit tumor metastasis through improved host immunity.

https://www.nature.com/articles/s41467-018-05023-9
Zhiguo Zhang Lab at Columbia University found that a novel enhancer located between the promoters of marker of proliferation Ki67 (MKI67) and O6-methylguanine-DNA-methyltransferase (MGMT) genes, is activated in TMZ-resistant patient-derived xenograft (PDX) lines and recurrent tumor samples. This enhancer may regulate MGMT expression and promote TMZ resistance in GBM.

https://www.nature.com/articles/s41467-018-05373-4

Yibin Kang Lab at Princeton University recently published a review on metastatic niche functions and therapeutic opportunities.

https://www.nature.com/articles/s41556-018-0145-9

Zheng-gang Liu Lab at US National cancer institute revealed that necroptosis of tumor cells leads to tumor necrosis and promotes tumor metastasis.

https://www.nature.com/articles/s41422-018-0058-y

Xiaohua Wu Lab at The Scripps Research Institute found the concerted roles of FANCM and Rad52 in the protection of common fragile sites which are prone to chromosomal breakage and are hotspots for chromosomal rearrangements in cancer cells.

https://www.nature.com/articles/s41467-018-05066-y
**Research Highlights**

**Mingjun Zhang** and **Zui Pan**’s Labs at The Ohio State University and UT Arlington found that near infrared fluorescent peptide nanoparticles could enhance esophageal cancer therapeutic efficacy.  
[https://www.nature.com/articles/s41467-018-04763-y](https://www.nature.com/articles/s41467-018-04763-y)

**Wenbin Lin** Lab at The University of Chicago revealed that nanoscale metal-organic frameworks enhance radiotherapy to potentiate checkpoint blockade immunotherapy.  
[https://www.nature.com/articles/s41467-018-04703-w](https://www.nature.com/articles/s41467-018-04703-w)

**Shao-Cong Sun** Lab at MD Anderson Cancer Center found that TBK-binding protein 1 regulates IL-15-induced autophagy and NKT cell survival.  
[https://www.nature.com/articles/s41467-018-05097-5](https://www.nature.com/articles/s41467-018-05097-5)

**Zhenkun Lou** Lab at Mayo Clinic reported that ZNF506-dependent positive feedback loop regulates H2AX signaling after DNA damage.  
[https://www.nature.com/articles/s41467-018-05161-0](https://www.nature.com/articles/s41467-018-05161-0)

**Rihe Liu** and **Leaf Huang**’s Lab at University of North Carolina showed that synergistic and low adverse effect cancer immunotherapy by immunogenic chemotherapy and locally expressed PD-L1 trap.  
[https://www.nature.com/articles/s41467-018-04605-x](https://www.nature.com/articles/s41467-018-04605-x)
Xiaochun Li Lab at UT Southwestern Medical Center collaborating with Jiawei Wang at Tsinghua University in China revealed the structures of human Patched and its complex with native palmitoylated sonic hedgehog.

https://www.nature.com/articles/s41586-018-0308-7

Li Ma Lab at MD Anderson Cancer Center published that SKP2- and OTUD1-regulated non-proteolytic ubiquitination of YAP promotes YAP nuclear localization and activity.

https://www.nature.com/articles/s41467-018-04620-y

Zhenglun Zhu Lab at Brigham and Women’s Hospital recently found that the homeobox protein VentX reverts immune suppression in the tumor microenvironment.

https://www.nature.com/articles/s41467-018-04567-0

Xiaojun Ren Lab at University of Colorado Denver published a paper on live-cell single-molecule dynamics of PcG proteins imposed by the DIPG H3.3K27M mutation. Their data provide mechanisms in which the cancer-causing histone mutation alters the binding and search dynamics of epigenetic complexes.

https://www.nature.com/articles/s41467-018-04455-7
Zhengping Zhuang’s Lab at NIH revealed that pharmacologic inhibition of protein phosphatase-2A achieves durable immune-mediated antitumor activity when combined with PD-1 blockade. [https://www.nature.com/articles/s41467-018-04425-z](https://www.nature.com/articles/s41467-018-04425-z)

