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Journal of Antivirals & Antiretrovirals

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Proceedings of 2nd World Congress on

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Accelerating Scientific Discovery

Virology

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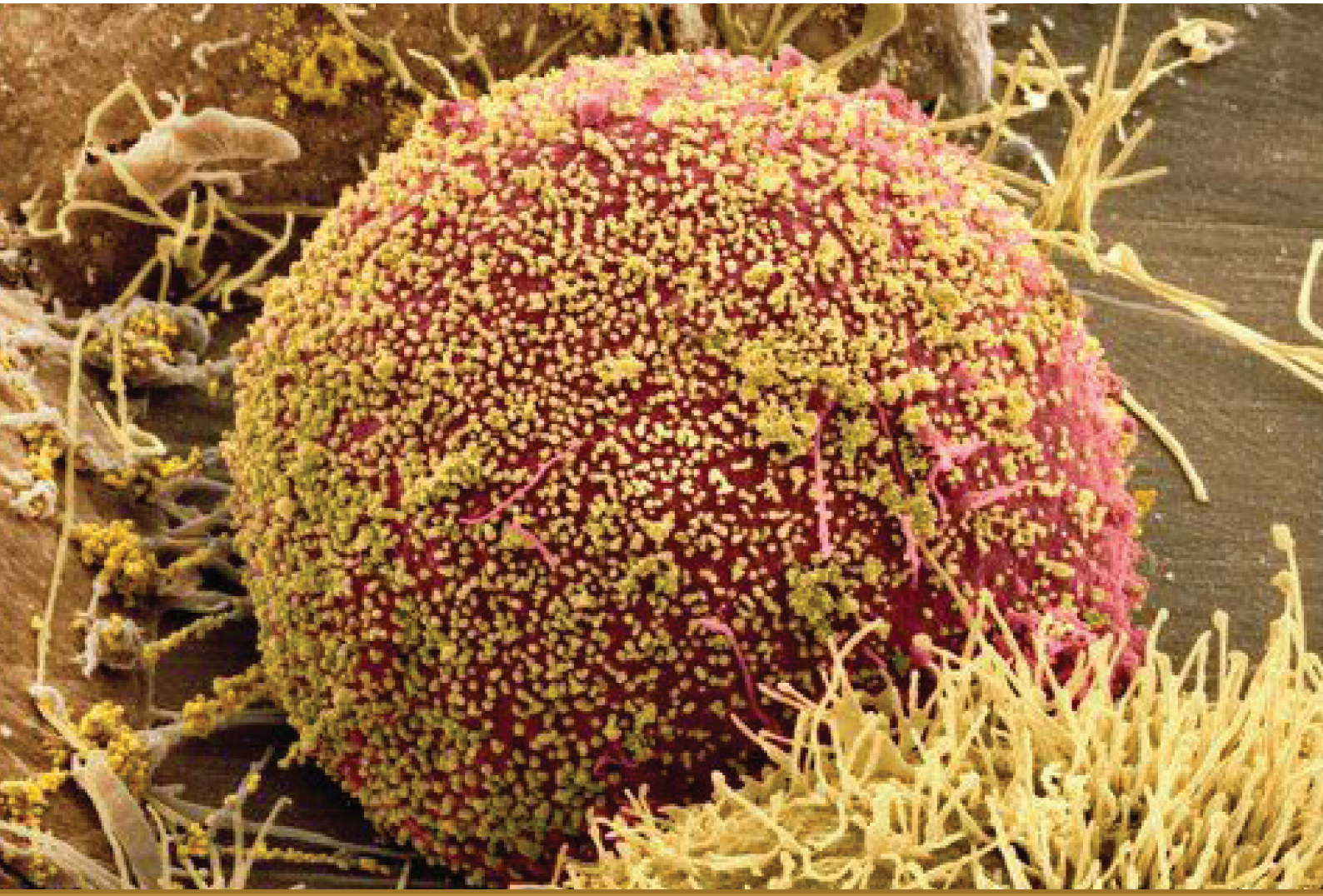

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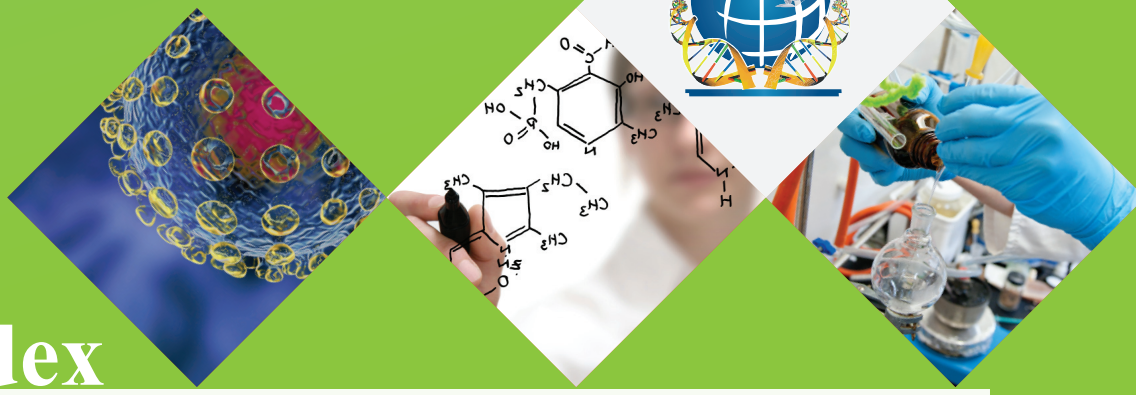

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Impact Factors (IF) and Index Copernicus Values (ICV)

OMICS Publishing Group - Journal Name	PubMed Abbreviation	IF	ICV
Journal of Analytical & Bioanalytical Techniques	J Anal Bioanal Tech	2.63	5.33
Journal of AIDS & Clinical Research	J AIDS Clin Res	4.9	5.3
Journal of Addiction Research & Therapy	J Addict Res Ther	1.13	5.22
Journal of Aquaculture Research & Development	J Aquacult Res Dev	0.62	5.15
Journal of Allergy & Therapy	J Allergy Ther	1.07	4.47
Journal of Anesthesia & Clinical Research	J Anesth Clin Res	0.48	5.13
Journal of Antivirals & Antiretrovirals	J Antivir Antiretrovir	2.65	4.81
Journal of Biometrics & Biostatistics	J Biom Biostat	1.5	4.47
Journal of Bioequivalence & Bioavailability	J Bioequiv Availab	2	-
Journal of Bioanalysis & Biomedicine	J Bioanal Biomed	1.21	6.17
Journal of Biosensors & Bioelectronics	Biosens Bioelectron	3.57	-
Journal of Bioterrorism & Biodefense	J Bioterror Biodef	1.27	4.66
Journal of Bioremediation & Biodegradation	J Bioremed Biodeg	3.5	5.07
Journal of Cancer Science & Therapy	J Cancer Sci Ther	4.25	5.09
Journal of Cell Science & Therapy	J Cell Sci Ther	3.14	4.14
Journal of Clinical & Experimental Ophthalmology	J Clin Exp Ophthalmol	0.5	4.9
Journal of Carcinogenesis & Mutagenesis	J Carcinogen Mutagen	4.14	5.04
Journal of Chemical Engineering & Process Technology	J Chem Eng Process Technol	-	5.04
Journal of Clinical & Experimental Cardiology	J Clin Exp Cardiolog	1.07	5.3
Journal of Clinical & Cellular Immunology	J Clin Cell Immunol	2.11	4.45
Journal of Clinical Research & Bioethics	J Clin Res Bioeth	1.14	4.1
Journal of Clinical & Experimental Dermatology	J Clin Exp Dermatol Res	1.13	5.27
Journal of Computer Science & Systems Biology	J Comput Sci Syst Biol	2.53	5.81
Journal of Cytology & Histology	J Cytol Histol	1.04	4.4
Journal of Chromatography & Separation Techniques	J Chromat Separation Techniq	2.07	4.81
Journal of Datamining in Genomics & Proteomics	J Data Mining Genomics Proteomics	1.33	4.55
Journal of Diabetes & Metabolism	J Drug Metab Toxicol	3.21	6.07
Journal of Drug Metabolism & Toxicology	J Diabetes Metab	1.27	-
Journal of Earth Science & Climatic Change	J Earth Sci Climate Change	0.66	-
Journal of Food Processing & Technology	J Food Process Technol	2.15	4.87
Journal of Forensic Research	J Forensic Res	0.05	4.4
Journal of Genetic Syndromes & Gene Therapy	J Genet Syndr Gene Ther	1.44	-
Hydrology: Current Research	Hydrol Curr Res	0.38	4.93
Journal of Molecular Biomarkers & Diagnosis	J Mol Biomark Diagn	2.04	4.37
Journal of Microbial & Biochemical Technology	J Microb Biochem Technol	2.04	5.51
Journal of Neurology & Neurophysiology	J Neurol Neurophysiol	0.83	-
Journal of Nanomedicine & Nanotechnology	J Nanomed Nanotechnol	5.72	4.04
Journal of Proteomics & Bioinformatics	J Proteomics Bioinform	2.22	9
Pharmaceutica Analytica Acta	Pharmaceut Anal Acta	1.4	5.33
Journal of Pharmacogenomics & Pharmacoproteomics	J Pharmacogenomics Pharmacoproteomics	1.3	4.71
Journal of Petroleum & Environmental Biotechnology	J Petrol Environ Biotechnol	3.14	4.81
Journal of Plant Pathology & Microbiology	J Plant Pathol Microb	0.42	4.54
Journal of Steroids & Hormonal Science	J Steroids Horm Sci	1.66	4.37
Journal of Thermodynamics & Catalysis	J Thermodynam Cat	0.14	-
Journal of Tissue Science & Engineering	J Tissue Sci Eng	2.17	4.88
Journal of Vaccines & Vaccination	J Vaccines Vaccin	1.73	6.26
Journal of Veterinary Science & Technology	J Vet Sci Technol	-	4.22

Impact Factor Calculation:

*Unofficial 2011 Impact Factors were established by dividing the number of articles published in 2010 and 2011 to that were cited in 2011 based on a search of the Google Scholar Citation Index database, by the number of articles published in the previous two years (2010 and 2011).
A = the number of times, articles published in 2010 and 2011, were cited by indexed journals during 2011
B = the total number of "citable items" published by that journal in 2010 and 2011.

Impact Factor (IF) = A/B
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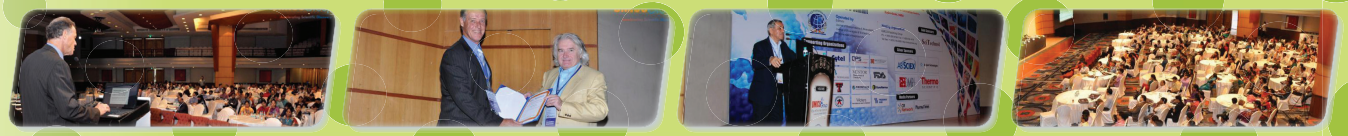

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Journal of Antivirals & Antiretrovirals

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Antivirals are compounds that are used to prevent or treat viral infections. Antiretrovirals are medications used for treating the infections caused by retroviruses, primarily HIV. The Journal of Antivirals & Antiretrovirals provides an open access platform to bring awareness about viral infections, and emphasize on the innovative and contemporary methods of research to manufacture pharmaceuticals.

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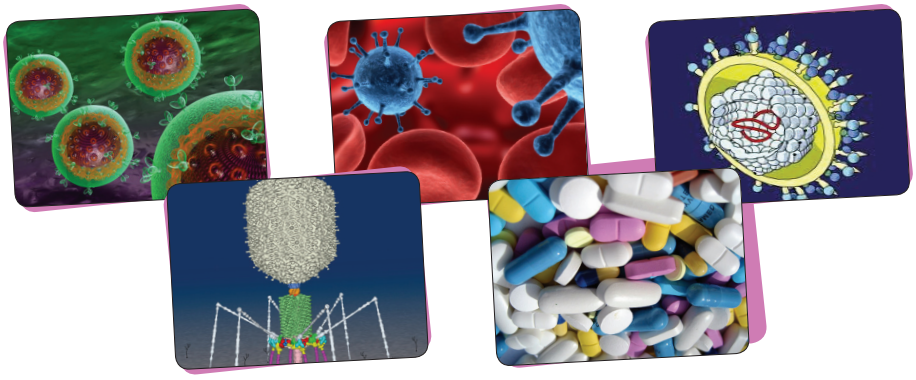

Upcoming Special Issues

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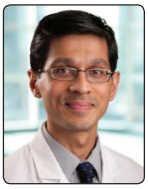	**Hepatitis: Genomics and Sequencing Analysis** **Sandeep Mukherjee** Section of Gastroenterology and Hepatology Nebraska Medical Center, USA Tel. +1-402-559-3139 Fax: +1-402-559-6132	University of Nebraska Medical Center
	**Epidemology: Viral Diseases** **Sita Awasthi** Division of Infectious Disease University of Pennsylvania Philadelphia, USA Tel. +1-215-662-3186 Fax: +1-215-349-5111	Penn
	**Clinical & Basic Hepatology: Diagnosis** **Jiatong Zhuo** Deputy Director Centers for Diseases Control and Prevention Guangxi Zhuang Autonomous Region, China Tel. +86-711-530-2374 Fax: +86-771-531-9615	CDC
	**HIV Drugomics** **Alfredo Garzino-Demo** Department of Microbiology and Immunology University of Maryland School of Medicine, USA Tel. +1-410-706-4689 Fax: +1-410-706-4694	University of Maryland Baltimore
	**Genetics: Viral Diseases** **Zhen Li** Deputy Director Institute of Animal Science and Veterinary Medicine Shanghai Academy of Agricultural Sciences Shanghai, P.R.China Tel. +86-21-62206391	saln UK-China Sustainable Agriculture Innovation Network
	**Antivirals: New Drug Delivery Systems** **Yiguang Jin** Beijing Institute of Radiation Medicine China Tel. +86-106-693-1220 Fax: +86-106-821-4653	

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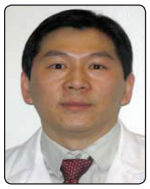
	**Antivirals: New Drug Delivery Systems** **Jingdong Tian** Duke University USA Tel. +1-919-684-3494 Fax: +1-919-684-4488	
	**Antivirals: New Drug Delivery Systems** **Limin Chen** University of Toronto Toronto, Canada Tel. +1-416-946-3435 Fax: +1-416-978-8528	University of Toronto
	**Antiretrovirals: New Drug Delivery Systems** **Marvin S Reitz** Institute of Human Virology University of Maryland Biotechnology Institute USA Tel. +1-410-706-4679 Fax: +1-410-706-4694	University of Maryland University College
	**HIV: New Therapeutics** **Zhiwei Chen** The University of Hong Kong Hong Kong Tel. +852-2819-9831 Fax: +852-2817-7805	香港大學 THE UNIVERSITY OF HONG KONG
	**Stability of RNA Virus Attenuation Approaches** **Chu Jang Hann** Department of Microbiology National University of Singapore, Singapore Tel. +65-6516-3278 Fax: +65-6776-6872	NUS National University of Singapore
	**Antiretroviral Drug Development for HIV: Challenges and Perspectives** **Honglin Zhou** Center for Resuscitation Science Hospital of the University of Pennsylvania Philadelphia, USA Tel. +1-215-746-8938 Fax: +1-215-746-1224	University of Pennsylvania Health System

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Journal of AIDS & Clinical Research

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Acquired Immuno Deficiency Syndrome is a disease of the human immune system caused by HIV. Clinical research is a branch of medical science that determines the safety and effectiveness of medications, devices, diagnostic products and treatment regimens intended for human use. Journal of AIDS & Clinical Research is a multidisciplinary journal that aims to keep scientists, clinicians, researchers, students, and general public aware of the latest research on AIDS.

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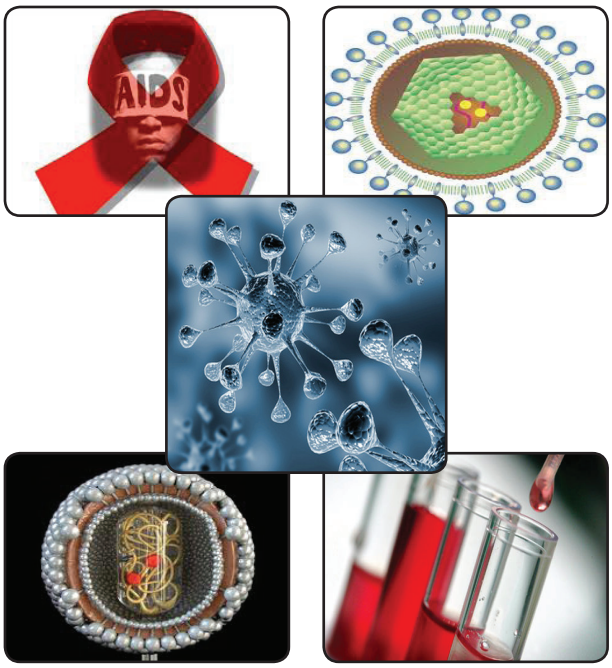
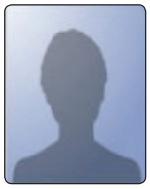


Upcoming Special Issues

Handling editor	Title of the special issue	Logo of the University / Institute
	**Risk Behaviors: HIV/AIDS** **Karl Peltzer** Human Sciences Research Council, South Africa Tel. +27-12-3022637	HSRC Human Sciences Research Council
	**Pharmacology of Antiretroviral Agents: HIV** **Di WU** Department of Clinical Pharmacology & Therapeutics The Children's Hospital of Philadelphia, USA Tel. +1-215-590-8797	The Children's Hospital of Philadelphia® Hope lives here.
	**Co-infections: HIV** **Claudia Goulston** Department of Infectious Diseases and Internal Medicine University of Utah, USA	THE UNIVERSITY OF UTAH
	**Epidemiology and Prevention: HIV** **Han-zhu Qian** Department of Medicine Vanderbilt University School of Medicine, USA Tel. +1-615-343-3159	VANDERBILT UNIVERSITY
	**Epidemiology and Prevention: HIV** **Paula Frew** Division of Infectious Diseases Emory University, USA	EMORY UNIVERSITY
	**Drug Resistance: HIV** **Claudia Goulston** Department of Infectious Diseases and Internal Medicine University of Utah, USA	THE UNIVERSITY OF UTAH

Handling editor	Title of the special issue	Logo of the University / Institute
	**Risk Analysis and Modeling, Matrics: HIV** **Berhanu Tameru** Center for Computational Epidemiology, Tuskegee University, USA Tel. +1-334-724-4275	TUSKEGEE UNIVERSITY
	**Pharmacodynamics and Pharamacokinetics of New Therapeutics: HIV** **Shi Lizheng** Department of Health Systems Management and Internal Medicine, Tulane University, USA	Tulane University
	**HIV Latency and Reactivation** **Chifu Huang** Center for Oral Health Research University of Kentucky, USA Tel. +1-859-323-0281	UK UNIVERSITY OF KENTUCKY
	**Physical Activity Associated with HIV** **Randall Keyser** Department of Rehabilitation Science, George Mason University, USA	GEORGE MASON UNIVERSITY
	**Vaccine Research: HIV** **Mariana Varela** Inflammation College of Medical, University of Glasgow, Scotland	University of Glasgow
	**Vaccine Research: HIV** **Marc Van Regenmortel** Research Institute of the School of Biotechnology University of Strasbourg France	UNIVERSITÉ DE STRASBOURG

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2nd International Conference on
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Emerging Cell Therapies
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DoubleTree by Hilton Philadelphia, USA

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October 29-31, 2012
DoubleTree by Hilton Chicago-Northshore, USA

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International Conference on
Anesthesia & Perioperative Care
November 26 - 28, 2012
Hilton San Antonio Airport, USA

International
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DoubleTree by Hilton Philadelphia Center City, USA

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DoubleTree by Hilton Philadelphia Center City, USA

International Conference on
Obesity & Weight Management
December 3-5, 2012
DoubleTree by Hilton Philadelphia, USA

3rd International Conference on
Pharmaceutics & Novel Drug Delivery Research
April 8-10, 2013
Hilton Suites Chicago/Magnificent Mile, USA

2nd World Congress on
Metabolomics & Systems Biology
April 8-10, 2013
Hilton Suites Chicago/Magnificent Mile, USA

3rd International Conference on
Clinical & Experimental Ophthalmology
April 15-17, 2013
Hilton Chicago/Northbrook, USA

3rd International Conference on
Clinical & Experimental Dermatology
April 15-17, 2013
Hilton Chicago/Northbrook, USA

3rd International Conference on
Clinical & Experimental Cardiology
April 15-17, 2013
Hilton Chicago/Northbrook, USA

4th World Congress on
Bioavailability & Bioequivalence
May 20-22, 2013
DoubleTree by Hilton Beijing, China

2nd International Conference and Expo on
Biometrics & Biostatistics
June 10-12, 2013
Hilton Chicago/Northbrook, USA

2nd International Conference on
Gastroenterology & Urology
June 10-12, 2013
Hilton Chicago/Northbrook, USA

2nd International Conference and Exhibition on
Biosensors & Bioelectronics
June 17-19, 2013
Hilton Chicago/Northbrook, USA

2nd International Conference and Exhibition on
Neurology & Therapeutics
June 17-19, 2013
Hilton Chicago/Northbrook, USA

3rd World Congress on
Virology
July 11-13/8-10, 2013
Embassy Suites Las Vegas, USA

3rd International Conference on
Proteomics & Bioinformatics
July 15-17, 2013
DoubleTree by Hilton Philadelphia Center City, USA

2nd International Conference and Exhibition on
Nutritional Science and Therapy
July 15-17, 2013
DoubleTree by Hilton Philadelphia Center City, USA

4th International Conference on
Biomarkers & Clinical Research
July 16-17, 2013
DoubleTree by Hilton Philadelphia Center City, USA

2nd International Conference on
Earth Science & Climate Change
July 22-24, 2013
Embassy Suites Las Vegas, USA

2nd International Conference and Exhibition on
Addiction Research & Therapy
July 22-24, 2013
Embassy Suites Las Vegas, USA

International Conference on
Animal & Dairy Sciences
July 23-24, 2013
Embassy Suites Las Vegas, USA

2nd International Conference and Exhibition on
Nephrology & Therapeutics
July 29-31, 2013
Embassy Suites Las Vegas, USA

3rd International Conference on
Vaccines & Vaccination
July 29-31, 2013
Embassy Suites Las Vegas, USA

2nd International Conference and exhibition on
Pathology
August 5-7, 2013
Embassy Suites Las Vegas, USA

International Conference on
Integrative Biology Summit
August 5-7, 2013
Embassy Suites Las Vegas, USA

International Conference and Exhibition on
Personalized Medicine & Molecular Diagnostics
August 5-7, 2013
Embassy Suites Las Vegas, USA

2nd International Conference and Exhibition on
Orthopedics & Rheumatology
August 19-21, 2013
Embassy Suites Las Vegas, USA

International Conference on
Physical Medicine & Rehabilitation
August 19-21, 2013
Embassy Suites Las Vegas, USA

International Conference on
Dental & Oral Health
August 19-21, 2013
Embassy Suites Las Vegas, USA

International Conference and Exhibition on
Solar Systems & Power Generation Technologies
August 19-21, 2013 Hyderabad, India

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OMICS Group Conferences

Accelerating Scientific Discovery

2nd World Congress on

Virology

August 20-22, 2012 Embassy Suites Las Vegas, USA

Relevant Conferences

International Conference on Pathology

August 27-29, 2012 DoubleTree by Hilton Philadelphia Center City, USA

Theme: "To Promote the Rapid Translation of Scientific Research into the Practice of Pathology and Laboratory Medicine"

International Conference on Pathology (Pathology-2012) is a remarkable event which brings together a unique and International mix of large and medium pathology research leading universities and pathology research institutions making the conference a perfect platform to share experience, foster collaborations across industry and academia, and evaluate emerging technologies across the globe.

