15th International Congress on American Pathology and Oncology Research &

International Conference on Microbial Genetics and Molecular Microbiology

December 03-04, 2018 | Chicago, USA

Keynote Forum DAY 1

American Pathology & Molecular Microbiology 2018

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Bing-Hua Jiang

The University of Iowa, USA

MicroRNAs and epigenetic change in cancer development and therapeutic resistance

Cancer incidences have been increasing worldwide. Rapid progress in cancer diagnosis and treatment has been accomplished during last 10 years. However, the therapeutic resistance still is the major hinder for cancer treatment. Our goal is to reveal new molecular mechanisms of chemoresistance and radioresistance and to identify novel diagnosis and treatment options to overcome therapeutic resistance in lung, breast and ovarian cancers. We found that suppression of several miRNAs including miR-152, miR-148a and miR-145; induction of reactive oxygen species (ROS) and epigenetic changes play important roles in cancer development and therapeutic resistance. To understand the mechanism of miRNA suppression, we found that DNA methylation, transcriptional regulation, and histone H3 lysine 27 trimethylation are key factors in inducing miRNA suppression which regulates cancer development and autophagy. We also find that in addition to the gene mutations, signaling molecule activation and ROS are new mechanism of acquired resistance of lung cancer cells to EGFR-TKIs treatment.

Biography

Bing- Hua Jiang obtained his PhD degree from Mississippi State University in 1994, then started the first post-doc training in The Johns Hopkins University School of Medicine (JHU). He originally cloned hypoxia-inducible factor 1α (HIF- 1α) in JHU. He identified different functional domains of HIF- 1α for regulating HIF-1 transcriptional activation activity and many direct targets of HIF-1 including VEGF and heme oxygenase-1. He then moved to the Scripps Research Institute to have further post-doc training in 1997, where he studied the mechanism of PI3K in regulating different functions in different cells and animal. He initially demonstrated that PI3K and AKT play important roles in tumor angiogenesis by inducing VEGF and HIF- 1α expression. Since he established his own lab in 2000, our lab has demonstrated that oxidative stress and microRNA dysregulations are important in cancer development, drug resistance, tumor growth, and angiogenesis through epigenetic changes. He have published more than 157 research papers in peer-reviewed journals and his papers have more than 30,000 citations, H-index: 71.

bing-hua-jiang@uiowa.edu

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Weibiao Cao

Rhode Island Hospital and Brown University, USA

Mechanisms of the progression from Barrett's esophagus to esophageal adenocarcinoma: Role of NADPH oxidase NOX5-S

E sophageal adenocarcinoma (EA) has increased in incidence over the past several decades and is characterized by a poor prognosis. Gastroesophageal reflux disease (GERD) complicated by Barrett's esophagus (BE) is a major risk factor for the development of EA. There is a progression from BE to dysplasia and to EA. However, the mechanisms of progression from BE to EA are not fully understood. We found that NOX5-S is present in Barrett's cells BAR-T and EA cells FLO and OE33 and is overexpressed in FLO cells and EA tissues. NOX5-S mRNA is also increased in Barrett's mucosa with high-grade dysplasia. Pulsed acid treatment significantly increases NOX5-S expression and H₂O₂ production in BAR-T and OE33 cells and Barrett's mucosa. These data suggest that NOX5-S may be a source of overproduction of reactive oxygen species (ROS) in BE and in EA cells. We also found that acid-induced increase in NOX5-S expression may depend on activation of Rho kinase, ERK1/2 MAP kinases, and cAMP response element-binding protein. The acid may also induce production of platelet-activating factor, which activates signal transducer and activator of transcription 5 (STAT5) and then upregulates NOX5-S. In addition, NOX5-S mediates an acid-induced increase in cell proliferation in Barrett's cells BAR-T and EA cells (OE33 and FLO). NOX5-S-mediated increase in cell proliferation may depend on the activation of COX2 and microsomal prostaglandin E synthase 1 (mPGES1), and downregulation of p16 via promoter methylation. NOX5-S also mediates acid-induced DNA damage. These data suggest that persistent acid reflux present in BE patients may upregulate NOX5-S, increase production of ROS and cell proliferation, and cause DNA damage, thereby contributing to the progression from BE to dysplasia and to EA.

Biography

Weibiao Cao obtained his MD degree from Zhejiang Medical University in Hangzhou, China in 1986. He worked as a Research Associate and an Assistant Professor in Division of Gastroenterology, Rhode Island Hospital, RI, the USA from 1996 to 2007. He was trained as a Pathology Resident from 2007 to 2011 and a GI pathology fellow from 2011 to 2012 in the Department of Pathology, Rhode Island Hospital and Brown University, USA. He has been an attending pathologist since 2012, an Associate Professor since 2013 and a Director of the autopsy service since 2017 in Department of Pathology, Rhode Island Hospital and Brown University, USA and an Editorial Board Member of Scientific Reports (impact factor 4.26) since 2015.

weibiao_cao@brown.edu

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University of Illinois College of Medicine, USA