Hai Yan’s Lab at Duke University Medical Center recently expanded the genomic landscape of TERT promoter wildtype-IDH wild type glioblastoma. [https://www.nature.com/articles/s41467-018-04448-6](https://www.nature.com/articles/s41467-018-04448-6)

Min Yu Lab at University of Southern California found that TAK1 mediates microenvironment-triggered autocrine signals and promotes triple-negative breast cancer lung metastasis. [https://www.nature.com/articles/s41467-018-04460-w](https://www.nature.com/articles/s41467-018-04460-w)

Mien-Chie Hung Lab at MD Anderson Cancer Center just uncovered that STT3-dependent PD-L1 accumulation on cancer stem cells promotes immune evasion. [https://www.nature.com/articles/s41467-018-04313-6](https://www.nature.com/articles/s41467-018-04313-6)

Hui-Wen Lo at Wake Forest University School of Medicine recently found that truncated glioma-associated oncogene homolog1(tGLI1) mediates mesenchymal glioblastoma via transcriptional activation of CD44. [http://cancerres.aacrjournals.org/content/78/10/2589.full-text.pdf](http://cancerres.aacrjournals.org/content/78/10/2589.full-text.pdf)
**Research Highlights**

**Yang Shi** Lab at Boston Children’s Hospital revealed that LSD1 ablation stimulates anti-tumor immunity and enables checkpoint blockade.  
[https://www.cell.com/cell/fulltext/S0092-8674(18)30715-3](https://www.cell.com/cell/fulltext/S0092-8674(18)30715-3)

**Da Yang** Lab at University of Pittsburgh identified *EPIC1* as an oncogenic IncRNA that interacts with MYC and promotes cell-cycle progression in cancer through IncRNA epigenetic landscape analysis.  
[https://www.cell.com/cancer-cell/fulltext/S1535-6108(18)30110-7](https://www.cell.com/cancer-cell/fulltext/S1535-6108(18)30110-7)

**Bing Xia** Lab at Rutgers Robert Wood Johnson Medical School provided evidence of intertissue differences in the DNA damage response and the pro-oncogenic role of NF-kB in Mice with disengaged BRCA1-PALB2 interaction.  
[http://cancerres.aacrjournals.org/content/78/14/3969](http://cancerres.aacrjournals.org/content/78/14/3969)

**Sue-Hwa Lin** Lab at MD Anderson reported that Organelle-derived Acetyl-CoA can promote prostate cancer cell survival, migration, and metastasis via activation of Calmodulin Kinase II,  
[http://cancerres.aacrjournals.org/content/78/10/2490](http://cancerres.aacrjournals.org/content/78/10/2490); plus they also reported that osteoblast-secreted factors mediate dormancy of metastatic prostate cancer in bone via activation of the TGFβRIII-p38MAPK-pS249/T252RB pathway.  
[http://cancerres.aacrjournals.org/content/78/11/2911](http://cancerres.aacrjournals.org/content/78/11/2911)
**Research Highlights**

**Xiaoqi Liu** Lab at Purdue University revealed that Plk1-mediated phosphorylation of TSC1 enhances the efficacy of rapamycin. [http://cancerres.aacrjournals.org/content/78/11/2864](http://cancerres.aacrjournals.org/content/78/11/2864)

Also they demonstrated that inhibition of the Wnt/beta-Catenin Pathway overcomes resistance to enzalutamide in castration-resistant prostate cancer. [http://cancerres.aacrjournals.org/content/78/12/3147](http://cancerres.aacrjournals.org/content/78/12/3147)

**Guoan Chen** Lab at University of Michigan found that silencing of IncRNA MIR22HG triggers cell survival/death signaling via oncogenes YBX1, MET, and p21 in lung cancer. [http://cancerres.aacrjournals.org/content/78/12/3207](http://cancerres.aacrjournals.org/content/78/12/3207)