Scientific Tracks

Track 1: Anatomic Pathology

Track 1-1 Surgical pathology and cytopathology

Track 1-2 Veterinary pathology

Track 1-3 Cardiovascular pathology

Track 1-4 Gynecologic pathology

Track 1-5 Urological and renal pathology

Track 1-6 Pulmonary pathology

Track 2: Pathology Immunohistochemistry

Track 2-1 Diagnostic immunohistochemistry

Track 2-2 Tumor immunohistochemistry

Track 2-3 Diagnostic markers

Track 2-4 Antigen detection methods

Track 2-5 Monoclonal antibodies and their use in treatment

Track 3: Pathology and Laboratory Medicine

Track 3-1 Neuropathology

Track 3-2 Ophthalmic pathology

Track 3-3 Dental pathology

Track 3-4 Pediatric pathology

Track 3-5 Transplant pathology

Track 3-6 Infection control

Track 3-7 Serological and immunological tests

Track 4: Plant Disease Management

Track 4-1 Impact of environmental conditions on plant diseases

Track 4-2 Integrated disease management strategies

Track 4-3 Chemical control of plant diseases

Track 4-4 Biological control methods

Track 5: Plant Defense Mechanism

Track 5-1 Plant signaling behavior

Track 5-2 Production of defensive alkaloids/ alkaloid biosynthesis

Track 5-3 Production of glycosides and defense response

Track 5-4 Role of phenolics in plant defense

Track 5-5 Molecular mechanisms of disease resistance

Track 6: Areas of Current Research in Pathology

Track 6-1 Microbial infections and drug development

Track 6-2 Cancer genetics and molecular cancer therapeutics

Track 6-3 Immunology and response to infection

Track 6-4 Genomic influence in disease acquisition

Track 7: Plant Virology

Track 7-1 Viral pathogens and disease control

Track 7-2 Plant molecular virology

Track 7-3 Transmission and diagnosis of plant viruses

Track 7-4 Plant viruses and recent developments

Track 7-5 Biochemical changes in plants infected by the virus

Track 8: Bacterial Pathology and Disease Control

Track 8-1 Plant bacterial diseases - causes and prevention

Track 8-2 Bacterial meningitis - diagnosis and treatment

Track 8-3 Bacterial endocarditis - disease prophylaxis

Track 8-4 Role of bacterial endotoxins in pathogenesis

Track 8-5 Acquisition of bacterial resistance

Track 9: Forensic Pathology

Track 9-1 Forensic medicine

Track 9-2 Autopsy studies

Track 9-3 Environmental forensics

Track 9-4 Forensic DNA analysis

Track 10: Clinical Pathology and Diagnostic Tools

Track 10-1 Hematopathology and disease treatment

Track 10-2 Transfusion medicine

Track 10-3 Molecular pathology and disease diagnostics

Track 10-4 Immunopathology

Track 10-5 Dermatopathology

Track 11: Current Research in Plant Pathology

Track 11-1 Plant disease etiology

Track 11-2 Diagnosis of plant diseases

Track 11-3 Plant genomics and proteomics

Track 11-4 Plant-microbial interactions and plant cell biology

Track 11-5 Biology and ecology of plant pathogens

OMICS Group is organizing Pathology-2012 with the overwhelming support from more than 200 Editorial Board members of related journals, that includes Journal of Clinical & Experimental Pathology and Journal of Plant Pathology & Microbiology.

Organizing Committee Members

Confirmed Speakers Including

Amy Hong Zhang
University of Texas, USA

Guillaume Vogt
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Jason X. Cheng
University of Michigan, USA

Ozlen Saglam
Yale University, USA

Akanchha Kesari
Children's National Medical Center, USA

Xinkang Wang
Translational Science, USA

Jyotika Sharma
University of North Dakota, USA

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Hospital for Sick Children, Canada

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Naotaka Furuichi
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2nd World Congress on
Cancer Science & Therapy

September 10-12, 2012 Hilton San Antonio Airport, San Antonio, Texas, USA

Theme: "Emerging Trends in Cancer Science and Therapy"

The emerging therapies and medicines in cancer research are the result of painstaking scientific experiments and discoveries made in the various universities and research institutes. We invite all such laboratories and researchers to associate with us and share their research with the world. The goal of this conference is to contribute a global impact against cancer through research, training, education.

Scientific Tracks

Track 1: Cancer Cell Biology
Track 1-1 Cancer stem cells and metastatic growth
Track 1-2 Cell to cell signaling and membrane proteins
Track 1-3 Inflammation and inflammatory factors in cancer development
Track 1-4 Metastatic cell growth and adhesion; apoptosis and cell division
Track 1-5 Cancer biology
Track 1-6 Tumor virology and pathology
Track 1-7 Cancer systems biology

Track 2: Diagnostic and Prognostic Cancer Biomarkers
Track 2-1 Biomarkers in cancer therapy and molecular diagnostics
Track 2-2 Genetic and DNA methylation-based biomarkers
Track 2-3 Prognostic biomarkers
Track 2-4 Biomarkers based on cancer types
Track 2-5 Biomarkers for cancer cell process
Track 2-6 Biomarkers for pathogenic process
Track 2-7 Biomarkers for pharmacologic responses

Track 3: Advances in Cancer Detection and Imaging
Track 3-1 Fluorescence imaging techniques
Track 3-2 Digital mammography and computer-aided detection system
Track 3-3 Electrical impedance scanning
Track 3-4 Nanotechnology based detection
Track 3-5 Tumor microarrays

Track 4: Cancer: Management & Prevention
Track 4-1 Diet and physical exercise
Track 4-2 Chemoprevention
Track 4-3 Cancer epidemiology and prevention
Track 4-4 DNA damage and repair

Track 5: Cancer Therapy
Track 5-1 Radiotherapy and chemotherapy
Track 5-2 Gene therapy and tumor immunity
Track 5-3 Hormone replacement therapy
Track 5-4 Molecular-targeted therapies
Track 5-5 Cancer: gene expression and protein profiling; surgery and laparoscopy
Track 5-6 Stem cells and cancer cell therapy
Track 5-7 Senescence induction
Track 5-8 Bacterial treatment
Track 5-9 Telomerase therapy
Track 5-10 Drug therapies

Track 6: Clinical Cancer Research
Track 6-1 Clinical medicine
Track 6-2 Targeting anticancer drugs
Track 6-3 Therapeutic targets in cancer
Track 6-4 Clinical research in cancer immunology
Track 6-5 Nanotechnology and novel drug delivery systems
Track 6-6 Therapeutic cancer vaccines
Track 6-7 Antioxidants as cancer preventive agent

Track 7: Carcinogenesis and Mutagenesis
Track 7-1 Metabolism of carcinogens
Track 7-2 Identification and detection of carcinogens
Track 7-3 Biological and external factors for carcinogenesis
Track 7-4 Mutagenesis- directed, site-specific and mismatched
Track 7-5 Environmental mutagenesis

Track 8: OMICS in Cancer Research
Track 8-1 High-throughput DNA sequencing technologies
Track 8-2 Operomics
Track 8-3 Bioinformatics
Track 8-4 Viral oncoproteomics

Track 9: Organ-Specific Cancers
Track 9-1 Head and brain cancer
Track 9-2 Nasopharyngeal, oral cavity, and thyroid cancers
Track 9-3 Neck, breast cancer, ovarian, and cervical cancer
Track 9-4 Oesophageal and gastrointestinal cancers
Track 9-5 Rectum, liver, cholecyst, and pancreatic cancer
Track 9-6 Bone,Leukemia, and myelodysplasia
Track 9-7 Lymphoma, melanoma skin cancer, and sarcomas
Track 9-8 Lung and prostate cancer
Track 9-9 Colorectal or colon cancers
Track 9-10 Urology, kidney/renal cancers
Track 9-11 Pediatric oncology

Track 10: Cancer Genetics
Track 10-1 Oncogenes
Track 10-2 Tumour suppresor genes
Track 10-3 Viruses and human cancer
Track 10-4 Genetics and toxicity
Track 10-5 Gene silencing and genome imprinting
Track 10-6 Gene silencing and RNA interference
Track 10-7 Non-coding RNA in cancer
Track 10-8 DNA sequencing in cancerous cells

Track 11: Overcoming inequalities in cancer patients- A case study
Track 11-1 Cancer research: clinical and experimental

Cancer Science-2011 Report

The first International Conference on Cancer Science was organized at Renaissance Hotel, Las Vegas, USA during August 15-17, 2011. Prominent researchers from the NIH and other major industrial federations participated at this event leading it to a successful and well appreciated conference. New discoveries in cancer research and its subsequent analysis were shared at the conference. Opening remarks & Keynote lectures were delivered by eminent scientists attracting 300-500 attendees.

With the previous excellence & success we are happily announcing 2nd World Congress on Cancer Science & Therapy on September 10-12, 2012 at San Antonio, USA. Cancer Science-2012 is confident of delivering the same success & worldwide participation.

OMICS Group is organizing Cancer Science-2012 with the overwhelming support from more than 200 Editorial Board members of related journals, that includes Journal of Cancer Science and Therapy, Journal of Carcinogenesis & Mutagenesis and Chemotherapy : Open Access

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Chongqing University, China

Homer Black
Baylor College of Medicine, USA

George G Chen
The Chinese University of Hong Kong, China

Join your Peers @ Cancer Science-2012

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International Conference on
Biothreats and Biodefense

October 15-17, 2012 DoubleTree by Hilton Chicago-North Shore, USA

Theme: "Making Research Safer by Combating Threats from Forms of Life"

The International Conference on Biothreats and Biodefense (Biodefense-2012) tries to ground a platform bringing together scientific intellects to develop transformative counter measures for known and unknown biothreats. The Conference will serve the methodological ground for researchers and policymakers to discuss the materialistic concepts and take a holistic approach to address the challenges posed by technologies and new and re-emerging diseases. Biodefense-2012, a three day event consists of a scientific program of comprehensive talks, special sessions and oral & poster presentations of peer-reviewed contributions.

Scientific Tracks

Track 1: Public Health in Concern to Biothreats and Biodefense
Track 1-1 Epidemiology: health patterns in population
Track 1-2 Outbreak modeling of diseases
Track 1-3 Integrative approach for systematic health development
Track 1-4 Air and water quality for public health
Track 1-5 Global climate change and human health

Track 2: Biothreats due to Infectious diseases
Track 2-1 Microbial pathogens as biological threats
Track 2-2 Zoonotic diseases
Track 2-3 Anti- infectives as biothreat: chemical modifications
Track 2-4 Toxins as biothreat agents
Track 2-5 Increased resistivity of pathogens against drugs
Track 2-6 Virulent strains: genetic modifications in pathogens

Track 3: Biosurveillance-Turning Science into Tools for Inspection
Track 3-1 Biothreat surveillance systems
Track 3-2 Biothreat information systems

Track 4: Biocrimes-Legal context of Biothreats and Biodefense
Track 4-1 Scientific implication
Track 4-2 Social implication

Track 5: Biosecurity-Risk Assessment, Analysis and Management
Track 5-1 Inside threat of high containment labs
Track 5-2 Safety level determination
Track 5-3 Risk report and lab security
Track 5-4 Catastrophic biothreats: social vulnerability of the cascadic threats

Track 6: Biothreat Agents
Track 6-1 Physical agents
Track 6-2 Chemical agents
Track 6-3 Biological agents
Track 6-4 Radiological agents

Track 7: Biodetection-Exercising the Identification of Biological Threats
Track 7-1 Analytical approaches
Track 7-2 Nanotechnological approaches
Track 7-3 Genomic approaches
Track 7-4 Computational approaches
Track 7-5 Molecular approaches
Track 7-6 Clinical applications

Track 8: Environmental Biodefense-Defending Environment against Biological Terror
Track 8-1 Ecosystem restoration:reclamation affects on the natural diversity
Track 8-2 Biothreat associated with climate change
Track 8-3 Pollution measurement and management
Track 8-4 Outbreak investigation

Track 9: Technology Implementation in Biodefense Research
Track 9-1 Systems biology in medical development
Track 9-2 System integration and response for biologics threat detection
Track 9-3 Synthetic DNA
Track 9-4 RNA into biodefense
Track 9-5 Transduction and multiplexing
Track 9-6 Vaccines

Track 10: Government and Policies
Track 10-1 Scientific diplomacy for global security
Track 10-2 Controlling materials and information
Track 10-3 Priorities from WMD commission report
Track 10-4 Accessibility of point of care diagnostics to the developing world

Track 11: Challenges and Future Prospects
Track 11-1 Research resource development
Track 11-2 Research focus
Track 11-3 Human errors and system failures
Track 11-4 Funding outlook

Track 12: Role of Organizations in the Implementation of Biosecurity Measures
Track 12-1 Funding agencies
Track 12-2 Scientists/researchers
Track 12-3 Institutions, universities and industries
Track 12-4 Government bodies and law enforcement officials
Track 12-5 Media members and policy analysts

Track 13: Components of Biosecurity Measures
Track 13-1 Physical security
Track 13-2 Personnel reliability and security
Track 13-3 Scientific program management
Track 13-4 Pathogen control and accountability
Track 13-5 Transportation and information security
Track 13-6 Scientific biosecurity

OMICS Group is organizing Biodefense-2012 with the overwhelming support from more than 150 Editorial Board members of related journals, that includes Journal of Bioterrorism & Biodefense

Organizing Committee Members

Confirmed Speakers Including

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Aurelio Bonavia
Theraclone-Sciences, USA

Todd H. Rider
Massachusetts Institute of Technology, USA

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Michael Whiteside
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Charlotte Sortedahl
University of Wisconsin, USA

Jochebed Ade-Oshifogun
Chicago State University, USA

Jamie L. Johnson
Western Illinois University, USA

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Journal of Bioterrorism and Biodefense

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International Conference on
Clinical & Cellular Immunology

October 22-24, 2012 DoubleTree by Hilton Chicago-Northshore, USA

Theme: "Innovative Strategies for the Development of Immune Response"

International Conference on Clinical & Cellular Immunology (**Immunology-2012**) will ensure a highly interactive Scientifically stimulative and intense program. The plenary sessions and theme based conference will be addressed by an outstanding list of international speakers who will highlight recent advances in various aspects of immunology with focus on emerging research in the field of Clinical & Cellular Immunology. Immunology-2012, a three day event consists of a scientific program of comprehensive talks, special sessions and oral & poster presentations of peer-reviewed contributions.

Scientific Tracks

Track 1: Molecular and Cellular Immunology

Track 1-1 Infection and immune response

Track 1-2 Bacterial pathogenesis and immune response

Track 1-3 Animal virology and pathogenesis

Track 1-4 Molecular and functional immunology

Track 2: Innate and Adaptive Immune System

Track 2-1 Molecular pathways for human natural killer cells

Track 2-2 Structural and functional parameters of antimicrobial peptides (AMPs)

Track 2-3 Selective control mechanisms of AMPs

Track 2-4 Innate-adaptive interactions. How they regulate immune response?

Track 3: Inflammatory/Autoimmune Diseases

Track 3-1 Organ-specific autoimmune disorders

Track 3-2 Major histocompatibility complex classes

Track 3-3 Role of Th17 cells and their importance

Track 3-4 Linking of autoimmunity and inflammation with metabolic diseases

Track 3-5 Systemic lupus erythematosus-pathogenesis, diagnosis and treatment

Track 3-6 Novel discoveries in therapeutic options for rheumatoid arthritis

Track 3-7 Multiple sclerosis

Track 4: Hepatitis

Track 4-1 Acute and chronic viral hepatitis

Track 4-2 Hepatitis-induced cirrhosis

Track 4-3 Epidemiological aspects of hepatitis infections

Track 4-4 Challenges in the diagnosis and treatment of autoimmune hepatitis

Track 5: Immunomodulation and Immunotherapy

Track 5-1 Stem cell therapy

Track 5-2 Alternative targets for immunointervention

Track 5-3 Pharmacological immunosuppression

Track 5-4 Clinical strategies to induce transplantation tolerance

Track 5-5 Gene therapy for autoimmune diseases

Track 5-6 Induced immunosuppression therapy

Track 6: Metastasis and Cancer Therapy

Track 6-1 Epithelial stem cells and cancer

Track 6-2 Interplay of cancer stem cells and metastasis

Track 6-3 Advances in imaging and detection of cancer stem cells and metastatic precursors

Track 6-4 Model studies for hematopoietic microenvironments

Track 6-5 Genetic and epigenetic regulation of cancer cells

Track 6-6 Tumorigenesis and tumor suppression

Track 6-7 Cancer surveillance

Track 7: Vaccines in Immunology: New Insights and Development

Track 7-1 Recombinant vaccines and immunotherapeutics

Track 7-2 Cancer vaccines

Track 7-3 Bacterial and viral vaccines

Track 7-4 AIDS vaccines

Track 7-5 T-Cell vaccines

Track 7-6 Synthetic vaccines

Track 7-7 Contraceptive vaccines

Track 7-8 Anti inflammatory drugs and antiretroviral therapy

Track 8: Emerging Issues in Immunology Research

Track 8-1 Osteoimmunology

Track 8-2 Nano-medicine immunology

Track 8-3 Mucosal immunology and translational immunology

Track 8-4 Autophagy in immune regulation

Track 9: Biomarkers and Techniques in Immunology

Track 9-1 RISET biomarkers

Track 9-2 ITN biomarkers

Track 9-3 Hybridoma technology

Track 9-4 Assays for immunology

Track 9-5 Flow cytometry and immunoelectrophoresis

OMICS Group is organizing Immunology-2012 with the overwhelming support from more than 200 Editorial Board members of related journals, that includes Journal of Clinical and Cellular Immunology, Journal of Clinical Case Reports and Journal of Cytology and Histology

Organizing Committee Members

Confirmed Speakers Including

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Janet Plate
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University of Louisville, USA

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University of Nebraska Medical Center, USA

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International Conference on
Clinical Microbiology & Microbial Genomics

November 12-14, 2012 Hilton San Antonio Airport, USA

Theme: "Exploring New Territories to Fight Microbial Resistance"

International Conference on Clinical Microbiology & Microbial Genomics (**Clinical Microbiology-2012**) encourages in exchanging the knowledge and experiences with the developing trends in the corresponding fields. **Clinical Microbiology-2012**, a three day event consists of a scientific program of comprehensive talks, special sessions and oral & poster presentations of peer-reviewed contributions

Scientific Tracks

Track 1: Microbiology-Basic Science
Track 1-1 Microbial ecology
Track 1-2 Animal models including experimental treatment
Track 1-3 Pharmaceutical microbiology
Track 1-4 Food microbiology
Track 1-5 Agricultural microbiology
Track 1-6 Biofilm

Track 2: Antimicrobials & Microbial Techniques
Track 2-1 Mechanism of action and resistance
Track 2-2 Resistance surveillance
Track 2-3 Antimicrobials & clinical trials
Track 2-4 Surveys of molecular epidemiology of resistance and resistant genes
Track 2-5 *In vitro* antibacterial susceptibility and drug interaction studies
Track 2-6 Microbial strains & antibiotic applications
Track 2-7 Advances in microbial/biochemical methodology
Track 2-8 Microbiological techniques

Track 3: Medical Microbiology
Track 3-1 Medical microbiology and diagnosis
Track 3-2 Clinical parasitology
Track 3-3 Clinical mycology
Track 3-4 STD & vaginal infections
Track 3-5 Enteric infections
Track 3-6 Clinical aspects of antibiotics and resistance
Track 3-7 Resistance and mechanism of action of antifungals
Track 3-8 New therapeutic approaches in prevention of fungal infections
Track 3-9 Diagnosis of hepatitis and control

Track 4: Infection Control & Immunity
Track 4-1 Multifactorial mechanisms for regulation of inflammation
Track 4-2 Host pathogen interactions
Track 4-3 Immunity to microbial infections
Track 4-4 Usage of vaccines & therapies against infectious diseases
Track 4-5 Immune response, immune evasion & immune tolerance
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Track 4-7 Tuberculosis and its control
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Track 5: Diagnostics
Track 5-1 Diagnostic/Laboratory methods
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Track 6: Clinical & Experimental Pathology
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Track 9: Clinical ID
Track 9-1 Infection in the immune compromised host and transplant recipients
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Track 9-4 Lyme borreliosis and toxoplasmosis
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Track 10: Case Studies in Clinical Microbiology

Track 11: Microbial Genomics
Track 11-1 Bioinformatics sequence and genome analysis
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Track 11-3 Microarray expression analysis
Track 11-4 Proteomics
Track 11-5 Metagenomics
Track 11-6 Comparitive genomics
Track 11-7 Phylogenetics
Track 11-8 Microbial genetics
Track 11-9 Genome evolution and environmental genomics
Track 11-10 Single cell genomics review

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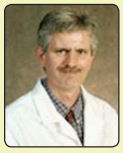

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Keynote Forum

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Keynote Lecture

Nevan J. Krogan

California Institute for Quantitative Biomedical Research School of Medicine, USA

Systems approaches for understanding the HIV-host functional interface

Biography

Dr. Krogan is an Associate Professor in the Department of Cellular and Molecular Pharmacology at the University of California-San Francisco and is an expert in the fields of functional genomics and systems biology. He was born and raised in Regina, Saskatchewan, Canada and obtained his undergraduate degree from the University of Regina. As a graduate student at the University of Toronto, Dr. Krogan led a project that systematically identified protein complexes in the model organism, *Saccharomyces cerevisiae*, through an affinity tagging-purification/mass spectrometry strategy. This work led to the characterization of 547 complexes, comprising over 4000 proteins, and represents the most comprehensive protein-protein interaction map to date in any organism. To complement this physical interaction data, Dr. Krogan developed an approach, termed E-MAP (or epistatic miniarray profile), which allows for high-throughput generation and quantitative analysis of genetic interaction data. Dr. Krogan's lab at UCSF focuses on applying these global proteomic and genomic approaches to formulate hypotheses about various biological processes, including transcriptional regulation, DNA repair/ replication and RNA processing. He is now developing and applying methodologies to create genetic and physical interactions between pathogenic organisms, including HIV and TB, and their hosts, which is providing insight into the human pathways and complexes that are being hijacked during the course of infection.

