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VIEW ON NEWS

Joining the force: ACACR teams up with *Genes & Diseases*

In May 2018, a Maryland-based professional association, namely the Association of Chinese Americans in Cancer Research (ACACR, Baltimore, MD, USA; http://www.acacr.org/) reached the agreement with the copyright holder of *Genes & Diseases*, Chongqing Medical University (Chongqing, China), to form an alliance in scientific publishing of exciting findings in basic, translational, and clinical biomedical research.

ACACR is a non-profit organization and currently comprises of approximately 1000 basic scientists, physician-scientists, and physicians in the areas of basic and translational research, as well as pharmaceutical scientists in the area of anticancer drug development and treatments. While originally from Mainland China and Taiwan, vast majority of the ACACR members are well-established principal investigators in biomedical sciences at Universities and research institutions in the US. Since most of the ACACR members are also active members of American Association of Cancer Research (AACR), ACACR has established a close association with AACR and actively promotes AACR's overall mission, which is to prevent and cure cancer through research, education, communication, and collaboration. The current President of ACACR is Shiyuan Cheng, MD, PhD, Professor of Neurology at the Ken & Ruth Davee Department of Neurology of Northwestern University Feinberg School of Medicine, Chicago, IL, USA, while Zhenkun Lou, PhD, Professor of Oncology at Department of Oncology of Mayo Clinic, Rochester, MN, USA, is the President-Elect for ACACR.

On behalf of the ACACR, Dr. John Z. Wang of Case Western Reserve University School of Medicine serves as the Executive Coordinator for the ACACR Alliance with *Genes & Diseases* to coordinate and facilitate the partnership. Joined by over 50 ACACR members, Dr. Wang will also serve as a Deputy Editor-in-Chief of *Genes & Diseases* to provide scientific expertise on the peer-review process. Dr. John Wang is currently Dale H. Cowan M.D. -Ruth Goodman Blum Professor of Cancer Research and vice chair for faculty development in the Department of Genetics and Genome Sciences at Case Western Reserve University School of Medicine. He is also a co-leader of the Cancer Genetics program at Case Comprehensive Cancer Center since 2014. He was named as a co-leader of the Standup to Cancer (SU2C) Colorectal Cancer Dream Team in 2017. Dr. Wang's research program focuses on defining mechanisms of oncogenesis at the molecular, regulatory and phenotypic levels and translating these discoveries to new cancer therapies. He co-discovered that PIK3CA is frequently mutated in human cancers, and recently found that PIK3CA mutations render colorectal cancer cells dependent on glutamine. He has successfully translated these discoveries into ongoing phase I/II clinical trials. He also discovered protein tyrosine phosphatase (PTP) gene mutations in human cancer and demonstrated that some of the mutated PTPs function as tumor suppressor. Dr. John Wang received his Ph.D. in Microbiology from University of Virginia in 2001. He was a postdoctoral fellow with Dr. Bert Vogelstein in the Sydney Kimmel Comprehensive Cancer Center at Johns Hopkins University. Dr. Wang joined the Department of Genetics and the Case Comprehensive Cancer Center in August, 2005.

The Editor-in-Chief, T.–C. He, MD, PhD of The University of Chicago Medical Center and Deputy Editor-in-Chief, Ailong Huang, Professor and the President of Chongqing Medical University, welcome the ACACR alliance with *Genes & Diseases*, and look forward to forging a strong partnership with ACACR to improve and enhance the publication quality of the journal.

Selected research highlights from ACACR members

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.

Peer review under responsibility of Chongqing Medical University.

https://doi.org/10.1016/j.gendis.2018.07.004
2352-3042/

Li Ding Lab at Washington University in St. Louis conducted a comprehensive analysis of oncogenic driver genes and mutations in >9000 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. 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.

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).

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).

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.

He, 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.

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.

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.

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.

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.

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.

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.

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.

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.

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).

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.

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.

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.

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.

Wenyi Wei Lab at Harvard Medical School found that phosphorylation of EZH2 by AMPK suppresses PRC2 methyltransferase activity and oncogenic function.

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.

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.

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.

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.

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.

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.

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.

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.

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.

Fei Li
Editorial Office of Genes & Diseases, Chongqing, China

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