**Wei Gu** Lab at Herbert Irving Comprehensive Cancer Center found that Peli1 modulates the subcellular localization and activity of Mdmx. [http://cancerres.aacrjournals.org/content/78/11/2897](http://cancerres.aacrjournals.org/content/78/11/2897)

**Zigang Dong** Lab at the Hormel Institute found that TRAF1 is critical for regulating the BRAF/MEK/ERK pathway in non-small celllcarcinogenesis. [http://cancerres.aacrjournals.org/content/78/14/3982](http://cancerres.aacrjournals.org/content/78/14/3982)

**Y. Alan Wang** Lab at MD Anderson Cancer Center carried out an in vivo screen and identified PYGO2 as a driver for metastatic prostate cancer. [http://cancerres.aacrjournals.org/content/78/14/3823](http://cancerres.aacrjournals.org/content/78/14/3823)
Research Highlights

Hongbo Chi Lab and Jiyang Yu Lab at St Jude Children’s Research Hospital published a work on Hippo/Mst signaling couples metabolic state and immune function of CD8α+ dendritic cells. https://www.nature.com/articles/s41586-018-0177-0

Bing Li Lab at University of Louisville found that expression of adipocyte/macrophage fatty acid-binding protein in tumor-associated macrophages promotes breast cancer progression. http://cancerres.aacrjournals.org/content/78/9/2343

Liguo Wang Lab at Mayo Clinic found that prevalent homozygous deletions of Type I Interferon and defensing genes in human cancers associate with immunotherapy resistance. http://clincancerres.aacrjournals.org/content/24/14/3299

Haifa Shen Lab at Houston Methodist Research Institute found that SMAD4 gene mutation renders pancreatic cancer resistance to radiotherapy through promotion of autophagy. http://clincancerres.aacrjournals.org/content/24/13/3176

Kwok-kin Wong Lab at Laura and Issac Perimutter Cancer Center reported targeting HER2 aberrations in non-small cell lung cancer with osimertinib. http://clincancerres.aacrjournals.org/content/24/11/2594
Guojun Li Lab at MD Anderson Cancer Center published that TGFβ1 genetic variants predict clinical outcomes of HPV-positive oropharyngeal cancer patients after definitive radiotherapy. http://clincancerres.aacrjournals.org/content/24/9/2225

Min Li Lab at University of Oklahoma Health Sciences Center reported that ZIP4 promotes pancreatic cancer progression by repressing ZO-1 and Claudin-1 through a ZEB-dependent transcriptional mechanism. http://clincancerres.aacrjournals.org/content/24/13/3186

Shulin Li Lab at MD Anderson Cancer Center showed T-cell homing therapy for reducing regulatory T cells and preserving effector T-cell function in large solid tumors. http://clincancerres.aacrjournals.org/content/24/12/2920

Gloria Su Lab at Columbia University discovered that DCLK1+ cells originated from a cell lineage distinct from pancreatic PDX1+ progenitors, which challenges the current concept that DCLK1+ cells serve as tumor stem cells for pancreatic ductal adenocarcinoma. https://www.ncbi.nlm.nih.gov/pubmed/29526803
Linheng Li Lab at University of Kansas Medical Center found that retinoid-sensitive epigenetic regulation of the hoxb cluster maintains normal hematopoiesis and inhibits leukemogenesis.  

Sumin Kang Lab at Emory University School of Medicine found that MAST1 drives cisplatin resistance in human cancers by rewiring cRaf-independent MEK activation.  
https://www.cell.com/cancer-cell/abstract/S1535-6108(18)30269-1

Qing Yi Lab at Lerner Research Institute, Cleveland Clinic found that Th9 cells represent a unique subset of CD4+ T cells endowed with the ability to eradicate advanced tumors.  
https://linkinghub.elsevier.com/retrieve/pii/S1535-6108(18)30218-6
ACACR thanks the sponsors

Gold Sponsors
Tanon (biotanon.com)

Silver Sponsors
Medchemexpress, NJ (medchemexpress.com)