1 (i) : Vaccines and Host Cell Responses

Session Chair
Radha Maheshwari
Uniformed Services University of the Health Sciences, USA

Session Co-Chair
Ralph Tripp
University of Georgia, USA

Title: **Modulation of interferon pathway by Chloroquine, Tunicamycin, and environmental pollutants: Correlation with the worsening of virus infection**
Radha Maheshwari, Uniformed Services University of the Health Sciences, USA

Title: **Proteomic analysis indicates the association of calreticulin down-regulation and cyclophilin A up-regulation with Type I interferon antagonism of Japanese encephalitis virus NS5 protein**
Cheng-Wen Lin, China Medical University, Taiwan

Title: **Molecular modeling and conformational epitope prediction of nucleocapsid protein region from Japanese encephalitis virus**
A.G. Ingale, North Maharashtra University, India

Title: **Novel approaches for inactivation of viruses for the development of second generation vaccine**
Paridhi Gupta, Uniformed Services University of the Health Sciences, USA

Title: **Autoimmune diseases; prophylaxis by vaccination against their microbial triggers**
Duncan D. Adams, University of Otago, New Zealand

Title: **Development of genetically recombinant rabies vaccine**
Muhammad Saleem Haider, Punjab University, Pakistan

Title: **HIV-1 p6 – A novel interaction partner to the host-cellular protein CypA**
Sara M. Ø. Solbak, University of Bergen, Norway

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Modulation of interferon pathway by Chloroquine, Tunicamycin, and environmental pollutants: Correlation with the worsening of virus infection

Radha K. Maheshwari[1], Paridhi Gupta[1,2], Veena Menon[1], Anuj Sharma[1], Manish Bhomia[1,2], Jing Han[3], Raj K. Puri[3] and N.Balakathiresan[1]

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Previously we have shown that treatment with chemotherapeutic agents such as chloroquine (CHL) or tunicamycin (TM) or exposure to environmental pollutants e.g., cadmium (Cd), manganese (Mn) or lead (Pb) and co-infection with malaria can cause increased pathogenesis and mortality associated with several viruses including Semliki Forest Virus (SFV), Venezuelan equine encephalitis virus (VEEV) and encephalomyocarditis virus (EMCV) in mice.In the present study, we have investigated the mechanism of enhanced pathogenesis by these agents. A global gene expression analysis of brains from mice infected with VEEV and treated with TM, revealed increased and early modulation of genes involved in VEEV pathogenesis such as interferon (IFN) signaling and antigen presentation pathway. Quantitative RT-PCR identified down-regulation of IFNα receptor-1 (IFNAR1) transcript levels. MicroRNAs predicted to down-regulate the IFNAR1 expression were also found to be significantly up-regulated in these mice. Similarly, down-regulation of IFNAR1 expression was also observed in SFV infected mice treated with Cd or VEEV infected mice treated with CHL.

Though the precise mechanism(s) underlying the potentiating effects of these agents on viral infections is not fully understood, results from our study revealed an impairment of the IFN pathway. These results are particularly important since the use of CHL as an antiviral agent has recently been advocated in humans based on in-vitro studies. Our findings are thus, of particular importance from a public health perspective, suggesting that indiscriminate exposure/use of agents like antimalarials can predispose the population to increased morbidity from viral infections. Therefore, in depth studies are warranted before recommending the use of CHL against important virus infections in humans especially in HIV and malaria endemic areas.

The opinions expressed herewith are those of the authors and do not reflect those of USUHS, BITS or FDA.

Biography

Radha K Maheshwari received his PhD from Kanpur University, Kanpur, India in the field of Virology. He performed his post-doctoral research at National Institute of Arthritis, Metabolism and Digestive Diseases, NIH from 1977-1980. Currently, he is a Professor in the Pathology department at Uniformed Services University of The Health Sciences, Bethesda. He has been appointed as an adjunct faculty member at Birla Institute of Technology and Sciences, India to coordinate the research activity of exchange graduate students with the preceptor USUHS faculty. He has also served as an adjunct faculty member at Georgetown University, Washington, D.C. from 1977 to 1982. Over the past 30 years, he has established highly successful and productive national and international collaborations and published more than 125 scientific papers in highly reputed journals. Areas of research in his laboratory include: studying the molecular mechanisms of viral pathogenesis and neuro-degeneration by alphaviruses, development of vaccine candidates against alphavirus infections, prevention of ischemia/reperfusion and hemorrhage induced injuries, enhancement of wound healing and cancer chemoprevention by phytochemicals, identification of biomarkers against TBI and PTSD.

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Proteomic analysis indicates the association of calreticulin down-regulation and cyclophilin A up-regulation with Type I interferon antagonism of Japanese encephalitis virus NS5 protein

Cheng-Wen Lin and Tsung- Han Hsieh
Department of Medical Laboratory Science and Biotechnology, China Medical University, Taiwan

Japanese encephalitis virus (JEV) non-structural protein 5 (NS5) exhibits a type I interferon (IFN) antagonistic function. This study intends to characterize the type I IFN antagonism mechanism of JEV NS5 protein using proteomic approaches. TE-671 human neuroblastoma cells transfected with the control vector and NS5-expressing plasmid were tested their responses to interferon (IFN)-β; the expression profiles of transfected cell lines were analyzed using two-dimensional electrophoresis (2-DE) and mass spectrometric (MS). JEV NS5 reduced IFNβ-induced responses, e.g. IFN-sensitive response element (ISRE) promoter activity, mRNA expression of PKR and OAS, phosphorylation of STAT1 and STAT3. Proteomic analysis and Western blotting demonstrated JEV NS5 up-regulating peroxiredoxin-1, heat shock protein 60, stress induced phosphoprotein 1, and cyclophilin A, and down-regulating heterogeneous nuclear ribonucleoprotein H3, prohibitin, thioredixin and calreticulin in human medulloblastoma TE671 cells in the presence of IFNβ. Calreticulin down-regulation and cyclophilin A up-regulation implied the activation of Ca2+-depedent phosphatase calcineurin. The calcineurin inhibitor, cyclosporin A (CsA), sensitized IFNβ-induced responses in NS5-expressing cells. Importantly, the combined treatment of CsA and IFNβ induced a time-dependent increase of STAT1, STAT3, mTOR and AKT phosphorylation in NS5-expressing cells. The combined treatment of CsA and IFNβ also activated mRNA expression of IFN-stimulated genes, showing more potently inhibitory effects on virus yield. JEV NS5 induced calreticulin down-regulation and cyclophilin A up-regulation, being associated with Type I interferon antagonism via the activation of Ca2+-depedent phosphatase calcineurin. This study shows insights into a possible mechanism of Type I interferon antagonism by JEV NS5, being applicable for elucidating JE pathogenesis.

Biography

Professor Cheng-Wen Lin has completed his Ph.D from National Tsing-Hua University in 2003. He is the Dean of Student Affairs in China Medical University since 2009. He has published more than 50 papers in reputed journals.

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Molecular modeling and conformational epitope prediction of nucleocapsid protein region from Japanese encephalitis virus

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The 3D structure of a protein is a prerequisite for structure based drug design as well as for identifying the conformational epitopes that are essential for the designing vaccines.

A 3-dimensional model (3D) was developed for the nucleocapsid protein of Japanese encephalitis virus. A homology modeling method was used for the prediction of the structure. For the modeling, one template proteins were obtained by mGenTHERADER, namely the high-resolution X-ray crystallography structure of NS3 protease helicase of murry vally encephalitis virus(2WV9). By comparing the template protein a rough model was constructed for the target protein using SWISSMODEL, a program for comparative modelling. The model was validated using protein structure checking tools such as Verify3D and Anola for reliability. The total of 138 such epitope regions/sites have been identified by kolaskar and tongaokar method. Conformational epitopes are mapped from the 3D structure of nucleocapsid protein of Japanese encephalitis virus modeled using the concept of an antigenic domain. The information thus discussed provides insight to the molecular understanding of nucleocapsid protein of JE virus. The predicted 3-D model may be further used in characterizing the protein in wet laboratory.

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Novel approaches for inactivation of viruses for the development of second generation vaccine

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Alphaviruses are highly infectious enveloped viruses that have been identified as emerging infectious pathogens that have credible bioweapon and bioterror capacity. Currently, there is no FDA approved vaccine for any of the alphaviruses. Therefore, development of safe and efficacious vaccine against alphaviruses is urgently required. Traditional methods of virus inactivation for vaccine preparation have inherent drawbacks like poor immunogenicity due to loss or damage of surface epitopes and in some cases incomplete inactivation. Here we describe novel approaches of virus inactivation which result in better immunogenicity and complete inactivation of viruses.

A photoactive aryl azide, 1, 5 iodonapthyl azide (INA), was used to inactivate Venezuelan equine encephalitis virus (VEEV) and chikungunya virus (CHIKV). INA has been shown to partition into the hydrophobic domain of the bio-membrane and upon short irradiation with UV light covalently binds to the transmembrane domain of the membrane proteins without affecting their ectodomains. In another approach, we used a radio-protective Mn2+-peptide-phosphate complex, which was originally identified in highly radio-resistant bacteria, Deinococcus radiodurans. VEEV was inactivated by exposing it to supralethal doses of gamma irradiation (up to 50,000 kGy) in the presence of Mn2+-peptide-phosphate complexes.

Both the inactivation strategies resulted in completely inactivated and highly immunogenic virus particles. INA treatment resulted in inactivation of the infectious viral genome whereas gamma irradiation resulted in denaturation of the viral genome. INA-inactivated VEEV also protected mice from aerosol challenge with the virulent virus. Our findings show that INA acts by two different mechanism of virus inactivation, one by targeting the viral envelope proteins and second by targeting the infectious RNA genome. These findings present novel approaches towards developing highly immunogenic, safe and completely inactivated second generation viral vaccine(s).

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Autoimmune Diseases; prophylaxis by vaccination against their microbial triggers

Duncan D. Adams
University of Otago, New Zealand

It is now apparent that all autoimmune diseases have microbial triggers, exemplified by Type A beta-haemolytic streptococci for rheumatic carditis and the more recent discovery by Ebringer that the bacteria Proteus mirabilis trigger rheumatoid arthritis and other bacteria, Kebsiella pneumoniae trigger ankylosing spondylitis. The pathogenesis of the autoimmune diseases is now solved, being Burnet's Forbidden Clone Theory. This states that somatic mutations in multiplying lymphocytes produce the Forbidden Clones of lymphocytes that cause the autoimmune diseases by accidentally reacting with a host component instead of a microbial one. The genetics of the autoimmune diseases is also solved, being Adams and Knight's H Gene Theory, which states that Histocompatibility antigens, major, minor and H-Y, dictate the immune response repertoire by deleting complementary clones and so alter the risk of occurrence of the various autoimmune diseases. It is apparent that prophylaxis of autoimmune diseases could be achieved by vaccinating against their triggering microbes. This has been demonstrated with Salk's poliomyelitis vaccine, which has prevented the leg paralyses that can now be seen to have been rare autoimmune complications of virtually universal poliovirus infection. Search for microbial triggers of autoimmune diseases is an urgent medical research necessity, none greater than for the probable virus that triggers schizophrenia.

Biography

Duncan Adams entered Medicine with a view to doing research on asthma. Sir Charles Hercus apprenticed him to Dr HD Purves to use radioactive iodine in thyroid research. This led to discovery of the autoantibodies that cause Graves' disease, winning the Van Meter Prize, and enabling confirmation of Burnet's

Forbidden Clone Theory of the pathogenesis of the autoimmune diseases. With John Knight, Adams solved the genetics of autoimmune disease with the H Gene Theory, confirmed by Alan Ebringer who has discovered the microbial triggers of two autoimmune diseases. Discovering and vaccination against microbial triggers will enable prophylaxis of autoimmune diseases.

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Development of genetically recombinant rabies vaccine

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[3]Veterinary Research Institute, Pakistan
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Pakistan is among those few countries where Rabies is endemic and great fear for human as well as livestock poulation. Countless animals which are exposed to rabies died either due to non availiblity of potent rabies vaccine or use of sheep brain originated Semple vaccine. Farmers cannot afford to buy expensive tissue culture vaccine. Here we describe a genetic recombinant rabies virus cell culture vaccine. The backbone of the virus is rabies virus ERA, with the glycoprotein (G) substituted with a Pakistan rabies virus G. The resultant genetically recombined virus was recovered by reverse genetic system. Hundred percent and eighty percent mice survived vaccinated through intramuscular route and per oral route respectively when they were exposed to the challenge rabies virus while eighty percent unvaccinated mice died when they were exposed to rabies virus challenge. On the basis of our results, we suggest our vaccine a good candidate for Rabies vaccination in Pakistan after trials in large animals. It will not only save precious lives, livestock population but also save a huge amount of foreign exchange and provide cell culture vaccine virus to farmer at very cheap rates.

Biography

Muhammad Saleem Haider has completed his Ph.D at the age of 31 years from University of London, Imperial College of Science, Technology and Medicine, UK and postdoctoral studies from University of Toronto, Canada. He is the Director of Institute of Agricultural Sciences, University of the Punjab, Lahore, Pakistan. He has published more than 50 papers in reputed journals and serving as an Editor In-Chief for Mycopath, and editorial and reviewer member for many journals of International repute. He is also member for a no. of professional organizations and office bearer as Vice President (Punjab) for Pakistan Phytopathological Society.

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HIV-1 p6 – A novel interaction partner to the host-cellular protein CypA

Sara M. Ø. Solbak
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Novel drug targets are needed to ensure optimal drugs in the treatment of viral diseases. Targeting host-cellular components necessary for the virus has been suggested as one approach to overcome problems with drug resistance. However, an obstacle to this approach is our lack of knowledge about the viruses host-cellular interactions. The 52 amino acid HIV-1 p6 protein is known to be necessary for the formation of new infectious HIV-1 viruses. Intriguingly, we recently discovered a novel interaction between HIV-1 p6 and the host-cellular protein Cyclophilin A (CypA). A particular structural feature of p6 is the unusually high relative content of proline residues, located at positions 5, 7, 10, 11, 24, 30, 37 and 49 in the sequence. We detected proline cis/trans isomerism for all these proline residues to such an extent that more than 40% of all p6 molecules contained at least one proline in a cis conformation. 2D 1H NMR analysis of full-length HIV-1 p6 and p6 peptides established that CypA interacts as a PPIase with all proline residues of p6. Only catalytic amounts of CypA were necessary for the interaction with p6 to occur, strongly suggesting that the observed interaction is relevant in vivo. In addition, SPR studies revealed binding of full-length p6 to CypA, and that this binding was significantly stronger than for any of its N- or C-terminal peptides, indicating a super-stoichiometric interaction involving simultaneous binding of CypA to three identified binding domains of p6. Accordingly, we have discovered a novel virus-host cellular interaction where the mode of interaction involves both transient enzyme–substrate interactions and a more stable binding.

Biography

Sara M.Ø. Solbak completed her Ph.D in March 2012 at the Department of Chemistry and Centre for Pharmacy, University of Bergen. During her Ph.D she performed structural and functional studies of viral proteins, primarily by use of biophysical methods. By profession she is a pharmacist graduated from the Danish University of Pharmaceutical Sciences in 2006. Currently she is working as a Postdoc at Uppsala University, where she intend to adapt new relevant technology to continue research with the ultimate purpose to uncover unknown mechanisms at a sub-cellular level, relevant for the development of novel antiviral drugs. Her research results have so far been published in 5 scientific papers.

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1 (ii) : Regulation of Viral Infection

Session Chair
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University of Puerto Rico School of Medicine
Puerto Rico

Session Co-Chair
Pawel Ciborowski
University of Nebraska Medical Center, USA

Session Introduction

Title: **Cathepsin B: A cysteine protease associated with HIV-neurocognitive disorders and aging**
Loyda M. Meléndez, University of Puerto Rico School of Medicine, Puerto Rico

Title: **Early stages of interaction between HIV-1 and human cells**
Alisa Bukrinskaya, D.I.Ivanovsky Institute of Virology, Russia

Title: **TBA**
Bassel Sawaya, Temple University, USA

Title: **Curcumin inhibits rift valley fever virus replication in human cells**
Aarthi Narayanan, George Mason University, USA

Title: **Venezuelan Equine Encephalitis Virus interacts with host cellular micro-RNA processing machinery to facilitate viral replication**
Kylene Kehn-Hall, George Mason University, USA

Title: **Role of cystatin B/STAT-1 interaction in HIV replication**
Linda Rivera-Rivera, University of Puerto Rico School of Medicine, Puerto Rico

Title: **Expression of histone deacetylases and acetyltransferases in HIV-1 infected monocyte-derived macrophages**
Ariel Burns, University of Nebraska Medical Center, USA

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Cathepsin B: A cysteine protease associated with HIV-neurocognitive disorders and aging

Loyda M. Melendez
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University of Puerto Rico School of Medicine, Puerto Rico

Chronic human immunodeficiency virus type one (HIV-1) infection leads to a spectrum of neurological and cognitive abnormalities, known collectively as HIV-associated neurocognitive disorders (HAND). HAND manifest in milder forms during despite effective combination antiretroviral therapy.The pathogenesis of HAND is thought to involve HIV-infected perivascular macrophages and microglia, whose activation leads to the release of pro-inflammatory cytokines and other soluble factors toxic to neurons. One factor that may be involved in macrophage-mediated HIV neurotoxicity is cathepsin B, a member of the cysteine protease family. We recently demonstrated that cathepsin B is upregulated in HIV-1 infected monocyte-derived macrophages (MDM), and secreted in a form that has increased neurotoxic activity in vitro and no longer interacted with its normal inhibitors, cystatin B and cystatin C. We recently observed increased expression of cathepsin B and cystatin B in monocytes of women with cognitive impairment, increased cathepsin B activity in plasma, and decreased cathepsin B activity in the CSF. A study of post-mortem brain tissue suggests that cathepsin B is also upregulated in brains from patients with HAND. We will present recent data related to the mechanisms whereby cathepsin B is dysregulated after HIV infection and how this dysregulation causes neuronal cell damage. This work was supported in part by NIH grants R01-MH08316-01, RCMI-NCRR-G12RR03051, SNRP-NINDS-1-U54NS431.