Bacterial view of the chemical periodic table: Genes (and proteins) for each element of the chemical periodic table

icrobial activities often provide the basis for useful environmental and agricultural biotechnology, as well as frequently \mathbf{L} causing problems. Essentially all bacteria have genes for toxic metal ion resistances and these include those for Ag⁺, AsO₂⁺, AsO43-, Cd2+, Co2+, CrO42-, Cu2+, Hg2+, Ni2+, Pb2+, TeO32-, Tl+, and Zn2+. Resistance to inorganic Hg2+ and to organomercurials such as CH₁Hg⁺ and phenylmercury involve a series of metal-binding and membrane transport proteins as well as the enzymes mercuric reductase and organomercurial lyase. Hg is methylated and demethylated by microbial processes. The methylmercury of concern in human food is of microbial origin and microbial bioremediation and phytoremediation can clean polluted sites. Arsenic resistance and metabolizing systems occur in three forms, the widely-found ars operon that is present in most bacterial genomes and many plasmids, the more recently-recognized the aso genes for the periplasmic arsenite oxidase that serves as an initial electron donor in aerobic resistance to arsenite and the functionally-related arr genes for arsenate reductase that serves as a terminal electron acceptor in anaerobic respiration. The largest group of resistance systems function by energy-dependent efflux of toxic ions. Some of the efflux resistance systems are ATPases and others are chemiosmotic ion/proton exchangers. For example, Cd²⁺ efflux pumps of bacteria are either inner membrane P-type, ATPases or three polypeptide RND chemiosmotic complexes consisting of an inner membrane pump, a periplasmic- bridging protein and an outer membrane channel. Silver compounds are increasingly used in industrial, environmental and medical applications. A cluster of 9 silver-specific genes make proteins that bind extracellular Ag⁺ or pump internalized Ag⁺ out from the cells, using membrane potential or ATP hydrolysis for energy. The SilE periplasmic Ag⁺ binding protein is an unusual small soluble protein that binds 5 Ag⁺ cations with 10 histidine residues.

Biography

Simon Silver has over 60 years of bacterial molecular genetics research experience, including a PhD from MIT, postdoctoral times at the MRC (UK) and the University of California Berkeley, followed by professorial appointments at Washington University (St Louis) and the University of Illinois (Chicago). He has published almost 250 papers and edited 9 published monographs. He was Editor in Chief of 2 journals and editor or editorial board member of more than a dozen more.

simon@uic.edu

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Shunyou Gong

Northwestern University Feinberg School of Medicine, USA

Basic principles and emerging concepts in Epstein-Barr virus-positive marginal zone lymphoma

E pstein-Barr virus (EBV) is well known to be associated with various types of B cell, T cell, and NK cell lymphomas. The roles febV in lymphomagenesis are now better elucidated through decades of research. Marginal zone lymphomas (MZL) are a heterogeneous group of small B-cell lymphomas and traditionally EBV negative. However, recent studies have identified EBV-positive extranodal MZLs particularly in transplant recipients and at least partially responsive to reduced immunosuppression, suggesting that these should be regarded as a form of post-transplant lymphoproliferative disorders (PTLD). We expanded the spectrum of EBV+ MZLs by identifying the first case of nodal MZL, and more cases of extranodal MZL but in non-transplant settings that included iatrogenic immunosuppression, congenital immune deficiency, and increased age as the only potential cause of immune dysfunction. These cases were either EBV latency I or II, with a typical plasmacytoid and/or monocytoid B cells positive for EBV in all cases. Unlike published series that were predominantly IgA-positive, our cases were either positive for IgG or IgM. Cases arising from cutaneous sites and salivary glands demonstrated differing characteristic features in morphology. Our data show that EBV+ MZLs can arise in a variety of clinical settings and are most often extranodal. Most patients had a clinically indolent disease with response to reduction of immune suppression, or immunochemotherapy. As these lymphomas warrant different management strategies compared to EBV-negative cases, a high suspicion must be kept, and relevant workup should be performed for nodal and extranodal MZLs in post-transplant and non-transplant but immunocompromised patients.

Biography

Shunyou Gong is the director of Hematology and Hematopathology at the Ann & Robert H Lurie Children's Hospital of Chicago and assistant professor at Northwestern University Feinberg School of Medicine. His clinical interests and areas of expertise include pediatric hematopoietic malignancies, inherited bone marrow failure syndromes, and bleeding or thrombotic disorders. As first-author or corresponding author, he has published many landmark papers on prestigious journals including Cell, Blood, and American Journal of Surgical Pathology.

shunyou.gong@northwestern.edu

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Astghik Z Pepoyan

International Association for Human and Animals Health Improvement, Armenia

Probiotics for Familial Mediterranean fever disease

amilial Mediterranean fever (FMF), the disorder caused by mutations in the inflammasome pyrin, usually meet in people of Mediterranean origin, but it may be described for any ethnic group, too. While there is no cure for this disorder, one may be able to relieve signs and symptoms of FMF or even prevent them altogether by adhering to a highly effective treatment: colchicine, 1-2mg/day, for life. While colchicine is effective for FMF, it is not without side effects: many patients taking the colchicine report general gastrointestinal upset. Physicians suggest probiotics but this varies greatly by a doctor. The impact of probiotic formulations Narine and probiotic Colibacteron (Vitamax-E, Yerevan) on blood and gut microbiota of patients with FMF were previously shown by us (Balayan et al., 2015; Pepoian et al., 2015). Our data proposed that M694V/V726A pyrin inflammasome mutations differently act on microbial community structure in male/female FMF patients (Pepoyan et al., 2018). Research questions will be discussed: (i) are structure and function of the fecal microbiota of FMF patients taking or not taking probiotics any different, and (ii) are the observed changes systemic or simply due to individual differences among FMF patients?

Biography

Astghik Z Pepoyan has completed her PhD in 1990 and Doctoral degree in 2002 (Institute of Biochemistry at NAS RA, Armenia). She is the President of the International Association for Human and Animals Health Improvement, and the Head of Food Safety and Biotechnology department at Armenian National Agrarian University. She is the author of 250 scientific publications.

apepoyan@gmail.com