Bronze/event Sponsors
Alphacait (alphacait.com)
Dear ACACR Community,

At our second annual meeting in April, Dr. John Wang of Case Western discussed about the possibility of having an ACACR peer-review journal. But most of you expressed concerns about its feasibility due to financial and administrative constraints. At the same time, several members of the ACACR leadership circle suggested to forge a partnership with a journal already in operation. One such journal came up is *Genes & Diseases*, which happens to be run by one of our ACACR founding members Dr. T.-C. He of University of Chicago. The ACACR leadership asked T.-C. to contact the journal’s copyright holder Chongqing Medical University. We are delighted to inform you that the University welcomes the partnership with ACACR. The leadership has appointed John to represent the interest of ACACR, as an Executive Director for ACACR Alliance with *Genes & Diseases*, and works closely with the Editorial Office to revamp the editorial board and related publishing guidelines/polices. In coming months, we will also work out a detailed agreement with the copyholder institute.

*Genes & Diseases* is a peer-reviewed open-access journal launched in September 2014 ([https://www.journals.elsevier.com/genes-and-diseases/](https://www.journals.elsevier.com/genes-and-diseases/)) (see the attached). The journal publishes quarterly by Elsevier Publishing. When our partnership is established, the journal’s peer review will be under the joint responsibilities of Chongqing Medical University (Chongqing, China) and ACACR. The journal primarily focuses on publishing investigations on the molecular bases and experimental therapeutics of human diseases including cancer. The 2017 CiteScore is 4.74, which was ranked at 89th percentile in Biochemistry (#43/398), 87th percentile in Genetics (#12/91), 83rd percentile in Molecular Biology (#62/367) and 83rd percentile in Cell Biology (#43/264). Publication formats include full length research article, review article, short communication, emerging and enabling technologies, correspondence, perspectives, commentary, views on news, newsletter, and research watch. It has been indexed in ESCI, Scopus, ScienceDirect, DOAJ, and PubMed (pending on final approval). The journal is eligible for SCI selection in October 2018. The current hypothetical impact factor is between 5 and 6.
We are excited about this opportunity and encourage all of you to support and actively participate in all aspects of the partnership on behalf of ACACR. We expect this will be an exciting, at the same time challenging intellectual exercise. With your support and commitment, we are confident we will accomplish our goals and make our partnership with *Genes & Diseases* a successful endeavour.

ACACR Executive members
TC He: Chief Editor of *Genes & Diseases*
Zhenghe Wang: Senior Associate Editor of *Genes & Diseases*

https://www.sciencedirect.com/journal/genes-and-diseases
Postdoctoral Fellow and Senior Research Associate Positions in Colorectal Cancer

The Ohio State University James Comprehensive Cancer Center seeks candidates for Postdoctoral Fellows and Senior Research Associates to study molecular mechanisms of colorectal cancer metastasis and drug resistance and to identify and validate novel targets associated with cell survival, invasion, angiogenesis and metastasis using cell culture and mouse models. The goal is to develop effective therapies to treat advanced colorectal cancer patients. The annual salary is $47,484.00 - $65,313.00.

The ideal candidate should have a Ph.D (or doctoral equivalent) degree in Biochemistry, Molecular Biology, Cell Biology, Genetics or related area with a track record of publication in peer-reviewed journals. Expertise in cell culture and mouse models is required and experience with immunology or bioinformatics is preferred.

Applicants should send curriculum vitae, and description of research accomplishments to Wang.12230@osu.edu.
Department of Genetics at Louisiana State University Health Sciences Center (LSUHSC) in New Orleans is seeking postdoctoral scholars with an interest and dedication to translational research and a focus on the role of miRNAs in cancer biology, diagnosis, and therapy. This is a unique opportunity for a motivated candidate to enter a fostering environment of collaborative basic and clinical cancer researchers. Candidates with a PhD and/or MD with strong hands-on experience in cell/molecular biology, signaling transduction, biochemistry, murine models of cancer, or genomics, are encouraged to apply. Applications should contain a current curriculum vitae, list of publications, brief statement of previous research experience and interest, as well as contact information for three references. Inquiries should be addressed to Dr. Yaguang Xi (xilabpostdoc@gmail.com).