Biography

Dr. Meléndez earned her Ph.D. degree in Experimental Pathology & Immunology cum laude from Emory University School of Medicine in 1990, where she also completed post-doctoral training in Hematology and Pediatric Infectious Diseases in collaboration with the Centers for Disease Control. Sheis a Professor in the Department of Microbiology, University of Puerto Rico School of Medicine. She directs the Viral Neuroimmunology Laboratory and the RCMI Translational Proteomics Center at the University of Puerto Rico Medical Sciences Campus (UPR-MSC). Dr. Meléndez is a co-inventor of one patent and has published over 30 manuscripts.

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Early stages of interaction between HIV-1 and human cells

Alisa Bukrinskaya
D.I.Ivanovsky Institute of Virology, Russia

Introduction: HIV-1 belongs to the lentivirus genus of Retroviridae family which is characterized by long incubation period and this period could be prolonged for 10 years and even longer. Meanwhile, it was recently shown that the early events during weeks or months after acute infection determine the future progression of infection. Important targets for HIV-1 is lymphoid tissue of the gut, and its cells are highly sensitive to the virus.

Results: Pulse-chase experiments showed that the amount of Gag precursor during chase is diminishing in cytosol fraction and increases in membrane fraction (the place of viral assembly). Meanwile,the matrix protein, the part of Gag precursor on Gag N terminus, appears in cytosol fraction as individual protein soon after Gag synthesis and its amount is increasing during chase.

Conclusion: These experiments show that matrix protein is cleaved from the part of Gag precursor molecules soon after Gag synthesis and could participate in assembly of virus particles as an individual protein. Meanwile, the other part of Gag precoursor is cleaved much later, during the release of virus particles from the cells. It could be suggested that matrix protein possesing two transportation signals - membranotropic and nucleophilic- could take part in the process of virus assembly and in intracellular transport of viral genomic RNA from nuclei to the membrane. Meanwhile, the part of matrix protein still as a part of Gag protein helps Gag to interact with host lipids due to its membranotropic signal and to start the virus assembly.

Biography

Alisa Bukrinskaya was born in Russia in 1928. In1948 Alisa Bukrinskaya entered the Moscow Medical Institute (now the Moscow Medical Academy). On the period of 1952 to 1955, Alisa Bukrinskaya was a postgraduate student at the Microbiology Department of the same Institute. From 1955 until now I work in the D.I. Ivanovsky Institute of Virology in Moscow as a scientific worker, then senior scientific worker, and then as the head of the laboratory and head of the Virology faculty in Medical University. Since 1986 Alisa Bukrinskaya was the Correspondent Member of Russian Academy of Medical Sciences.

In 1993 Alisa Bukrinskaya was invited to the Medical School of the University of Massachusetts as professor and worked there till 2005. In 2005 Alisa Bukrinskaya returned to Moscow to her laboratory in Ivanovsky Institute.

Alisa Bukrinskaya is author for 300 scientific papers published in the Russian, European and American Journals of 5 monographs and 2 text books in Virology. Since 1985 Alisa Bukrinskaya dedicated all her research to the problem of AIDS.

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Curcumin inhibits rift valley fever virus replication in human cells

Aarthi Narayanan
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Rift Valley fever virus (RVFV) is an arbovirus that is classified as a select agent, an emerging infectious virus and an agricultural pathogen. Understanding RVFV-host interactions is imperative to the design of novel therapeutics. Here, we report that an infection by the MP-12 strain of RVFV induces phosphorylation of the p65 component of the NFκB cascade. We demonstrate that phosphorylation of p65 (serine 536) involves phosphorylation of IκBα and occurs through the classical NFκB cascade. A unique low molecular weight complex of the IKK-β subunit can be observed in MP-12 infected cells that we have labeled as IKK-β2. The IKK-β2 complex retains kinase activity and phosphorylates an IκBα substrate. Inhibition of the IKK complex using inhibitors impairs viral replication thus alluding to the requirement of an active IKK complex to the viral life cycle. Curcumin, a well-documented NFκB inhibitor, strongly down regulates levels of extracellular infectious virus. Our data demonstrate that curcumin binds to and inhibits the kinase activity of the IKK-β2 complex in infected cells. Curcumin partially exerts its inhibitory influence on RVFV replication by interfering with IKK-β2 mediated phosphorylation of the viral protein NSs and by altering cell cycle of treated cells. Curcumin also demonstrates efficacy against ZH501, the fully virulent version of RVFV.

Curcumin treatment down regulates viral replication in the liver of infected animals. Our data point to the possibility that RVFV infection may result in the generation of novel versions of host components (such as IKK-β2) that by virtue of altered protein interaction and function, qualify as unique therapeutic targets.

Biography

Dr. Aarthi Narayanan received her Ph.D. in Genetics and Biochemistry from The University of Georgia at Athens. She did her post-doctoral research at the NIH where the focus of her research was on host-virus interactions. In 2007 she took up the position of Research Assistant Professor in the National Center for Biodefense and Infectious Diseases. Dr. Narayanan's research focus is to understand the interactions between the human host and viruses belonging to diverse families including Bunyaviruses, Alphaviruses, and HIV.

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Venezuelan Equine Encephalitis Virus interacts with host cellular micro-RNA processing machinery to facilitate viral replication

Kylene Kehn-Hall
National Center for Biodefense and Infectious Diseases, George Mason University, USA

Venezuelan Equine Encephalitis Virus (VEEV) causes disease in both equine and humans that exhibit overt encephalitis in a significant percentage of cases. Despite being recognized as an emerging threat, relatively little is known about the virulence mechanisms of VEEV. Interference with critical host-pathogen interactions is an important area that can be utilized for therapeutic development. Recent publications implicate miRNA interactions in the pathogenesis of various viral diseases. While many viruses down-regulate the miRNA pathway to facilitate replication, some viruses such as Hepatitis C Virus utilize specific miRNAs and the RNAi machinery to enhance their replication. Based on this, we have begun to study miRNA processing in connection with VEEV replication. Our data indicate that knockdown of miRNA processing machinery significantly hinders VEEV replication. Loss of Drosha and DGCR8 had the greatest effect on VEEV production, indicating the need for nuclear processed miRNAs. In addition, siRNA knockdown of Ago2 decreased viral replication, which was confirmed with Ago2 null cells. Ago2 null cells also demonstrated significantly reduced VEEV capsid production and VEEV-GFP expression driven from the subgenomic promoter. These results were confirmed with a small molecule inhibitor of miRNA processing (ACF). Bioinformatic analysis indicated that five cellular miRNAs have complementarity to the VEEV subgenomic promoter. Anti-miRNA inhibitors to these 5 cellular miRNAs demonstrated that inhibition of miR-3683 reduced VEEV replication. Taken together, these findings indicate that loss of RNAi machinery severely limits VEEV replication and that specific cellular miRNAs contribute to VEEV replication.

Biography

Dr. Kylene Kehn-Hall received her Ph.D. in Biochemistry and Molecular Biology from The George Washington University studying retroviral pathogenesis and breast cancer biology. She did her post-doctoral research at the FBI Counterterrorism and Forensic Science Research Lab, focusing on assay development. In 2007 she took a Research Scientist position at USAMRIID, where she worked towards the identification of novel therapeutics for hemorrhagic fever viruses. Currently, she is a tenure-track Assistant Professor at the National Center for Biodefense and Infectious Diseases at George Mason University. Dr. Kehn-Hall's research is focused on the development of therapeutics for emerging infectious diseases, specifically Bunyaviruses, Alphaviruses, and HIV.

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Role of cystatin B/STAT-1 interaction in HIV replication

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Cystatin B, a cysteine protease inhibitor, is correlated with increased HIV replication in monocyte-derived macrophages (MDM) as demonstrated by proteome and gene silencing with siRNA. Subsequently, the signaling mechanisms for cystatin B in HIV replication were related to its interaction with signal transducer and activator of transcription-1 (STAT-1). Whereas STAT-1 activates HIV-1 replication, high levels of tyrosine phosphorylated STAT-1 (STAT-1PY) has been associated with HIV-1 inhibitory activity. In MDM, high levels of cystatin B correlate with a reduction of STAT-1PY. The aim of this study is to elucidate the mechanism by which cystatin B contributes to HIV-1 replication by regulation of STAT-1PY. We used immunoprecipitation and LC-MS/MS to identify the proteins interacting with cystatin B in order to elucidate the relationship between cystatin B, STAT-1 phosphorylation and HIV replication in MDM. Cystatin B/STAT-1 interaction associated with the increase HIV replication in MDM is part of a multiprotein complex that includes other proteins. Cystatin B interacted with many different proteins in HIV infected cells including regulatory, glycolytic, redox, structural protein, transport, and signaling proteins. Among them we found the Major Vault Protein (MVP), coded by an IFN-γ responsive gene that interferes with IFN-γ activated JAK/STAT signals) and Pyruvate Kinase M2 isoform (PKM2) that has been reported as an up-regulated protein associated with the effects of cocaine on the enhancement of HIV-1 replication. In situ Proximity Ligation Assay (Duolink) was used to determine how the HIV infection alters the cystatin B/STAT-1 interaction and the STAT-1PY in uninfected and HIV-infected MDM at 12 days post-infection. Cystatin B and STAT-1 interact directly and this interaction increases after HIV infection. However, the small interaction between STAT-1/STAT1PY observed in control uninfected was decreased after HIV infection. To test that cystatin B inhibits the IFN-β response, we performed luciferase reporter gene assays in Vero cells, which are IFN deficient. We demonstrated that cystatin B inhibited the IFN-β response by preventing STAT-1 translocation to the nucleus and decreasing levels of STAT-1PY. Taking together, we concluded that the cystatin B/STAT-1 interaction inhibits the signaling pathway mediated by IFN-α by regulating STAT-1PY and/or it nuclear translocation. This action would prevent the expression of IFN-dependent antiviral genes and promote HIV replication mediated by LTR and IRF-1. This work was supported in part by NIH grants F32MH094210-01A1, R01-MH08316-01, RCMI-NCRR-G12RR03051, SNRP-NINDS-1-U54NS431.

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Expression of histone deacetylases and acetyltransferases in HIV-1 infected monocyte-derived macrophages

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[2] Department of Chemistry, Nicolaus Copernicus University, Poland

Approximately 15-30% of breast cancers over-express the HER2/neu receptor. Historically, over-expression of HER2/neu has been identified using IHC or FISH, both of which are invasive approaches requiring tissue samples. Patients with positive IHC/FISH tests can have the option of receiving HER2/neu targeted treatment. However, recent evidence has shown that some tumors identified as "negative" using these methods also respond to HER2/neu targeted therapy. Shedding of the extracellular domain (ECD) of the receptor into the circulation has led to the development of serum test of HER2 ECD as an additional approach to probe HER2/neu overexpression. Yet more than 10 years after the first serum HER2 ECD test was approved by the FDA, serum HER2 testing has yet to be widely used in clinical practice. We have developed an assay to reduce serum interference that commonly occurs in practice and discourages the use of ELISA type of assay. Using this refined assay, we are able to accurately correlate serum HER2 ECD levels with tissue HER2/neu status. With this test, we can monitor HER2 ECD as a biomarker over the course of disease progression and treatment. It will also help screen patients who have no available tissue samples for HER2/neu targeted therapies.

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Keynote Lecture

Pooja Jain

Drexel University College of Medicine, USA

The tug-of-war between dendritic cells and chronic viral infections

Biography

Pooja Jain is an Associate Professor in the Department of Microbiology and Immunology, Drexel University College of Medicine (DUCOM), Philadelphia. She also holds a joint faculty appointment in the Institute for Molecular Medicine and Infectious Diseases as well as in the Drexel Institute for Biotechnology and Virology Research within DUCOM. She has received a PhD in Microbiology from the Central Drug Research Institute, India in 2001 and completed her postdoctoral training from the Texas Tech University Health Science Center as well as from the DUCOM. She has published 40 peer-reviewed articles and is serving as an Editorial board member/reviewer for 5 reputed journals.

2 (i) : Viral Breakout, Transmission and Prevention

Session Chair

Andreas Nitsche
Robert Koch Institute, Germany

Session Introduction

Title: **Old viruses and new techniques: Investigations of host-range mechanisms of Cowpox viruses**
Andreas Nitsche, Robert Koch Institute, Germany

Title: **The outbreak time distribution feature of the acute infectious disease mainly in asymptomatic infection and the trend prediction skill during the outbreak**
Jiatong Zhuo, Center for Disease Control and Prevention of Guangxi Zhuang Autonomous Region, China

Title: **Adherence to antiretroviral therapy and its challenges in people living with HIV and accessing healthcare in a federal medical centre in Nigeria**
Grace Pennap, Nasarawa State University, Nigeria

Title: **High rate of transfusion-transmitted virus (TTV) infection in the patients with leukemia**
Mohammad Javad Kazemi, Islamic Azad University, Iran

Title: **Modeling of dengue fever temporal variations in central Thailand**
Siriwan Wongkoon, Walailak University, Thailand

Title: **A 20-years retrospective cohort study of HIV/AIDS situation among hill tribe vulnerable population, Thailand**
Tawatchai Apidechkul, Mae Fah Luang University, Thailand

Title: **Hepatitis A virus replication in salivary glands: A possible source of HAV transmission**
Luciane Almeida Amado Leon, Instituto Oswaldo Cruz- FIOCRUZ, Brazil

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Old viruses and new techniques: Investigations of host-range mechanisms of Cowpox viruses

Andreas Nitsche and Andreas Kurth
Robert Koch Institute, Germany

Poxviruses comprise a fascinating virus family. Not only is the most prominent poxvirus, the orthopoxvirus Variola virus, the first virus for which an effective vaccination has been developed, but also is the disease caused by Variola virus, smallpox, still the only infectious disease that could be eradicated by systematic worldwide vaccination. After declaration of the successful eradication of smallpox by WHO in 1980, vaccinations were halted to prevent vaccination-related severe adverse effects. Since then, the number of zoonotic infections with orthopoxviruses has been increasing. In South America infections of animals and humans with Vaccinia virus-related orthopoxviruses are being observed. Monkeypox viruses, causing overall mortality rates of 10%, are endemic in Africa, and in Europe we are witnessing increasing numbers of animal and human infections with Cowpox viruses.

In contrast to Variola virus, which had been exclusively infecting humans, the host range of the zoonotic poxviruses is generally higher, with Cowpox viruses representing a species that has been shown to infect a large variety of vertebrate species. The mechanisms that explain the ability of certain poxviruses to cross species barriers are not understood at all.

In this talk an overview of the peculiarities of Cowpox virus and the transcriptome- and proteome-based approaches to elucidate the pathogenic potential of cowpox viruses is presented.

Biography

Andreas Nitsche is a virologist who received his PhD from the Free University of Berlin in 2001. Today he is head of the Centre for Biological Security 1 "Highly Pathogenic Viruses" of the Robert Koch Institute, head of the German Reference Laboratory for Poxviruses and head of the RKI Core Facility for DNA Sequencing. He has published more than 80 papers in renowned journals with a focus on molecular diagnostics and poxviruses.

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The outbreak time distribution feature of the acute infectious disease mainly in asymptomatic infection and the trend prediction skill during the outbreak

Jiatong Zhuo
Center for Disease Control and Prevention of Guangxi Zhuang Autonomous Region, China

For the acute infectious disease mainly in symptomatic infection has been described in the text book quite often. However, for those acute infectious disease mainly in asymptomatic infectious was barely presented. Collection of the outbreaks of the polio outbreak in Laibin County, Guangxi, China 1991 the Japanese encephalitis outbreak in Bobai County, Guangxi, China 1999 polio outbreak in Tajikistan in 2010. All these three out breaks experienced 5 generation incubation before the outbreak peak appeared. For the Japanese Encephalitis, the 5 incubations finished within 34, about 7 days for each, For the polio, the 5 incubations finished within 90, about 18 days for each. As acute infectious disease, there is very much apparent different time distribution feature between the those mainly in symptomatic and that mainly in asymptomatic. The skill to predict the trend in the type mainly in asymptomatic infectious disease should be much alerted , each case have to be treat as outbreak and the containment measures have to be taken to prevent outbreak peak occurred. If the containment not in time, the outbreak peak will appeared finally. This is why the WHO's requirement of the each case of polio has to contain as outbreak. Take each case seriously and contain it timely to prevent outbreak peak is the key skill to the outbreak trend prediction and the outbreak control as well.

Biography

Jiatong Zhuo is a professor and deputy director of Guangxi Autonomous Region Center for Disease Control and Prevention. He has spent most of his scientific carrier in the children vaccine preventable disease. Under his effort, Guangxi Zhuang Autonomous Region reach the goal of polio free in 2000. He created this new strategy to select high risk county with historic surveillance data and carrying mass campaign in the sub-tropic area to get the measles incidence 0.5/million in since 2010 after his finished his public health policy/management study in Emory in 1998 and make the measles elimination in the forefront of China Dr. Zhuo also was a founder of communicable disease and public health even emergency information delivery and administrative management networks system in Guangxi in 2001 and made the SARS very much timely controlled in the spring of 2003. Dr. Zhuo is the Chinese State Council Special Stiphend Awared scientist and the member of Technical Advise Committee on Immunization of Health Ministry of China.

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Adherence to antiretroviral therapy and its challenges in people living with HIV and accessing healthcare in a federal medical centre in Nigeria

Grace Pennap
Microbiology Unit, Nasarawa State University, Nigeria

Adherence to antiretroviral therapy (ART) among people living with HIV is pivotal in reducing viral transmission, preventing the emergence of drug resistant viral strains and improving the life expectancy of these people. A base line survey of the level of adherence to ART and its challenges was carried out among adults accessing this healthcare service at a Federal Medical Centre in Nigeria. Two hundred and fifty patients were recruited for this facility based cross-sectional study. Patients ≥ 18 years who consented to participate were interviewed using a structured questionnaire. The level of adherence in this cohort was 62.8%. It was found to be higher in patients whose spouses knew their HIV status (47.9%), were living with their families (61.7%), and among those that used an alarm to remind themselves to take their medication (50.5%). However, common reasons for non-adherence were found to be: forgetfulness (51.5%), avoiding the drug side effect (14.5%), living far away from the Medical Centre (8.1%) and inability to afford the cost of transportation to the Medical Centre (6.5%). Patient's educational level, marital status and occupation were found to be significantly associated with adherence to antiretroviral therapy in this cohort ($p \leq 0.05$). Adherence was poor in this study population. The decentralization of ART services to primary healthcare facilities and the reintensification of patients' education and counseling is advocated.

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High rate of transfusion-transmitted virus (TTV) infection in the patients with leukemia

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[2]Shiraz Transplant Research Center-Shiraz University of Medical Sciences, Iran

Extrahepatic disorders of TT virus infection may have role in introducing and complicating the clinical outcomes in patients with leukemia. Therefore, in this study, the prevalence of TT virus infection was evaluated in the patients with leukemia. In a cross sectional study, EDTA-treated blood samples were collected from 66 patients who clinically, pathologically, and laboratory confirmed with hematological malignancies. Then the plasma and Buffy coats were extracted from collected blood samples and stored in -70°C till protocols preformed. The DNA of TT virus was diagnosed in samples of studied patients by an in-house semi-nested PCR protocol. Also some possible risk factors of leukemia and TT viral infection including: age, gender, marriage, education, occupation, and also history of smoking and transfusion were statistically analyzed for all studied patients with leukemia.The genome of TT virus has been detected in 42% of plasma samples of patients with leukemia. Also the TT virus DNA has been detected in 64.1% of Buffy coats of patients with leukemia. On the other hand the HBsAg and HCVAb were found in 24% and 2% of plasma samples of the patients with leukemia, respectively. Diagnosis of the high rate of TT virus infection in both plasma and especially buffy coat samples of the patients with leukemia re-emphasis on the importance role of TT virus pathogenesis in complication of the outcomes of hematological malignancies.

Biography

Mohammad Javad Kazemi has completed his Ph.D at the age of 31 years from Islamic azad university-north Tehran branch. He is faculty member of Islamic azad university ashkezar- branch,ashkezar, Iran

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Modeling of dengue fever temporal variations in central Thailand

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The incidence and distribution of dengue related illness have grown dramatically in recent decades. Monitoring and predicting dengue incidence can facilitate early warning and disease control and prevention. This study aimed at developing a predicting model on the incidence rate of dengue fever in central of Thailand using time series analysis. Data on monthly-notified cases of dengue fever over the period 1981-2011 were collected from the Bureau of Epidemiology, Department of Disease Control, Ministry of Public Health. Seasonal Autoregressive Integrated Moving Average (SARIMA) model was performed using data on monthly incidence rate of dengue fever from 1981 to 2009, and validated using the monthly rate collected for the period 2010 to 2011. The SARIMA(1,0,1)(1,1,1)12 model has been found as the most suitable model with the least Root Mean Square Error (RMSE) of 4.114, Mean Absolute Percent Error (MAPE) of 20.175 and Bayesian information criterion (BIC) of 2.915. The residuals in the model appeared to fluctuate randomly around zero, with no obvious trend in variation, as the predicted incidence values increase. The model demonstrated goodness-of-fit with R2 of 92.90%. Our findings indicate that SARIMA model is useful tool for monitoring dengue incidence in central, Thailand. Furthermore, this model can be applied to surveillance data for predicting trends in dengue incidence in Thailand.

Biography

Siriwan Wongkoon has completed her Ph.D in Computational Science at Walailak Univerisy, Thailand. She has done on "Thailand GLOBE Mosquito Protocol" for Thai schools with Assoc. Prof. Dr. Mullica Jaroensutasinee and Assoc. Prof. Dr. Krisanadej Jaroensutasinee, the GLOBE Thailand Program, the Institute for the Promotion of Teaching Science and Technology (IPST) and Center of Excellence for Ecoinformatics, NECTEC/Walailak University. She has published more than 28 papers in reputed local and international journals.