LSU Health is an Equal Opportunity Employer for females, minorities, individuals with disabilities and protected veterans.
Two Postdoctoral Fellow positions are available in the Department of Structural and Cellular Biology at Tulane University School of Medicine, New Orleans, LA, USA. The postdoctoral fellow will conduct independent research on prostate cancer. This position requires participation in projects focused on inflammation and prostate cancer initiation and progression using genetically modified animal models. It also includes in vitro and ex vivo experiments on cell lines and animal tissues. The postdoctoral fellow will analyze and summarize collected data in written form and together with principal investigator write manuscripts and publish results of research in scientific journals. Formal application for the job shall go to Tulane Jobs website. For informal inquiries, please email your CV and contact information to: zyou@tulane.edu. The job is available until filled.
Postdoctoral Fellows in Cancer Biology

One position of postdoctoral fellow to study novel molecular mechanisms of cancer progression is available in Dr. Wenliang Li’s lab at the Institute of Molecular Medicine (IMM) of the University of Texas Health Science Center at Houston (UTHealth).

Metastasis is responsible for over 90% of cancer death but its mechanisms are still poorly understood. Research in Dr. Li’s lab is to study novel regulators of cancer metastasis, epithelial-mesenchymal transition (EMT), and drug resistance through a unique combination of RNAi/cDNA screens, cancer genomics, molecular cell biology, mouse models and patient specimens. The goals of the research are to gain new knowledge of cancer progression, identify novel cancer drug targets and develop better therapeutics.

Qualified candidates should have a doctoral degree, strong background and good first-author publications in cancer biology, molecular cell biology, or signal transduction. Prior experience in mouse models is desirable but not required. The postdoc fellow is expected to have strong work ethic, critical thinking abilities, excellent organization and communication skills.

For those interested, please email a CV, contact information of three references, and a cover letter describing past achievements and research interests to Dr. Wenliang Li at: wenliang.li@uth.tmc.edu. Please follow this link (www.uthouston.edu/imm/profile.htm?id=2565029) to learn more about the research programs and recent publications in Dr. Li’s lab. Applicants for other types of positions, such as technicians and research scientists, may also be considered.

Salary of the postdoctoral fellow will follow NIH and university guidelines, with a minimum of $47,484 annually. The Institute is located in the heart of the Texas Medical Center, the world's largest medical center, in the 4th largest US city with a relatively low living cost. UTHealth is an EEO/AA employer. UTHealth does not discriminate on the basis of race, color, religion, gender, sexual orientation, national origin, genetics, disability, age, or any other basis prohibited by law. EOE/M/D/F/V.
Postdoctoral Research Associate

Summary
The laboratory of Dr. Taosheng Chen studies the roles of nuclear receptors PXR and CAR and the CYP3A subfamily of drug–metabolizing enzymes in regulating drug-induced liver toxicity, tumorigenesis, and cancer drug resistance. We develop novel chemical probes/therapeutic leads by using a multidisciplinary approach (biology, chemistry and structural biology), and use them to interrogate the function of PXR, CAR and CYP3A in order to overcome drug toxicity, drug resistance and tumorigenesis in cellular and animal models (Lin et al, Nat Commun. 8:741, 2017).

Responsibilities
The postdoctoral fellow will investigate the molecular mechanisms responsible for the aberrant expression of CYP3A in extrahepatic cancers (possible mechanisms involve transcription and/or alternative splicing, both independent of and dependent on PXR).

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 or other evidence of scientific accomplishment are encouraged to apply. Expertise in cell and molecular biology and significant experience in regulation of gene transcription and splicing are desirable. Candidates must have (or soon receive) a PhD or MD degree.

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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.
我们将会在收到付款后五-七个工作日发出收据。
3. 可以直接在手机上，只用填写姓名和email 等基本信息即可注册。注册链接: http://www.acacr.org/membership.html

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。