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A 20-years retrospective cohort study of HIV/AIDS situation among hill tribe vulnerable population, Thailand

Tawatchai Apidechkul
School of Health Science, Mae Fah Luang University, Thailand

Introduction: Thailand has been reported as the highest HIV/AIDS epidemic area in the world last few years. Most of HIV/AIDS had been reported from the north of Thailand. Northern Thailand is the favorite living places of hill tribe people who migrated from the south of China through Myanmar last 150 years ago. Nowadays almost 600,000 people were living in these areas with differences of culture and life styles. The objective aimed to investigate the situation of HIV/AIDS among hill tribe marginalized and vulnerable population.

Materials and Methods: The retrospective cohort study was conducted. The systematic data extraction from the medical records in 16 hospitals in northern Thailand during 1990-2010 was performed. The six main hill tribe people: Akha, Lau, Karen, Yao, Kmong, and Lisu were the target population. Chi square test was analyzed.

Results: Totally 3,130 cases were recruited into the study. 54.6% were male, the first case had been reported in 1990, and the highest incident case had been reported in the year 2004 with 461 cases followed by 2005 (343 cases), and 2006(302 cases) respectively. The highest cumulative case had been reported from Mae Fah Luang Hospital (25.8%), followed by Mae Suai hospital (18.8%). 46.0%were Akha, 19.7%were Lahu, and 9.5% were Yao. 38.8%were 31-40 years old, followed by 21-30 years old(33.6%), and 41-50 years old (13.4%). 44.4%were agriculture, 32.0% were employee. 91.6% were infected by sexual intercourse, 5.7% were mother to Child. 24.0% were receiving ARV, 30.7% were receiving OI treatment, and 9.5% were tested CD4 level. Male had higher of survival rate than female (p-value>0.001), and male were younger than female at the age of infection (p-value>0.001). There was statistically significant difference of mode of infection by tribe (p-value>0.001).

Conclusion: Specific health education programs and empower them for using condom are needed to setting up for HIV/AIDS prevention and control among hill tribe people in Thailand.

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Hepatitis A virus replication in salivary glands: A possible source of HAV transmission

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Instituto Oswaldo Cruz- FIOCRUZ, Brazil

Hepatitis A is one of the most reported viral hepatitis worldwide and is recognized as an important public health disease, especially in developing countries. Hepatitis A virus (HAV) causes a self-limited infection and is transmitted through the faecal-oral route. However, there are still unsolved questions concerning its transmission and its replication in extrahepatic tissues. To further clarify some of these aspects, an experimental infection study in cynomolgus monkeys was conducted. It was demonstrated the viral antigen and an active replication of HAV in salivary glands of these animals, through detection of HAV intermediate replicative (minus strand RNA). These results established one of the mechanisms of the HAV presence in saliva and also suggest that the saliva is a potential source of HAV transmission. Corroborating these findings, our previous studies have demonstrated a higher frequency of HAV in saliva than in corresponding serum sample of children enrolled in hepatitis A outbreaks. The HAV persistency and viral load in saliva was similar of matched serum. Interestingly, in some cases viral genotypes in saliva specimens were found to be different from the genotypes detected in the corresponding serum specimens. This distribution of different genotypes of HAV in blood and saliva also reinforces the occurrence of extrahepatic independent replication. In summary, if HAV-RNA was consistently detected in saliva, and the anti-HAV titer is correlated with those of blood, the collection of saliva could also provide a simple, cheap and non-invasive means of detecting and monitoring hepatitis A.

Biography

Dr. Luciane Amado is a researcher at Laboratory of Technological Development in Virology at the Fiocruz and has been involved in viral hepatitis studies for over ten years. She has completed her Ph.D and postdoctoral research in Virology the Department of Virology from Instituto Oswaldo Cruz-Fiocruz, Brazil. Her current position is a researcher of the Department of Virology from Fiocruz, Brazil and professor of Virology in the Department of Biotechnology from Rio de Janeiro State University, Brazil. She has published more than 30 articles in reputed journals and serving as a reviewer of repute scientific journals.

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2 (ii) : Viral Fundamental Mechanisms

Session Chair

Vicente Planelles
University of Utah, USA

Session Introduction

Title: **HIV-1 Latency in Memory T cells**
Vincente Planelles, University of Utah, USA

Title: **Vpu is responsible for HIV-1 evasion of natural killer cells**
Edward Barker, Rush University Medical Center, USA

Title: **RelA participates in the regulation of the coupling between HCV RNA replication and HCV translation in huh7.5.1 cells**
Lumin Zhang, National Institutes of Health, USA

Title: **Role of SNARE proteins in HIV-1 assembly and release**
Anjali Joshi, Texas Tech University Health Sciences Center, USA

Title: **Discovery and analysis of cellular elongation factors that are critical for HIV-1 reverse transcription**
David Harrich, Queensland Institute of Medical Research, Australia

Title: **SAMHD1 enzymatic activity toward non-canonical nucleotides**
Sarah Amie, Rochester University, USA

Title: **P-body components LSM1, GW182, DDX3, DDX6 and XRN1 are recruited to WNV replication sites and positively regulate viral replication**
Harendra S. Chahar, Texas Tech University Health Sciences Center, USA

Title: **Differential expression/stability of core protein during HCV infection and its effect on viral life cycle**
Muhammad Sohail Afzal, National University of Science & Technology, Pakistan

Title: **Influence of pathogen-associated molecular patterns on HIV-1 latency**
Alberto Bosque, University of Utah School of Medicine, USA

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HIV-1 Latency in Memory T cells

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University of Utah, USA

Background: Central memory, CD4+ T lymphocytes (TCM) harbor the majority of latent HIV-1 proviruses in vivo. We have developed a latency assay based on cultured TCM cells. We have so far identified two signaling pathways that can reactivate latent viruses in cultured TCM: antigenic stimulation and incubation with IL-2+IL-7. Antigenic stimulation reactivates virtually all latently infected cells, and successfully depletes the reservoir. Despite the potency of antigenic stimulation, anti-CD3/CD28 treatment has been shown to exert deleterious effects in the host. In contrast, IL-2+IL-7 incubation reactivates one tenth of the latently infected population, while inducing homeostatic cell proliferation. Consequently, cells are able to proliferate in response to IL-2+IL-7 stimulation, in the absence of viral reactivation, thus propagating latent proviruses through mitosis. Thus, other signaling pathways need to be identified. This work describes a third pathway that we have recently identified, in which massive viral reactivation is achieved with minimal T cell activation.

Methods: We have used a high-throughput variation of our published assay of viral latency in TCM, and identified novel candidate compounds that reactivate latent HIV-1. The biological properties of candidate compounds from the screen were further investigated in order to examine their ability to induce activation markers (CD25 and CD69) and proliferation. This was done in parallel with antigenic stimulation and homeostatic proliferation inducers as a comparison.

Results: We have identified a compound ("C7") which, when incubated with latently infected TCM cells at nanomolar concentrations, displays viral reactivation ability that is about 80% of that with anti-CD3/CD28. The activation profile of the C7-treated cells was indistinguishable from that of untreated cells as evidenced by a lack of increase in the expression of CD25 and CD69. C7 induced proliferation although at much lower levels than anti-CD3/CD28 treatment did.

Conclusions: We demonstrate the existence of compounds that can reactivate latent HIV in TCM cells with comparable efficiency to antigenic stimulation, but with very limited or no ability to induce the expression of activation markers. These results demonstrate that signaling pathways exist, which can be specifically lead to activation of latent proviruses in primary cells. Key signaling elements controlling these pathways should be considered as novel targets.

Biography

Vicente Planelles obtained his Ph.D. from the University of California at Davis in 1991 and then conducted postdoctoral studies at UCLA until 1995. Between 1996 and 2002 he was Assistant Professor at the University of Rochester. In 2002 he became Associate Professor at the University of Utah School of Medicine, and in 2008 he became Professor of Microbiology and Immunology. He has published more thatn 80 papers and reviews on many aspects of HIV-1 and related lentiviruses.

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Vpu is responsible for HIV-1 evasion of natural killer cells

Edward Barker
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Natural killer (NK) cells are recognized as being crucial in the defense against viruses. NK cells interaction with virus-infected cells ultimately leads to the death of the virus-infected cells. However, HIV-infected cells are refractory to lysis by NK cells. We have found that resistance to lysis is not due to the failure of HIV to activate NK cells but rather to HIV's ability to prevent NK cell release of its lytic granules. These outcomes are the result of the combined action of viral proteins that both lead to NK activation but ultimately act to suppress the lytic function of NK cells. The key findings from our laboratories, demonstrate that: 1) HIV-1 Nef down modulates HLA-A and –B and 2) HIV-1 Vpr induces ligands to NK cell activation receptor NKG2D. Both of these events lead to NK cell activation. However, HIV-1 Vpu counters NK cells' ability to degranulate by down modulation of NTB-A a homotypic ligand to the NK cell coactivation receptor, NTB-A. NTB-A on the infected cells is critical for eliciting NK cell cytolytic response because degranulation requires both simultaneous engagement of activation with coactivation receptors. Vpu acts to prevent NTB-A surface expression by retention of NTB-A within the trans-Golgi network of the infected cell. Vpu actions on NTB-A are independent of Vpu's activity against CD4. Vpu acts on NTB-A in a similar fashion as Vpu mediated suppression of the host cell innate factor tetherin/BST-2. The ultimate goal will be to use the knowledge gained from studies on Vpu-NTB-A interactions to devise novel therapeutic approaches aimed at rendering HIV-infected cells sensitive to NK cell killing.

Biography

Ed Barker has completed his Ph.D. at the University of Illinois at Chicago and postdoctoral studies from University of California at San Francisco. Currently, he is an Associate Professor in the Department of Immunology and Microbiology at Rush University Medical Center in Chicago. Ed is working on his sixth year of an NIH grant to study how HIV evades natural killer cells.

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RelA participates in the regulation of the coupling between HCV RNA replication and HCV translation in huh7.5.1 cells

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Hepatitis C virus, HCV, is a positive-strand RNA. After released into cytoplasm, HCV RNA severs as a template for the viral translation and transcription. Therefore, the coupling between the viral translation and transcription plays an important role in the modulation of HCV replication. In order to escape the systemic surveillance, HCV develops a complexity strategy to coordinate these two processes. Of these, host factors have been implicated in involvement in the regulation of this adjustment. Although Huh7.5.1 cells is permissive for the study of HCV1a replication in culture system, a low viral production still restricts explore in the understanding of HCV living cycle. Recent studies suggest that a sustained NF-kB activation is a major factor for the impediment of viral replication. To further clarify the role of NF-kB in the HCV replication, we used shRNA to inhibit the activation of RelA. Intriguingly, we found that RelA silencing remarkably suppressed HCV IRES mediated translation. The expressions of viral proteins were also inhibited. Inversely, RelA silencing improved the production of HCV. The further investigation showed that this enhancement was mediated by the increment of HCV RNA replication through the inhibition of interferon beta response. In summary, our results suggest that RelA may participate in the coupling of HCV1a RNA translation and HCV1a RNA transcription, and so regulate HCV1a replication.

Biography

Lumin Zhang has completed his Ph.D at the age of 32 years from Nagoya University. Now, he is a visiting fellow in National Institutes of Health.

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Role of SNARE proteins in HIV-1 assembly and release

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Retrovirus assembly is a complex process that requires the orchestrated participation of viral components and host-cell factors. The concerted movement of different viral proteins to specific sites in the plasma membrane allows for virus particle assembly and ultimately budding and maturation of infectious virions. The soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) proteins constitute the minimal machinery that catalyzes the fusion of intracellular vesicles with the plasma membrane, thus regulating protein trafficking. Using siRNA and dominant negative approaches we demonstrate here that generalized disruption of the host SNARE machinery results in a significant reduction in human immunodeficiency virus type 1 (HIV-1) and equine infectious anemia virus particle production. Further analysis of the mechanism involved revealed a defect at the level of HIV-1 Gag localization to the plasma membrane. Our findings demonstrate for the first time a role of SNARE proteins in HIV-1 assembly and release, likely by affecting cellular trafficking pathways required for Gag transport and association with the plasma membrane.

Biography

Dr. Anjali Joshi is a Research Instructor in the Department of Biomedical Science at Texas Tech University Health Sciences Center. She pursued her PhD in Feline Immunodeficiency virus from North Carolina State University, Raleigh, USA. Immediately after completing her PhD, she received four years of post doctoral training in the Lab of Dr. Eric Freed, Head of the viral assembly section at the National Cancer Institute, Frederick. At NCI, she worked on various aspects of retrovirus assembly including the role of cellular factors in this pathway, role of viral domains in determining the site and process of assembly and several basic aspects of cellular trafficking pathways and retrovirus biology.

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Discovery and analysis of cellular elongation factors that are critical for HIV-1 reverse transcription

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Using protein fractionation combined with an in vitro endogenous reverse transcription assay and mass spectrometry, we identified a cellular protein complex composed of subunits of eukaryotic elongation factor 1 (eEF1) that was able to stimulate late steps of HIV-1 reverse transcription in vitro. Further studies showed that the eEF1A and eEF1G subunits of eEF1 are important components of the HIV-1 reverse transcription complex (RTC) as evidenced by: (a) co-localization of eEF1G and eEF1A with reverse transcriptase (RT) in HIV-1 infected cells; (b) co-purification eEF1 subunits with RTC isolated from infected cells; (c) markedly reduced HIV-1 reverse transcription when eEF1A or eEF1G were down regulated by siRNA. Protein- protein interaction between recombinant RT and eEF1 complex subunits isolated from HEK293T cells was investigated by surface plasmon resonance. Both RT51 and 66 subunits were able to bind to eEF1A with a Kd of 5.4 nM and 1.9 nM, but not to eEF1B, eEF1D or eEF1G. Recent experiments indicate that eEF1A is a major RT binding protein and most likely a mediator of eEF1A complex and HIV RTC interaction as knock-down of eEF1A expression in cells by siRNA treatment resulted in significantly reduced binding of HIV RT to proteins in the cell lysate. Binding to RT could be restored by over-expression of exogenous eEF1A by plasmid transfection. The interaction of RT and eEF1A are refined by mapping the binding domains within the two proteins. The role of RT-eEF1A interaction in HIV reverse transcription represents a potential new target for anti-HIV drug screening.

Biography

David is an Australian Research Council Future Fellow and Group Leader of Molecular Virology at the Queensland Institute of Medical Research. After completing a PhD at UCLA in 1994 in Experimental Pathology of HIV-1 with Prof. Richard Gaynor, he undertook is post-doctoral studies at the University of Texas Southwestern Medical Centre under an NIH Fellowship in immunology and virology. He moved to Australia in 1997 to lead a lab in the National Centre of HIV Virology Research investigating HIV reverse transcription. He has published 50 peer-reviewed paper and serves on the Editorial board of several leading journals including PLoSONE.

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SAMHD1 enzymatic activity toward non-canonical nucleotides

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Macrophages are able to maintain a long-lived viral reservoir of HIV. Although HIV-1 is able to infect macrophages, it does so at a much slower rate compared to $CD4^+$ T cells. This is due to their low dNTP pools, which are needed as substrates for HIV reverse transcriptase (RT) to synthesize viral DNA. It has recently been shown that the low dNTP levels found in macrophages are not just the result of maintaining a static lifecycle, but the result of a newly discovered myeloid specific host restriction factor, SAMHD1, which is able to hydrolyze dNTPs into nucleosides. Another mechanism of host restriction in macrophages is the high level of dUTP (58 fold higher than TTP), which is frequently misincorporated by RT; however HIV-1 has evolved to package host nuclear uracil DNA glycosylase (UNG2) to remove dUMPs from its genome. This mechanism was not adopted by HIV-2. Instead, HIV-2 has a separate anti-restriction capability, which allows it to replicate efficiently in macrophages. HIV-2 evolved the accessory protein Vpx to direct proteasomal degradation of SAMHD1 thereby increasing dNTP pools. We hypothesize that degradation of SAMHD1 will reduce the concentration disparity of dUTP and TTP during viral replication. Therefore, we predict that SAMHD1, which is only present in macrophages infected by HIV-1, is able to selectively decrease the levels of canonical dNTPs and not non-canonical dUTP resulting in the virus to evolve separate antiviral activities from HIV-2 to counteract uracilation of its' genome.

Biography

Sarah Amie is in the process of completing her PhD at the University of Rochester. She is in the Microbiology Immunology department under the advisement of Dr. Baek Kim. Her research has focused on the incorporation of ribonucleotides by HIV-1 reverse transcriptase in macrophages and the implications of their persistence in viral DNA. She also has been trying to elucidate the activity of SAMHD1, an HIV-1 host restriction factor in myeloid cells, on non-canonical nucleotides.

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P-body components LSM1, GW182, DDX3, DDX6 and XRN1 are recruited to WNV replication sites and positively regulate viral replication

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In mammalian cells, proteins involved in mRNA silencing and degradation localize to discrete cytoplasmic foci called processing or P-bodies. These include Sm-like protein LSM1, GW182, 5'-3' exonuclease XRN1, dead box RNA helicase DDX3 etc. West Nile Virus (WNV) infection causes depletion of P-bodies. However, how this P-body depletion occurs and what happens to the P body proteins is not known. WNV infected HeLa cells were analyzed for P-bodies by immunostaining, for protein levels by western blotting. Transient knockdown of P body components was achieved by specific siRNA transfection, WNV replication was measured by FACS analysis and qRTPCR. In WNV infected cells 36 hPI, complete depletion of LSM1, GW182, DDX3 and XRN1 P-bodies was observed but levels of these proteins remained equal in uninfected and infected cells. On the other hand, we found that many P-body components including LSM1, GW182, DDX3, DDX6 and XRN1, but not others like DCP1 and EDC4 are recruited to the viral replication sites as evidenced by their colocalization at perinuclear region with viral NS3. Kinetic studies suggest that the component proteins are first released from P-bodies in response to WNV infection within 12 h post infection, followed by recruitment to the viral replication sites by 24-36 h post infection. These data suggests that in response to WNV infection p body components relocalize to WNV replication complexes and this in-turn might cause depletion of P-bodies. Silencing of the recruited proteins individually with siRNA interfered with viral replication to varying extents suggesting their collective requirement for efficient viral replication. Thus, the P-body proteins might provide novel drug targets for inhibiting viral infection.

Biography

Harendra S Chahar completed his Ph.D from All India Institute of Medical Sciences, New Delhi India-2011. He works in the Center of Excellence in Infectious Disease Research at Texas Tech University, El Paso, Texas in Dr. Manjunath Swamy's group. Their primary area of interest is to understand RNAi mechanism in special reference to flaviviruses and develop novel therapeutics.

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Differential expression/stability of core protein during HCV infection and its effect on viral life cycle

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HCV core protein plays a critical role in viral assembly as a structural component as well as a regulator of assembly site formation. Core recruits replication complexes and envelope glycoproteins to the vicinity of cytosolic lipid droplets.

In current study, we observed a differential core protein expression/stability during infectious HCV life cycle. We analyzed the expression kinetics of HCV proteins in a single viral cycle assay using a CD81-negative Huh-7 derivative cell line. It was observed that most viral proteins accumulated with a half maximal value at 25 to 27 hours post-electroporation in this system. In contrast, the half maximal accumulation of core was reached at 33 hours post-electroporation, indicating a 6-to-8-hour delay in core expression, compared to other HCV proteins. The delay in core expression was confirmed in infected Huh-7 cells using an immunofluorescence-based assay.

Our results showed an increase in core expression during late step of the viral life cycle. core was found to turn over with a half-life of approximately 90 minutes when measured at early time points of HCV infection, or in heterologous expression systems. Strikingly, there was a ten-fold increase of core half-life over the course of infection, whereas other viral proteins half-lives were not increased by more than two times.

As core protein stabilized itself during viral life cycle, to check the effect of this differential core turnover during viral life cycle, core protein was expressed at different concentration and viral replication was quantified. Core protein down regulates the viral replication in concentration/stability dependent manner. As core recruits replication complex on lipid droplets (LDs) for viral assembly, the effect of differential core stability on viral assembly was observed by expressing core in stable cell line expressing sub-genomic replicon for different time points. Our results showed that core protein at later time points after stabilized by itself, more efficiently recruits replication complex to LDs, most probable viral assembly sites.

Altogether, these results indicate that core is an unstable protein, which is stabilized when expressed at higher expression levels. In the course of HCV infection, this stabilization down regulate the HCV replication, and recruits HCV replication complex more efficiently on LDs, and shift the viral life cycle from replication to assembly. This delayed core expression may constitute a mechanism participating in the regulation of the HCV life cycle.

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Influence of pathogen-associated molecular patterns on HIV-1 latency

Alberto Bosque
University of Utah School of Medicine, USA

Pathogen-associated molecular patterns (PAMPs) are molecules present on microbes and are recognized by cells of the innate immune system to activate innate immune responses and protect the host form infections. PAMPS are recognized by Toll-like receptors (TLRs) and other pattern recognition receptors (PRRs). CD4 T cells have been shown to express TLRs, however the effects of PAMP recognition and its downstream signaling on the HIV-1 latent reservoir are largely unknown. We have developed a system whereby naive cells from the periphery of healthy donors are induced to undergo normal development ex vivo in the presence of select cytokine cocktails or antigenic stimulation through CD3/CD28. These cells are infected while in the activated state, and return to quiescence as central memory cells (TCM). Infection of these ex-vivo generated memory cells leads to latency with a high frequency (about 90% of infections) and leads to a polyclonal population of integrated viruses. Using this paradigm, we have explored the influence of PAMPs on HIV-1 latency in cultured TCM.

We have found that the triacetylated lipopeptide Pam3CSK4, a TLR2 agonist, can reactivate latently infected cultured TCM cells. Interestingly, other tested TLR2 agonists, such as the diacetylated lipopeptides Pam2CSK4 or FSL-1; the yeast cell wall glucan Zymosan or the heat-killed Listeria monocytogenes failed to induce reactivation of latent viruses. Moreover, LPS and Poly(I:C)LMW/LyoVecTM, TLR4 and RIG-I/MDA-5 ligands, respectively, reactivated latent HIV only in a subset of human blood donors. The mechanisms behind these differences are under investigation.

Biography

Dr. Alberto Bosque is a Research Assistant Professor in the Department of Pathology at the University of Utah. He completed his Ph.D in Human Immunology at the University of Zaragoza, Spain. After completing his Ph.D, he undertook his postdoctoral training at University of Utah School of Medicine. In 2011, he became Research Assistant Professor at the Division of Microbiology and Immunology at the University of Utah. He has published more than 20 papers in reputed international journals, in the areas of apoptosis, autoimmunity and HIV-1 latency.

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Keynote Lecture

Fatah Kashanchi

George Mason University, USA

Discovery of novel complexes in infected cells

Biography

Dr. Kashanchi received his Ph.D. in 1990 in Microbiology with emphasis on HIV gene expression. He then moved to Washington, DC for his post doctoral and Research Associate fellowship at National cancer Institute, National Institutes of Health from 1991-1998. He was Tenured at the George Washington University medical school as a full Professor in 2004. He moved to GMU as director of research in 2010.

Research interests include Human retroviruses, biodefense viral agents, Cell cycle, host-pathogen interactions, small molecule and peptide inhibitors against transcription machinery, RNAi machinery and its components, proteomics and metabolomics, and humanized mouse models.

The current research in the Kashanchi Lab is focused on defining transcriptional and chromatin mediated regulation of HIV and HTLV-1 infected cells. Their studies have resulted in novel concepts regarding promoter-bound proteins that regulate all events of mRNA biogenesis (including capping, elongation, termination, poly A addition, splicing), nuclear-cytoplasmic transport, and activation of nonsense mRNA degradation. Among biothreat agents, the Kashanchi lab is interested in Rift Valley fever virus (RVFV) and Venuzueln Equine Encepalitis virus (VEEV) replication in vitro and in vivo and defining crucial host-pathogen interactions that are imperative to pathogenesis.

3 (i) : Model Systems and Population to Studies Viral Infectivity

Session Chair
Sita Awasthi
University of Pennsylvania, USA

Session Introduction

Title: **Humanized mouse model to study viral infections and evaluate antivirals**
Sita Awasthi, University of Pennsylvania, USA

Title: **Molecular signatures of Venezuelan equine encephalitis in mice**
Anuj Sharma, Uniformed Services University of the Health Sciences, USA

Title: **Transient transfection of BeWo cells with a truncated human endogenous retrovirus ERV3 env induces β-hCG**
Neal S. Rote, Case Western Reserve University School of Medicine, USA

Title: **Infectivity of avian Hepatitis E virus isolated from China**
Qin Zhao, Northwest A & F University, China

Title: **Human Mammary Tumor Virus (HMTV) and cancer**
Stella Melana, Mount Sinai School of Medicine, USA

Title: **Prognostic and developed scenarios of pandemic flu in Georgia**
Sophio Beridze, National Centre for Disease Control and Public Health of Georgia, Georgia

Title: **Knowledge, attitude, beliefs and practices about HBV vaccination and universal precautions in healthcare workers of a tertiary care centre in India**
Varsha Singhal, AIIMS, India

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Humanized mouse model to study viral infections and evaluate antivirals

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HSV-2 infections cause genital ulcer disease and their by increasing the risk of HIV-1 acquisition approximately three-fold in human. Efforts to prevent recurrent genital ulcer disease with acyclovir failed to reduce HIV-1 acquisition or transmission. HIV-1 vaccine trials have also been unsuccessful to date. A recent population-based model explored the impact of a prophylactic HSV-2 vaccination on HIV-1 incidence in Africa and reported that a vaccination campaign that reduces HSV-2 acquisition and reactivation by 75% in 10 years will reduce HIV-1 incidences by 30-40% after 20 years. If accurate, an HSV-2 vaccine can have a tremendous impact on controlling HIV globally; we have developed a trivalent HSV-2 subunit vaccine that is highly effective in preventing HSV-2 disease and latent infection of dorsal root ganglia in the mouse model and, genital recurrences and vaginal viral shedding in guinea pig model of recurrent HSV-2 infection. Our ultimate goal is to determine whether an HSV-2 vaccine will prevent genital ulcer disease and reduces susceptibility to HIV-1 infection.

To accomplish our goal, we have first established an HSV-2 vaginal infection in humanized mice. These animals have B- and T-cells of human origin. We have shown that severity of vaginal disease is dose dependent. The LD50 value for HSV-2 vaginal infection for a clinical isolate of HSV-2 strain 2.12 is 37 PFU. We have also shown that virus successfully replicates in mouse vaginal tissue and develop vaginal disease. Histochemical and immuno-histochemical analysis of infected humanized mice showed that vaginal tissue was disrupted, and immune cells migrated to the site of infection within first 3 days of infection. While no staining for HSV-2 was observed in liver and spleen, the proliferation of immune cells was observed in spleen within first 3 days of vaginal infection. We plan to infect HSV-2 infected humanized mice with HIV-1 and evaluate the markers of HIV-1 infection and HSV-2 infection.

Biography

Sita Awasthi has received her Ph.D in Biochemistry from Devi Ahilya University at Indore, India and her postdoctoral training from University of Pennsylvania at Philadelphia. Currently she is a Research Assistant Professor in Infectious Disease Division, Department of Medicine, Perelman School of Medicine at University of Pennsylvania, Philadelphia. Her research interests are HSV-2 vaccine development against genital herpes disease and HSV-2 HIV-2 co-infections. She has published numerous research articles and serving as an editorial board member of Journals of antivirals and anti retrovirals, Journal of Immunoassay and Immunochemistry. She has been a Board Member for the Association of Women in Science, Philadelphia chapter.

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Molecular signatures of Venezuelan equine encephalitis in mice

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Venezuelan equine encephalitis virus (VEEV) is an emerging infectious agent and a CDC category B priority pathogen. VEEV has caused periodic outbreaks involving several hundred thousands of equine and human cases. VEEV may also cause severe to fatal encephalitic disease in young and old subjects with underdeveloped or weak immune system. Inflammation in brain following VEEV infection is multifocal and believed to play a key role in the pathogenesis of VEEV. However, the underlying molecular mechanisms of VEEV encephalitis are poorly understood.

Molecular characterization of VEEV encephalitis and its kinetics were evaluated by gene expression profiling of VEEV infected mice brain using whole genome and pathway specific toll-like receptors (TLR) and extracellular matrix and adhesion molecule gene microarrays. Localized expression of translation products was evaluated by immunohistochemistry in the brain tissues.

VEEV infection of mice brain resulted in the differential modulation of several immune pathways such as antigen presentation, inflammation, apoptosis and response to virus. Specifically, VEEV infection up regulated Toll like receptor signaling pathways components and revealed a MyD88 biased TLR signaling. Comparative host gene expression analysis of mice infected with either neuroinvasive or non-neuroinvasive strains of VEEV identified signaling pathways specific to neurovirulent VEEV infection. A gene expression signature that was common to both the neuroinvasive and non-neuroinvasive strain of VEEV was also identified.

Cell to cell adhesion molecules and extracellular matrix protein genes such as ICAM-1, VCAM-1 and MMPs were also differentially regulated in the mouse brain after VEEV infection. Studies in ICAM1 knockout mice infected with VEEV demonstrated a delay in initiation of disease accompanied with reduced inflammation in the brain early in the infection. A twenty percent reduction in the mortality following VEEV infection was also observed in ICAM-1 knockout mice.

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Transient transfection of BeWo cells with a truncated human endogenous retrovirus ERV3 env induces β-hCG

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Case Western Reserve University School of Medicine, USA

Endogenous retroviral element ERV3 env is highly expressed during differentiation of villous cytotrophoblast to syncytiotrophoblast, an essential process in human placentation, two principal characteristics being intercellular fusion and production of the hormone hCG. We reported that, unlike other retroviral env regions that encode fusion proteins, ERV3 regulated the induction of the β subunit of hCG (β-hCG). The apparent biological relevance of ERV3 env was greatly diminish by a report of 2 adults with homozygous stop mutations leading to a "natural knockout" of ERV3 that was translated into a trunicated p25 molecule lacking the typical biologically active regions of exogenous or endogenous retroviral Env proteins. However, the p25 region has never been tested for capacity to induce expression of β-hCG. We cloned and inserted the entire ERV3 env open reading frame (ERV3) or the ERV3 p25 region into pCMV6-AC expression vectors (OriGene), transiently transfected BeWo cells, and monitored for levels of intracellular β-hCG by quantitative Western blot analysis, normalized to levels of actin, and data expressed as means (SD) of the ratio of β-hCG to actin of three independent experiments. β-hCG expression was not detectable in untreated BeWo (negative control) and maximum in forskolin-treated cells (positive control; 1.83 + 0.83). Transient transfection with vector alone did not affect β-hCG expression (0.07 + 0.13), whereas both ERV3 (1.14 + 0.18) and p25 (0.76 + 0.16) induced significantly ($P<0.01$) greater levels of β-hCG expression. Thus, ERV3 env is an atypical retroviral element with a unique trophoblast hormone regulatory site in the SU region.

Biography

Neal S. Rote completed his Ph.D. at Temple University School of Medicine and postdoctoral studies at Heidelberg University and UCLA School of Medicine. He is currently William Weir, M.D. Professor of Reproductive Biology and Professor of Pathology at Case Western Reserve University School of Medicine and Academic Vice Chair and Director of Research in the Department of Obstetrics and Gynecology, University Hospitals Case Medical Center, Cleveland, OH. He has published over 100 papers in reproductive biology, more than 60 chapters and books, been NIH-funded for more than 30 years, and served on many NIH review committees.

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Infectivity of avian Hepatitis E virus isolated from China

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Avian hepatitis E virus (HEV) was first isolated in the USA in 2001 from chickens with hepatitis-splenomegaly (HS) syndrome with the increased mortality, decreased egg production, blood fluid in the abdomen and an enlarged liver and spleen. In 2005, avian HEV was also isolated from healthy chicken flocks. At present, avian HEV infection is endemic in many countries including USA, Spain and China. In 2010, an avian HEV isolate named CaHEV was identified and characterized from a flock of 37-week-old broiler breeder hens with HS syndrome in Shandong, China. Based on the near-complete genomic sequence analysis, CaHEV shares the highest identity (98.3%) with avian HEV from Europe and belonged to avian HEV genotype 3. The infection stock of CaHEV generated by collecting the fecal and bile suspensions from the inoculated chickens was used to inoculate intravenously 15-week-old SPF chickens at approximately 10^4 GE of CaHEV stock (1 ml of the 10% fecal and bile suspension). Fecal shedding of viruses was detected from 3 to 54 day post inoculation (dpi) and viremia was detected in sera from 6 to 21 dpi in all challenged chickens. By 2 week post inoculation (wpi), all inoculated chickens had seroconversion and anti-avian HEV IgG was detected upto 12 wpi. In addition, big livers and spleens were found in two inoculated chickens necropsied at 8 wpi and subcapsular hemorrhages were observed in the livers from chickens necropsied at 6 to 7 wpi. These results showed that CaHEV is infectious.

Biography

Qin Zhao has completed his Ph.D in 2010 from Shandong Agriculture University, China. At present, he is a postdoctoral fellow in Dr. Zhou's lab in College of Veterinary Medicine, Northwest A & F University. He isolated and characterized avian HEV strain from China. His research focus is on the antigenicity and vaccine design for avian HEV. He has published 15 papers in reputed journals.

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Human Mammary Tumor Virus (HMTV) and cancer

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Eleven percent or more of cancers worldwide are linked to viral causes. In many cases individuals are infected with a virus but only a certain percentage develop cancer (19). Similar to other individuals who carry a high risk for developing cancer due to viral infections such as HPV, EBV, HBV, HCV, SV40 and McPyV results from our laboratory and that of others suggest that individuals who are infected with HMTV carry a risk of developing breast cancer.

Retroviral sequences homologous to the β-retrovirus mouse mammary tumor virus (MMTV), the etiological agent of mammary tumor in mice, are present in 40% of American women's breast cancers. A 660 bp sequence, homologous to MMTV env gene with no significant homology to any other viral or human sequence reported in the GenBank, is found in breast cancer and in the breast milk of 7.6% of healthy American women.

A complete provirus structure with 95% homology to MMTV has been isolated from two human breast tumors and named human mammary tumor virus (HMTV). β-retroviral particles from primary cultures of metastatic breast cancer cells (MSSM) have been isolated and characterized HMTV. Virion RNA is more than 90% homologous to MMTV RNA and to the HMTV proviral DNA. HMTV is able to infect a variety of cells bringing about striking molecular changes, as seen by co-culture experiments between MSSM cells and normal human epithelial breast cells, B and T human lymphocytes and human dendritic cells. Protein expression was only observed in 10-20% of the infected cells by FACS analysis suggesting the presence of innate resistance in human cells after HMTV infection, as well as in breast cancer cells. The human retroviral restriction factors APOBEC F and G, the TRIM proteins such as TRIM 1, TRIM 21 and TRIM 25 and Tetherin are all highly expressed in HMTV cells as measured by quantitative RT-PCR. Disruption of the cytoskeleton and evidence for epithelial-mesenchymal transition (EMT) are additional molecular changes seen in infected cells.

In conclusion, HMTV is able to infect a variety of cells bringing about striking molecular changes. Whether these changes play a role in tumorigenesis remains to be shown.

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Prognostic and developed scenarios of pandemic flu in Georgia

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National Centre for Disease Control and Public Health of Georgia, Georgia

Pandemic (H1N1) 2011 flu realistic picture was developed according to moderate scenario. The highest intensivity of pandemic flu cases was reported in places with population diversity. Duration of epidemic wave provoked by pandemic flu was 6 weeks. The peak of epidemic wave continued during 2 weeks. The incidence rate of disease was high in the under 15 years old then in other age groups. Under 1 year and 1-4 years age groups the numbers of cases were accordingly 8789 and 7167. Compared with seasonal influenza, the highest incidence rate was in age group 20-29 old and the number of cases were - 1954. The incidence rate was lower in 60 years and more age groups, as it was predicted by taking into consideration the circulation period of A(H1N1) virus subgroups. Early influenza-associated hospitalizations and the good management reduced the complications and death rate. Intensive therapy and artificial lung ventilation were used for hospitalized patients accordingly 5% and 1.1%. Death was reported in 20-59 years patients. Influenza was accompanied by other somatic or chronic infections.

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Knowledge, attitude, beliefs and practices about HBV vaccination and universal precautions in healthcare workers of a tertiary care centre in India

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[2]Department of Laboratory Medicine, Delhi State Cancer Institute, India

Healthcare Workers have a high risk of occupational exposure to many blood-borne diseases including HIV, Hepatitis B, and Hepatitis C viral infections and therefore Universal Precautions are very crucial for prevention of these infections. This study was conducted to assess awareness of healthcare workers of AIIMS regarding vaccination against Hepatitis B infection and their understanding of Universal Precautions. Their anti-HBs antibody titer was also measured. After ethical clearance and written consent, 446 healthcare workers who were categorized in 7 groups according to their work nature, were explained about the study and asked to fill a questionnaire regarding their vaccination status and practice of Universal Precautions. About 56.5% HCWs were vaccinated and 79% of them had protective levels (>10 IU/mL) of anti HBs antibody titers. However, protective levels were also detected in 19.35% of unvaccinated HCWs as natural immunity. 31.4% HCWs had history of Needle prick injury and only about half of them (47.5%) reported it. Regarding Universal precautions, 84.5% HCWs use gloves regularly, 10.7% use sometimes while only 4.7% HCWs have never used gloves. 38.9% of staff washed hands with disinfectant whereas rest with soap. 76.5% HCWs had knowledge of waste disposal and needle destruction while 67.6% had knowledge of proper management of blood spill. Hence there is need to educate our healthcare workers the importance of HBV vaccination and practicing Universal Precautions. In addition we need well planned and clear policies for healthcare workers about HBV screening, vaccination and serological response checkups including post-exposure management of needle prick cases.

Biography

Dr. Varsha Singhal has completed her MD in Laboratory Medicine from All India Institute of Medical Sciences and presently she is pursuing Senior Residency in Division of Clinical Microbiology, Department of Laboratory Medicine, AIIMS, New Delhi.

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3 (ii) : Immunology and Viral Pathogenesis

Session Chair
Ramila Philip
Immunotope Inc, USA

Session Co-Chair
Pooja Jain
Drexel University, USA

Session Introduction

Title: **Immunoproteomics analysis of infected cells for the identification of conserved MHC class I-presented virus specific T cell epitopes**
Ramila Philip, Immunotope Inc, USA

Title: **Unconventional CD8+ T cells ($CD4^{dim}CD8^{bright}$ cells) in anti-HIV immunity and neuropathogenesis**
Lena Al-Harthi, Rush University Medical Center, USA

Title: **HIV-1 Pathogenesis Env fusogenicity and coreceptor expression levels determine bystander apoptosis induction**
Himanshu Garg, Texas Tech University Health Sciences Center, USA

Title: **Changes in levels of Interleukin-8 in the serum of patients with hepatitis B virus infection correlate with HBe seroconversion and increased levels of Interleukin-8 indicate resistance to IFN-alpha therapy**
Staffan P.E. Sylavan, Uppsala University Hospital, Sweden

Title: **TBA**
John Connolly, Singapore Immunology Network, Singapore

Title: **Cutting edge application of flow cytometry in virology research**
Renold Capocasale, FlowMetric Inc., USA

Ramila Philip, J Antivir Antiretrovir 2012, 4:4
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Immunoproteomics analysis of infected cells for the identification of conserved MHC class I-presented virus specific T cell epitopes

Ramila Philip
Immunotope Inc. USA

Viruses such as influenza and dengue are highly infectious and are significant global public health problems and understanding the overall immune response to infection will contribute to appropriate management of the disease and development of effective vaccines. Although the current vaccines for these viruses target increasing humoral immunity, there is evidence that T-cell responses are extremely important for long lasting protection. Due to the persistence of cell mediated immunity after viral clearance, T-cell responses to conserved epitopes may afford cross strain protection. Identification of peptides displayed by cells infected with viral pathogens generates a library of epitopes and, by genomic database searching, the viral proteins of origin that are recognized and processed by the immune system are identified. Primarily, shared T cell epitope identification for family of viruses has taken the motif prediction or epitope mapping algorithm approach, which often differ from the naturally processed and presented antigenic peptides on infected cells. In the last decade, direct identification of HLA class I presented epitopes from infected cells has emerged as an alternate to the motif prediction method and is termed immunoproteomics.Immunotproteomics methodology is a powerful strategy aimed at the rapid, unambiguous identification of less abundant naturally processed and presented epitopes from infected cells. HLA-associated viral epitopes have been identified from dengue and influenza virus infected cells using ultrasensitive mass spectrometry based immunoproteomics methodology and characterized for cross strain reactive T cell response, which are associated with adaptive immunity and are of strategic importance for vaccine development.

Biography

Ramila Philip, PhD. is currently the President and Chief Scientific Officer at Immunotope, an adjunct professor at Drexel University and professor at the Institute for Hepatitis and Virus Research. Dr. Philip is an internationally recognized expert in immunotherapeutic vaccines and has taken several vaccine products from research stage to early phase clinical trials. She earned her Ph.D. in Immunology in India and did her postdoctoral training at the Institute of Immunology, Basel, Switzerland and was a junior faculty at Cancer Research Institute, University of California, San Francisco. She has over 75 peer reviewed publications in leading scientific journals.

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Unconventional CD8+ T cells ($CD4^{dim}CD8^{bright}$ cells) in anti-HIV immunity and neuropathogenesis

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Rush University Medical Center, USA

Conventional paradigm of T cell biology tells us that expression of the CD4 and CD8 molecules on mature T cells is mutually exclusive. However, my group and others have identified a unique subset of CD8+ T cells that co-express CD4 dimly on their surface. This subset is termed CD4dimCD8bright T cells or double positive (DP) T cells. DP cells represent a genuine phenotype of CD8+ T cells. They express αβTCR and αβCD8 and are not prematurely released from the thymus. Post TCR or superantigen stimulation, 15-60% of purified CD8+ T cells induce stable CD4 expression on their surface. DP cells constitute 3-5% of peripheral CD8+ T cells in healthy donors and are expanded to up to 15% among HIV long-term nonprogressors. DP cells are highly enriched in anti-viral (CMV and HIV) responses. In comparison to their CD8 single-positive T cell counterparts, DP cells constitute greater than 55% of anti-HIV and -CMV responses, as evaluated by MHC-I tetramer analysis, polyfunctional cytokine and effector molecule expression, and antigen-specific proliferation. DP cells are also highly enriched in β-catenin expression, a pro-survival transcriptional co-activator that drives CD4 induction on CD8+ T cells and may protect them from activation-induced cell death. DP cells are found in the CNS of HIV-infected NOD/SCID/IL-2rcγ-/- mice reconstituted with human peripheral blood lymphocytes and while they are susceptible to HIV infection, they are not depleted to the same extent as CD4+ T cells. Astrocyte-conditioned media also induces CD4 expression on CD8+ T cells. Our findings identify DP cells as a potent anti-viral T cell subset; however, their contributing role to HIV-associated neurocognitive impairment, whether protective or pathogenic, remains to be elucidated.

Biography

Dr. Al-Harthi is a Professor in the Department of Immunology/Microbiology at Rush University Medical Center in Chicago, IL. She has over 60 peer-reviewed publications and invited reviews/book chapters. Her research over the past 16 years has focused on HIV/host interactions, with a special emphasis on bridging basic and clinical science in the HIV/AIDS field. Because of her experience in HIV molecular biology, immunology, and neuroAIDS, she has been able to probe mechanistic questions that are clinically relevant to HIV/AIDS. She is actively investigating the molecular pathways by which Wnt/β-catenin signaling inhibits HIV replication, its impacts on HIV neuropathogenesis, and the role of host and viral factors in modulating β-catenin interaction with HIV.

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HIV-1 Pathogenesis Env fusogenicity and coreceptor expression levels determine bystander apoptosis induction

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HIV-1 infections lead to a progressive depletion of CD4 cells culminating in AIDS. The coreceptor usage by HIV varies from CCR5 (R5) tropic early in infection to CXCR4 (X4) tropic in later infections. While the coreceptor switch from R5 to X4 tropic HIV is well associated with progression to AIDS, the role of CCR5 in disease progression especially in patients infected exclusively with R5 isolates throughout the disease remains enigmatic. To better understand the role of CCR5 and R5 tropic HIV envelope in AIDS pathogenesis, we asked whether the levels of CCR5 and/or HIV Env-mediated fusion determine apoptosis of bystander cells. We generated CD4+ T cell lines expressing varying levels of CCR5 on the cell surface, to show that CCR5 expression levels correlate with bystander apoptosis induction. The mechanism of apoptosis involved caspase-3 activation and mitochondrial depolarization and was dependent on gp41 fusion activity as confirmed by fusion restricted gp41 point mutants and use of the fusion inhibitor T20. Interestingly, lower levels of CCR5 were able to support virus replication in the absence of bystander apoptosis. Our findings suggest that R5 HIV-1 mediated bystander apoptosis is dependent on both CCR5 expression levels as well as fusogenic activity of the Env glycoprotein.

Biography

Dr. Himanshu Garg completed a PhD in Immunology from North Carolina State University in 2003 working on Feline Immunodeficiency Virus. He subsequently completed five year Post Doctoral training at the National Cancer Institute focusing on HIV Env glycoprotein and its role in HIV Pathogenesis. He joined Texas Tech University Health Sciences Center as Research Instructor in 2009 and currently holds this position. He has published more than 20 peer reviewed articles in major journals on a variety of topics including HIV pathogenesis, HIV assembly and gene therapy.

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Staffan P.E. Sylvan, J Antivir Antiretrovir 2012, 4:4
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Changes in levels of Interleukin-8 in the serum of patients with hepatitis B virus infection correlate with HBe seroconversion and increased levels of I Interleukin-8 indicate resistance to IFN-alpha therapy

Staffan P.E. Sylvan
Department of Medical Sciences, Uppsala University Hospital, Sweden

The aim of this study was (1) to determine plasma values of CXCL-8/interleukin-8 (IL-8) in patients with different clinical manifestations of hepatitis B (HB) and (2) to analyse the correlation between presence of circulatory levels of IL-8 and levels of HB virus (HBV) DNA during the natural course of acute HB and interferon alpha-induced HBe and HBsAg seroconversion in patients with chronic HB (CHB). Serum IL-8, HBV DNA, and transaminases in serum were measured consecutively (before, during and after treatment) in patients with acute hepatitis B and IFN -alpha-treated patients with chronic HBV infection.

Patients (n=10) with acute HB infection exhibited high IL-8 levels during the acute phase of the infection and low during the resolution of the disease. The peak response of IL-8 was always preceded by the peak level of the transaminases and the reduction of HBV DNA. The peak level of IL-8 coincided with HBe and HBsAg seroconversion during the natural course of acute infection. The presence of IL-8 in circulation was dynamic during treatment in patients with chronic HB. Detectable levels of IL-8 were always measured after the reduction of HBV DNA and transaminase levels. Moreover, the IL-8 levels were significantly higher in patients who did not respond to IFN therapy than in patients who did respond to the therapy ($p<0.005$). The positive predictive value (PPV) of IL-8 serum levels below 69 pg/ml (mean value + 2.5 SD) in determining a virological response was 92% and the negative predictive value (NPV) 100%. IL-8 is associated with acute and chronic active hepatitis B and may be used as predictive marker for response to IFN-alpha therapy in patients with chronic hepatitis B.

Biography

Staffan P.E. Sylvan is a senior expert in infectious diseases and is the county medical officer for Uppsala County. As such he heads the local department of communicable disease control and prevention and has been very active in undertaking campaigns concerning the containment of the spread of communicable diseases such as pandemic influenza, Chlamydia, HIV and hepatitis A, B and C. He has a long standing research career particularly in the area of hepatitis immunology. He has published more than 60 papers in reputed journals

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Ralph A. Tripp, J Antivir Antiretrovir 2012, 4:4
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Keynote Lecture

Ralph A. Tripp

University of Georgia Influenza Pathogenesis and Immunology Research Center, USA

Novel vaccination and therapeutic strategies against respiratory syncytial virus

Biography

Prof Tripp received his doctorate in 1989 from Oregon State University in the field of viral immunity. He was awarded a National Research Service Award and studied adenovirus mechanisms of immune evasion under the tutelage of Dr. Linda Gooding at Emory University, and then was a post-doctoral fellow with 1996 Nobel Laureate Professor Peter C. Doherty at St. Jude Children's Research Hospital where he studied the mechanisms of T cell memory to influenza virus. Following these programs, Prof Tripp led a research team in vaccine studies for respiratory viral diseases, as a Section Chief, in the Respiratory and Enteric Viruses Branch at the CDC in Atlanta, GA. Prof Tripp now oversees research activities at the Animal Health Research Center at the Univ Georgia which is BSL2/BSL3 biocontainment facility where his laboratory develops platform enabling technologies in pathogen biosensing using nanotechnology-based approaches, antiviral drugs using small molecule and RNAi-based drugs, and animal and human vaccines using state-of-the-art technologies with an in-house GMP vaccine facility. Prof Tripp is also a co-founder and CSO of Argent Diagnostics, Inc, and of Trellis Biosciences-Georgia.

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4 (i) : Therapeutic Development

Session Chair
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Session Co-Chair
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Session Introduction

Title: DC-SIGN a potential new target for antiretroviral (HTLV/HIV) drug discovery
Zafar Khan, Drexel University College of Medicine, USA

Title: Broad spectrum anti-virals: Viral protein nuclear import as a target?
Kylie Wagstaff, Monash University, Australia

Title: A kinase inhibitor cocktail as a broad spectrum antiviral
Eric M Vela, Battelle, USA

Title: HIV, methamphetamine and combination antiretroviral therapy: Profiling of histones modification
Pawel Olszowy, University of Nebraska Medical Center, USA

Title: Therapeutic potential of medicinal plants against Hepatitis C virus
Usman Ali Ashfaq, Government College University, Pakistan

Title: Gold nanoparticle conjugated RSV peptides inhibit virus replication
Shree Singh, Alabama State University, USA

Title: Polymeric Prodrugs: Recent achievements and general strategies
Neeraj Agrawal, Pacific College of Pharmacy, India

Title: Effect of traditional Egyptian herbal medicine on treatment of HCV
Abdel Khalek H. Younes, Al- Azhar University Teaching Hospitals, Egypt

Title: Human Immunodeficiency Virus (HIV-1) Reverse Transcriptase inhibition by extracts of the *Phyllanthus Emblica*
Estari Mamidala, Kakatiya University, India

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DC-SIGN a potential new target for antiretroviral (HTLV/HIV) drug discovery

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Despite the susceptibility of dendritic cells (DCs) to human T-cell lymphotropic virus type 1 (HTLV-1) infection and the defined role of these cells in disease pathogenesis, the mechanisms of viral binding to DCs have not been fully delineated. Recently, a glucose transporter GLUT-1, heparan sulfate proteoglycans (HSPGs), and neuropilin-1 (NRP-1) were demonstrated to facilitate HTLV-1 entry into T cells. DCs express their own array of antigen receptors, the most important being the DC-specific intercellular adhesion molecule-3 (ICAM-3)-grabbing nonintegrin (DC-SIGN) with respect to retrovirus binding. Consequently, the role of DC-SIGN and other HTLV-1 attachment factors was analyzed in viral binding, transmission, and productive infection using monocyte-derived DCs (MDDCs), blood myeloid DCs, and B-cell lines expressing DC-SIGN. The relative expression of DC-SIGN, GLUT-1, HSPGs, and NRP-1 was first examined on both DCs and B-cell lines. Although inhibition of these molecules reduced viral binding, HTLV-1 transmission from DCs to T cells was mediated primarily by DC-SIGN. DC-SIGN was also shown to play a role in the infection of MDDCs as well as model B-cell lines. HTLV-1 infection of MDDCs was also achieved in blood myeloid DCs following the enhancement of virus-induced interleukin-4 production and subsequent DC-SIGN expression in this cell population. This study represents the first comprehensive analysis of potential HTLV-1 receptors on DCs and strongly suggests that DC-SIGN plays a critical role in HTLV-1 binding, transmission, and infection, thereby providing an attractive target for the development of antiretroviral therapeutics and microbicides. In this respect, we have developed both cell-based and cell-free high throughput screening assays in order to identify novel inhibitors of DC-SIGN interaction with HTLV-1 gp46 and HIV-1 gp120 proteins.

Biography

Dr. Khan obtained his Ph.D. degree from the Banaras Hindu University, India. He was a Senior Scientist and Deputy Director in the Central Drug Research Institute- a premier organization of the government of India. He served this institute for over 25 years before immigration to USA. He published extensively in high impact factor journals and obtained numerous patents. For many years Dr. Khan's frontier area of research has been on the microbial pathogenesis and therapy of infectious diseases including retroviruses and opportunistic infections. The current research efforts primarily focus on defining the mechanism of HTLV-1-induced neuroinflammation and demyelination in the central and peripheral nervous system in order to identify potential diagnostic markers and High-throughput targets for therapeutic interventions. Other areas of his research interests are HIV-1 therapeutics and microbicide development. Dr. Khan has developed collaborative INDO-US program with a view to develop a new database on HIV-1 cohorts (HIV clade B & C) for the purpose of genotyping, sequencing and novel therapeutic strategies to overcome HAART resistance in HIV/AIDS patients.

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Broad spectrum anti-virals: Viral protein nuclear import as a target?

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Viral disease is one of the greatest burdens of human health, with an urgent need for new anti-viral strategies. Our work examining a number of DNA and RNA viruses indicates that regulated protein movement into and out of the nucleus through the importin (IMP) superfamily of transporters is central to viral infection (see [1]). This is particularly striking in the case of RNA viruses where although viral replication generally occurs in the cytoplasm, gene products important in virus replication/assembly traffic into the host cell nucleus generally to dampen the host cell anti-viral response. Using a variety of in vitro and in vivo approaches, we have delineated the IMPs and targeting signals responsible for the nuclear import/export of specific viral proteins from DNA tumor viruses such as cytomegalovirus/Herpes Simplex Virus, as well as the RNA viruses Dengue (DENV), Respiratory Syncytial Virus (RSV), and Human Immunodeficiency Virus (HIV)-1. Intriguingly, many of the different viral gene products utilize either IMPβ1 or IMPα/β for nuclear import, and IMPβ homologue EXP1/CRM1 for nuclear export, implying that agents targeting their cellular nuclear transport proteins could represent crude broad spectrum anti-viral agents. Our recent results [2,3] support this idea, with 3 different novel inhibitors of IMPα/β -dependent nuclear import reducing infection by DENV as well as HIV-1, by reducing nuclear import of the non-structural protein 5 and integrase proteins, respectively. Nucleocytoplasmic transport thus appears to be a viable target of great significance in the fight against pathogenic viruses.

Biography

Dr. Wagstaff completed her Ph.D in 2007 at Monash University (Melbourne) where she has remained for her post-doctoral studies. She is presently an ARC Australian Post-Doctoral Research Fellow and manages a small group as part of the Nuclear Signalling Laboratory (Monash). Her research focusses on the transport of proteins into and out of the eukaryotic cell nucleus and its therapeutic applications, including the development of inhibitors of theis process as anti-viral agents and how the nuclear transport machinery may be exploited for drug delivery. She has 18 peer-reviewed publications in eminent journals (H-factor of 11) and numerous prestigious awards/prizes.

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A kinase inhibitor cocktail as a broad spectrum antiviral

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Viruses from the Arenaviridae, Flaviviridae, and Filoviridae families have the potential to cause hemorrhagic fever in humans, while viruses from the alphavirus family have the potential to cause debilitating disease. Currently, therapeutics for these viruses are limited and Ribavirin and supportive care remain the only substantial therapeutic options for viral hemorrhagic fever. In this report, we demonstrate that pre-treatment of host cells with a kinase inhibitor cocktail consisting of genistein and tyrphostin AG1478 leads to inhibition of infection in cells infected with numerous viruses including Venezuelan equine encephalitis virus, Dengue virus, West Nile virus, Pirital virus, Pichindé virus, Lassa virus, Flexal virus, Ebola virus, and Marburg virus. Additionally, we tested the affects of the kinase inhibitor cocktail in the Pirital virus (PIRV)-Syrian golden hamster model, since infection results in hemorrhagic fever and 100% mortality and because this model can be used to screen antivirals intended to treat hemorrhagic fever. Treating the PIRV-infected Syrian golden hamsters with the kinase inhibitor cocktail led to significant survival, lower viral titers in specific tissues and viremia, and a mitigation of disease signs. In all, the results from these studies demonstrate that a kinase inhibitor cocktail may serve as a broad spectrum antiviral that may be used as a therapeutic or prophylactic against a myriad of virus infections.

Biography

Dr. Vela has 12 years of experience as a virologist and is currently the Manager for Virology and Toxinology at the Battelle Biomedical Research Center. He has extensive experience with a myriad of viruses including highly pathogenic avian influenza, seasonal influenza, Pichindé virus, Lassa virus, Pirital virus, Flexal virus, Ebola virus, Marburg virus, Dengue virus, West Nile virus, Human Immunodeficiency virus, and Simian immunodeficiency virus. His animal modeling research has involved ferrets, guinea pigs, nonhuman primates, and hamsters. As a Post-Doctoral Fellow, Dr. Vela studied arenavirus pathology and as a graduate student (under the supervision of Dr. Jagannadha Sastry), Dr. Vela studied HIV-1 entry mechanisms into host cells.

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HIV, methamphetamine and combination antiretroviral therapy: Profiling of histones modification

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[1]University of Nebraska Medical Center, USA
[2]Nicolaus Copernicus University, Poland

Histones are key proteins that play an important role in maintaining and regulating chromatin. Five classes of histones have been identified and all, with the exception of H1,form anoctamer that DNA wraps around to form nucleosomes.In an equilibrium state, acetyltransferases (HATs) and deacetyltransferases (HDACs), as well as methylases and demethylases, keep the balance of transcriptional activation. Activation of chromatin is based on acetylation and de-methylation of lysine residues in histones. Parallel de-acetylation and methylation deactivates and disables the possibilities of biochemical processing of DNA.

The goal of this study is to analyze and quantitate changes in histones methylation and acetylation in monocyte derived macrophages upon a combination of HIV-1 infection, exposure to methamphetamine (METH) and/or drugs constituting combination antiretroviral therapy (cART). Histones were extracted using a commercially available kit, and thenseparated using either 1D gel electrophoresis or liquid chromatography.Purified histones were digested with either trypsin/chymotrypsinand theresulting fragments were analyzed using tandem mass spectrometry for identification and quantitation.

Here, we report differences in post-translational modifications of histones and the so called "histone code"as an effect of manipulation of our experimental system. We will also attempt to measure ubiquitination and phosphorylation as two additional modifications affecting transcriptional regulation.

Biography

Pawel Olszowy completed his Ph.D at Nicolaus Copernicus University in Torun, Poland in 2011. Since this time he is a postdoctoral researcher at University of Nebraska Medical Center in Omaha, Nebraska. He has published morethan 10 papers in peer review recognized journals. His main scientific interests are proteomics, separation techniques and mass spectrometry.

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Usman Ali Ashfaq, J Antivir Antiretrovir 2012, 4:4
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Therapeutic potential of medicinal plants against Hepatitis C virus

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Hepatitis C virus is a major cause of chronic liver diseases which can lead to permanent liver damage, hepatocellular carcinoma and death. The presently available treatment with interferon plus ribavirin, has limited benefits due to adverse side effects and high cost. Hence, there is a need to develop anti-HCV agents, which are less toxic, more efficacious and cost-effective. The present study was aimed at making a sustained search for antiviral compounds and studies their therapeutic potential as anti-HCV drug. To this end, in-vitro bioassay was developed for screening antiviral activity of medicinal plant extracts. Fifty herbs were collected from different parts of Pakistan on the basis of reports (undocumented) of antiviral activity against different viral infections. Firstly, the effects of medicinal plant extracts were studied on the cellular growth of liver and fibroblast cells. Subsequently, HCV infected liver cells were treated with medicinal plant extracts at non toxic doses and replication of viral RNA was measured by Quantitative real time RT-PCR. Two out of fifty herbs exhibited activity against HCV in our in-vitro assay. In order to identify the active ingredient, corresponding herbal extracts were fractioned by thin layer chromatography (TLC), column chromatography and HPLC. Purified fractions were tested for activity against HCV in in-vitro assay. Resultantly three active fractions against HCV were identified and combination of these active fractions with interferon may open new avenues of future HCV therapies.

Biography

Usman Ali Ashfaq completed his PhD in Research Project "Studies on the therapeutic effect of selected phytochemicals against Hepatitis C Virus from Center of Excellence in Molecular Biology, University of the Punjab, Lahore. Usman Ali Ashfaq has 23 International publications with almost 57 Impact factor. He has 1 year pre PhD experience in Center of Excellence in Molecular Biology, University of the Punjab (1st March 2010- 29 Feb-2011 and 7 month post PhD Research Experience (March 2011 to Nov 2012) in Allama Iqbal Medical College and Research center. Usman Ali Ashfaq has work experienced on antiviral drugs against HCV, siRNAs against viral and cellular genes, fibrosis, oxidative stress, apoptosis and steatosis leading to HCC.

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Gold nanoparticle conjugated RSV peptides inhibit virus replication

Shree R. Singh
Alabama State University, USA

Respiratory syncytial virus (RSV) belongs to the Paramyxoviridae family of Pneumoviruses which causes bronchiolitis in infants. Recent studies involving synthetic peptides have shown that peptides derived from the fusion protein of paramyxoviruses can bind to the F protein of RSV and block the necessary conformational changes needed during the infection. Gold nanoparticles have been used due to their efficient intracellular delivery and functionalization capacity. In the present study, two synthetic anti-RSV peptides were used to functionalization gold nanoparticles. The gold nanoparticles, peptides and the peptide-functionalized gold nanoparticles (fGNPs) were assessed for cytotoxicity to the HEp-2 cells in vitro using the MTT assay. The fGNPs were then used to evaluate their ability to inhibit RSV infection in various stages of infection including the pre-infection, viral binding stage and the post infection with RSV. The viral inhibition was assessed using the plaque reduction assay, immunofluorescence microscopy, qPCR and western blot. The MTT assay revealed that the gold nanoparticles, the peptides and the fGNPs were all non-cytotoxic to the HEp-2 cells at the highest concentrations i.e. 5nM, 50μM and 5nM respectively. The plaques assay as well as qPCR showed reduction in viral replication in vitro. The preliminary in vitro studies indicated that fGNPs were effective in inhibiting RSV replication. Animal experiments are being carried out to assess the in vivo effectiveness of fGNPs.

Biography

Shree R. Singh serves as Professor of microbiology and Director of Center for NanoBiotechnology at Alabama State University. He has been involved with vaccine development and immune system response against novel recombinant and protein vaccines for about 20 years. Dr. Singh has helped secure federal funding of over $20 million while at ASU. His recent research involves field of nanobiotechnology which uses novel nanomaterials to develop anti-viral molecules and develop nanosensors for detection of microorganisms. Dr. Singh has authored over 40 original research articles and several book chapters. Dr. Singh has made over 140 scientific presentations at scientific meetings and delivered many seminars in the US and other countries.

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Polymeric Prodrugs: Recent achievements and general strategies

Neeraj Agrawal
Pacific College of Pharmacy, India

It is well known that polymeric prodrug or polymer-drug conjugate is an effective and fast growing technique for improved use of drugs for therapeutic applications. Polymer conjugated drugs generally exhibit prolonged half-life, higher stability, water solubility, lower immunogenicity and antigenicity and specific targeting to tissues or cells.

The potential of the polymer-drug conjugates have already been proved by success of many products in the market for the treatment of different diseases. The discussion on the topic will cover a description of polymeric drug delivery systems along with recent advancements that have been made in the area of polymer therapeutics.

The points that will be addressed in the talk include: Rational for design of polymer-drug conjugates, requirements for selection of drug candidate for polymeric prodrug, requirements for selecting polymers as candidate drug carriers, classification of polymers, design and synthesis of polymeric prodrugs, strategies to reduce steric hindrances exhibited by polymers and the bio- components, strategies to enhance the reactivity of polymer and the drug by incorporation of spacers, structure-activity relationship of conjugates (SAR) and passive and active targeting of polymer-drug conjugates to specific site of drug action.

Biography

Dr. Neeraj Agrawal is Associate Professor in the Department of Pharmaceutical Analysis at Pacific College of Pharmacy, Pacific University, Udaipur, India. He is having eight years of teaching experience in Pharmacy profession. His research area is design and evaluation of polymeric prodrugs for sustained and site specific drug delivery. He is also involved in development of analytical methods using UV-Visible spectrophotometer and HPLC. He is having many publications in research and review journals of high profile published by Elsevier, Informa Healthcare, PDA etc. He has written a book entitled “Basics of Anatomy and Physiology” with Elsevier publication. He has also presented many research papers in National and International conferences.

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Effect of traditional Egyptian herbal medicine on treatment of HCV

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Acknowledgements: I do respect to my patients for their cooperation during the process of the study

Background/Aims: Hepatitis C is an progressing global health problem. The expense of the exciting regimen for treatment is not available for many patients .Herbal medicine have been used as complementary therapy in the treatment of liver diseases for a long time. In the current study the herbal medicine used in treatment of HCV are Milk Thistle , Phyllanthus , Garlic , Cinnamon , parsley , Black seed and AKHY-J-25 (mixture of herbs). The aims is to assess the efficacy of that complementary therapy in treating chronic hepatitis C.

Methods: Fifty one Patients with hepatitis C have been seen in our out patient clinic , With Twelve healthy control . Patients were interviewed to obtain detailed clinical data before and after treatment. Every patient received single oral capsule of herbal preparations powder , in early morning on an empty stomach with two cups of water and simple breakfast after two hours , from three months to two years and twelve control received placebo.

Results: Twenty percent of patients had no detectable HCV RNA in serum at 24 week treatment ,72.6% showed clinical and biochemical improvement with decline of PCR to lower limit and 7.4% showed clinical and biochemical improvement without change in level of PCR. Mean PCR, SGPT, SGOT, platelets, WBCs, neutrophils, Lymphocytes showed very highly significant results (p=0.0001), HG showed highly significant improvement(p=0. 001),Creatinine showed significant improvement (p= 0.009) and Blood urea and RBS showed non significant changes respectively (p = 23.98 , p = 2.2).

Conclusions: Traditional Egyptian herbal product showed significant improvement of hepatitis c.

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Human Immunodeficiency Virus (HIV-1) Reverse Transcriptase inhibition by extracts of the *Phyllanthus Emblica*

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Infectious Diseases and Metabolic Disorders Research Lab, Kakatiya University, India

Human immunodeficiency virus type-1(HIV-1) is the cause of acquired immune deficiency syndrome (AIDS), a major human viral disease with about 33.2 million people infected worldwide. The high cost of the HAART regimen has impeded its delivery to over 90% of the HIV/AIDS population in the world. The aim of the present study was to evaluate the invitro anti-HIV activity of Phyllanthus emblica plant extracts. Extracts were prepared from dried fruit in n-hexane, ethyl acetate and n butanol. Peripheral Blood Mononuclear Cells (PBMCs) isolated from healthy donors by ficoll-hypaque density gradient centrifugation method. A toxicity study was performed on all crude extracts by MTT assay using PBMCs isolated from whole blood. HIV-1 RT inhibition activity of the all solvent extracts of Phyllanthus emblica was determined. AQF and HXF fractions show highest inhibition of recombinant HIV-RT (91% and 89% respectively) at 1 mg/ml concentration. Chloroform (CFF) fraction shows highest inhibition of HIV-RT at 0.5 mg/ml and Carbon tetra chloride (CTF) fraction at 0.12 mg/ml concentration. The highest non-cytotoxic concentration (>95% cell viability) of HXF, CTF, CFF and AQF fractions are 0.02, 0.04, 0.02 and 0.02 mg/ml respectively. At 0.12 mg/ml and 0.5 concentrations 50% of the HIV-RT activity is inhibited in HXF and CTF fractions respectively. The fruit of Phyllanthus emblica extracts are shows anti-HIV-1 activity and this plant has great potential for developing useful drugs.

Biography

Dr. Estari Mamidala has completed his Ph.D at the age of 28 years from Kakatiya University. He is the Assistant Professor of Zoology, Kakatiya University, Warangal, Andhra Pradesh, India. He has attended more than 40 International and National conferences. He has published more than 30 papers in reputed journals. He has handled 6 research projects and awarded 12 M.Phils and one Ph.D.

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4 (ii) : Organ and Cell Type - Specific Virology

Session Chair
Honglin Luo
University of British Columbia, Canada

Session Co-Chair
Anand Mehta
Drexel University College of Medicine, USA

Session Introduction

Title: **The pro-viral role of autophagy in coxsackievirus-induced myocarditis**
Honglin Luo, University of British Columbia, Canada

Title: **Oncolytic viruses, can Parovirus B19 be naturally oncolytic: Novel clinical evidence indicates it may be oncolytic in leukemic children**
Janak Kishore, Sanjay Gandhi Post-graduate Institute of Medical Sciences, India

Title: **Effects of Hepatitis B virus S protein exposure on sperm membrane integrity and functions**
Tianhua Huang, Shantou University Medical College, China

Title: **Association of human endogenous retroviruses with multiple sclerosis**
Bjørn A. Nexø, Aarhus University, Denmark

Title: **The search for biomarkers of liver cancer: What our failures have taught us**
Anand Mehta, Drexel University College of Medicine, USA

Title: **Concentration levels of IL-10 and TNFα cytokines in patients with Human Papilloma virus (HPV) DNA+ and DNA- Cervical Lesio**
Husham Bayazed, Technical Institute, Iraq

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The pro-viral role of autophagy in coxsackievirus-induced myocarditis

Honglin Luo and Junyan Shi
University of British Columbia, Canada

Autophagy is a cellular process by which damaged organelles/proteins are enwrapped by double membrane vesicles (autophagosomes) and degraded following fusion with lysosomes. Autophagy is traditionally considered as an anti-viral host response. We recently provide evidence that the autophagy machinery can be utilized by coxsackievirus B3 (CVB3), one of the predominant viruses causing myocarditis, to achieve successful replication. Autophagy process is activated during CVB3 infection. Inhibition of autophagosome formation significantly reduces viral replication. Conversely, induction of autophagy results in increased viral replication. Blockage of autophagosome-lysosome fusion by gene silencing of the lysosomal protein LAMP2 promotes viral replication. These results suggest that autophagosomes are likely utilized by CVB3 as sites for active viral replication. To further explore the pro-viral mechanisms of autophagy, we examined the protein levels of p62. P62 is an adaptor protein mediating selective autophagy pathway by targeting ubiquitinated proteins and invading pathogens to the autophagy pathway. We found that CVB3 infection leads to marked decreases in the protein expression of p62 (~62 kDa), accompanied by the appearance of ~30 kDa fragments. This observation was further confirmed using a flag-tagged p62 construct, suggesting that p62 is cleaved after CVB3 infection. We further demonstrated that CVB3-induced cleavage of p62 dissociates its LIR and UBA domains from PB1 domain, resulting in the loss of its function in selective autophagy. Together, our results suggest that the autophagy adaptor protein p62 is cleaved during CVB3 infection. Cleavage of p62 may be a viral strategy to establish efficient viral replication in host cells.

Biography

Dr. Honglin Luo is an Associate Professor in the Department of Pathology and Laboratory Medicine/James Hogg Research Center at the University of British Columbia, Canada. Dr. Luo completed her MSc and MD training in China. She then pursued her postdoctoral training at the University of Washington. Dr. Luo has published over 60 refereed papers and served as an editorial board member of several journals.

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Oncolytic viruses can Parvovirus B19 be naturally oncolytic: Novel clinical evidence indicates it may be oncolytic in leukemic children

Janak Kishore
Sanjay Gandhi Post-graduate Institute of Medical Sciences, India

Virotherapy of cancer using oncolytic viruses are new biological therapeutics since such viruses can preferentially infect and destroy cancerous cells specifically while sparing the surrounding tissues but there are no reports on whether parvovirus B19 can have an oncolyticproperty ? Two independent studies were done to find the role of B19 infection and cases were followed up to one year including mortality data. First one comprised of 35 children with hematological malignancies (Kishore et al 2011); six children (17.1%) had B19 IgM antibodies (5 ALL, 1 NHL) compared to one in 30 controls ($p<0.05$).Of five B19 IgM positive ALL cases two had B19 DNA both by PCR (VP1-VP2 common) and nested-PCR (VP1 unique) indicating presence of B19 genome and giant pronormoblasts (lantern cells) while third case had B19 IgMand lanten cell. In second group 50 children with acute viral hepatitis(AVH) were studied with 60 controls and 13 (38.2%) children had B19 IgM antibodies and 5 had B19 DNA. Six of 16 (37.5%) FHF (fulminant hepatic failure) children were positive for B19 IgM antibodies and 3 had DNA also. Only 1 of 37 (2.7%) controls was positive for B19 IgM antibodies. Three cases each of FHF and AH were negative for hepatitis viruses but had B19 DNA.

Data on mortality indicates that among children with AVH four died and all had B19 infection in contrast five children with ALL died but none had B19 infection. In the third case one child with CLL was found to have anti-B19 IgM antibodies and DNA did not go in relapse for about one year. Hence there is a need to explore if B19 virus has oncolytic properties.

Biography

Professor Janak Kishore is Chief of Serology and Molecular Virology in the Department of Microbiology, Sanjay Gandhi Post-graduate Institute of Medical Sciences, India. He was Associate Editor Indian Journal of Virology, member National Academy Medical Sciences, American societies and Fellow of JICA, Japan. Dr Kishore taught for over 30 yrs with pioneer work on parvovirus B19, developed in-house PCR and ELISA for B19 and published three novel clinical associations of B19. He also worked on cytomegalovirus, enteroviralhemorrhagic conjunctivitis, rubella etc. Dr. Kishore published over 50 papers, served as reviewer for reputed journals, organized conferences, Chaired sessions and frequently invited to speak at international conferences.

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Effects of Hepatitis B virus S protein exposure on sperm membrane integrity and functions

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Research Center for Reproductive Medicine, Shantou University Medical College, China

Background: Hepatitis B is a public health problem worldwide, but only scant information about the influence of hepatitis B virus (HBV) infection on sperm quality is available. The purpose of this study was to investigate the effect of HBV S protein (HBs) on human sperm membrane integrity and functions.

Methods/Principal Findings: Reactive oxygen species (ROS), lipid peroxidation (LP), total antioxidant capacity (TAC) and phosphatidylserine (PS) externalization were determined. The terminal deoxynucleotidyl transferase-mediated dUTP nick end labeling (TUNEL) assays and flow cytometric analyses were performed. (1) After 3 h incubation with 25 μg/ml of HBs, the average rates of ROS positive cells, annexin V–positive/ propidium iodide (PI)-negative cells, Caspases-3,-8,-9 positive cells and TUNEL-positive cells were significantly increased in the test groups as compared to those in the control groups, while TAC level was decreased when compared with the control. The level of malondialdehyde (MDA) in the sperm cells exposed to 50 μg/ml of HBs for 3 h was significantly higher than that in the control ($P<0.05$-0.01). (2) HBs increased the MDA levels and the numbers of ROS positive cells, annexin V–positive /PI-negative cells, caspases-3, -8, -9 positive cells and TUNEL-positive cells in a dose-dependent manner. (3) HBs monoclonal antibody (MAb) and N-Acetylcysteine (NAC) reduced the number of ROS-positive sperm cells. (4) HBs decreased the TAC levels in sperm cells in a dose-dependent manner.

Conclusion: HBs exposure could induce oxidative stress and apoptosis in sperm cells, resulting in loss of sperm membrane integrity and causing sperm dysfunctions.

Biography

Professor Tianhua Huang and his group study on vertical transmission of infectious viruses via germ line for 19 years. He is the director of Research Center for Reproductive medicine of Shantou University Medical College, China. He has published more than 160 papers and won the second prize of the 7th Royan International Research Award in 2006. He is Deputy Secretary General of Chinese Environmental Mutagen Society and serves as an editorial board member of several academic journals.

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Association of human endogenous retroviruses with multiple sclerosis

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[2]Department of Neurology, Aarhus University Hospital, Denmark
[3]Department of Molecular Biology and Genetics, Aarhus University, Denmark
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Retroviruses can cause demyelinating diseases in sheep, mouse, macaque and man. In these cases, the retroviruses involved are contagious and propagate horizontally.

Recently, we described MS to be genetically associated with the endogenous retroviral locus HERV-Fc1, located on the X-chromosome (Nexø et al (2011) 6:16552). The association could be reproduced in several cohorts, encompassing a total of 1697 cases and 2828 controls. Also, we have described that expression of HERV-Fc1 RNA is 4-fold higher in plasma from MS patients with recent attacks, compared to patients in stable remission and from controls ($p = 0.001$) (Laska et al (2012) J. Virol., 86:3713-22). Thus, multiple sclerosis seems caused in part by endogenous retroviruses that are passed vertically as part of the chromosomes.

HERV-Fc1 has a defective pol gene. Therefore, a second virus may contribute to the activity. A statistical search showed that a virus, HERV-K13, on chromosome 19 interacts strongly with HERV-Fc1 in association to disease ((p-interaction) = 0.0003). Quantification of HERV-K13 RNA in plasma from MS patients and controls shows that this virus is approximately 4 fold increased in MS patients, in particular in those in a stable state ($p = 0.004$), but also in patients after attacks ($p = 0.03$) (Nissen et al (2012) manuscript in prep). Thus, expression of HERV-K13 seems associated with disease in general, while expression of HERV-Fc1 is specifically associated to attacks. These results move the association of retroviruses with MS from a result of purely scientific interest to a result with potential implications for the patients.

Biography

BAN holds a PhD from University of Copenhagen. He performed postdoctoral studies at The Johns Hopkins Medical School. He worked for a number of years at Novo Nordisk Inc, where he contributed to the development of NovoSeven®. For the last decade he has been an associated professor at Biomedicine, Aarhus University, Denmark. He has 90 publications in journals and monographs.

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The search for biomarkers of liver cancer: What our failures have taught us

Anand Mehta
Drexel University College of Medicine, USA

5% of all deaths worldwide are the result of liver disease. Many of these deaths could be prevented through the early detection of advancing disease and subsequent intervention. Changes in glycosylation have long been associated with illness. Using a comparative glycoproteomics approach we have identified biomarkers that can detect the early stages of liver cirrhosis and liver cancer, the two major causes of liver disease mortality. Briefly, these biomarkers are common serum proteins with altered glycosylation. The altered glycosylation observed is increased levels of fucosylation. Using a simple plate based approach we have been able to examine the use of these biomarkers in the management of those with liver disease. While many of the markers showed great promise in independent cohorts and publications, markers often had poor sensitivity and specificity. Analyses of false positives lead to the discovery of secondary glycan modifications that resulted in false signals. In addition, examination of poor sensitivity in certain cohorts identified the importance of using specific cancer sub-types for both discovery and validation. Benefits of this talk include insights into the role of glycosylation in biomarker discovery and development, the targeted glycoproteomics method used to discover biomarkers and the growing importance of liver disease worldwide.

Biography

Anand Mehta, is an Associate Professor of Microbiology and Immunology, Drexel University College of Medicine. Dr. Mehta received his graduate degree in Biochemistry from the University of Oxford. Dr. Mehta was one of the first to examine total serum for changes in glycosylation as a function of cancer development. By combining glycomics with proteomics, Dr. Mehta discovered several biomarkers of liver disease and liver cancer, some of which are already available commercially in Asia (GP73, HotGen Biotech, Beijing, China) or under development in the USA

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4 (iii) : Vaccine Efficacy

Session Chair
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Session Co-Chair
John Connolly
Singapore Immunology Network, Singapore

Session Introduction

Title: **Identification of Varicella Zoster virus tissue-tropic genes for developing a neuroattenuated vaccine against chickenpox and shingle**
Hua Zhu, UMNDJ-New Jersey Medical School, USA

Title: **Efficacy of eastern equine encephalitis virus vaccine candidates**
Pamela J. Glass, U.S. Army Medical Research Institute of Infectious Diseases, USA

Title: **Hepatitis B surface antigen variants in voluntary blood donors in Nanjing, China**
Yonglin Yang, Nanjing Red Cross Blood Center, China

Title: **B cell epitope mapping of avian Hepatitis E virus capsid protein**
En Min Zhou, Northwest A & F University, China

Title: **Enigma of HIV control**
Fallahian F, Tehran University of Medical Sciences, Iran

Title: **Evaluation of the efficacy of vaccination of partridge against H9N2 vaccine**
Nili, Shiraz University, Iran

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Identification of Varicella Zoster virus tissue-tropic genes for developing a neuroattenuated vaccine against chickenpox and shingles

Hua Zhu
UMNDJ-New Jersey Medical School, USA

Varicella zoster virus (VZV) is the causative agent of varicella (chickenpox) and herpes zoster (shingles). After primary infection, VZV establishes latent infection in sensory ganglia, and reactivates upon weakening of the immune system due to various conditions, resulting in a productive infection of sensory neurons and the surrounding skin tissue. However, little is known about the molecular basis of VZV latency and reactivation. In our previous work, we employed a VZV bacterial artificial chromosome system containing a green-fluorescent protein and a luciferase marker (VZV BAC luc) in conjunction with live bioluminescence imaging to create and characterize a comprehensive library of VZV single open reading frame (ORF) deletion mutants. We reported three VZV gene categories based on their requirement for viral replication in melanoma cells: essential (44 ORFs), non-essential with severe growth defects (8 ORFs) and fully dispensable (18 ORFs). We postulated that the latter category is comprised of elements responsible for specific tissue tropism. We now demonstrate that ORF7 is required for VZV replication in xenografts of human skin and dorsal root ganglia (DRG) in a severe combined immunodeficiency (SCID) mouse model. The ORF7 protein is a virion component and localizes to the Golgi compartment in infected cells. Loss of ORF7 function in the VZV mutant may serve the basis for the development of a new neuroattenuated varicella vaccine. Our studies also demonstrate the potential utility of this VZV replicating expression vector to develop recombinant vaccines against other important microbial pathogens such as HIV.

Biography

Hua Zhu has completed his Ph.D. from Columbia University and postdoctoral studies from Princeton University. He has been working on human cytomegalovirus and varicella zoster virus for most 20 years. He has published more than 40 research papers, review articles and book chapters, and serving as an Editor-in-chief of Journal of Antivirals & Antiretrovirals.

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Efficacy of eastern equine encephalitis virus vaccine candidates

Pamela J. Glass[1], Shelley P. Honnold[1,2], Russell R. Bakken[1], Jeffrey W. Cohen[1], Lori M. Rowan[1], Kevin B. Spurgers[1], Anuj Sharma[2], Michael D. Parker[1] and Radha Maheshwari[2]

[1]Virology Division, Viral Biology Branch, U.S. Army Medical Research Institute of Infectious Diseases, USA
[2]Department of Pathology, Uniformed Services University of the Health Sciences, USA

Eastern equine encephalitis (EEE) virus is a member of the Alphavirus genus in the family Togaviridae. EEE virus has the highest mortality of the alphaviruses with rates ranging from 33-75% in humans and 90% in horses. Although natural infections are acquired by mosquito bite, EEE virus is highly infectious by aerosol. Veterinary vaccines have been effective in the control of EEE virus and unlicensed vaccines have been used under investigational new drug status for at-risk individuals. However, there is no licensed vaccine available for EEE or any other Alphavirus.

Previously published studies demonstrated protective efficacy of low-dose formalin- and gamma-irradiated inactivated vaccine candidates for Venezuelan equine encephalitis (VEE) virus against subcutaneous challenge with wild-type virus. However, these candidates were only partially protective against an aerosol challenge. In this study, we compared the efficacy of inactivated EEE virus vaccine candidates of varying doses, schedules and routes of administration against an aerosol challenge. Formalin, gamma-irradiation and 1,5-iodonaphthylazide (INA) were used to inactivate a genetically modified strain of EEE virus (CVEV1219). BALB/c mice were administered one or two doses of the inactivated candidates by the subcutaneous (SC), intramuscular (IM), or intranasal (IN) routes and subsequently challenged 28 days after final vaccination by the aerosol route. INA-inactivated CVEV1219 was unable to provide substantial protection against an aerosol challenge by any route, dose or schedule tested. Both formalin- and gamma-inactivated CVEV1219 were able to protect against mice from lethal EEE virus aerosol challenge. Future studies will examine onset and duration of the protective inactivated vaccines.

Biography

Dr. Glass earned her Ph.D. in Virology from Baylor College of Medicine, Houston, TX, in 2001. Currently, she is the Chief of the Viral Biology Department, within the Virology Division at USAMRIID. She has over 10 years experience in viral vaccine, therapeutic and animal model development. The focus of her lab involves studies to examine the effectiveness and targets of potential therapeutics against viruses in cell culture and animal models as well as safety and efficacy of vaccines in animal models.

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Hepatitis B surface antigen variants in voluntary blood donors in Nanjing, China

Yonglin Yang
Nanjing Red Cross Blood Center, China

Hepatitis B virus (HBV) is still one of the serious infectious risks for the blood transfusion safety in China. One plausible reason is the emergence of the variants in the major antigenic alpha determinant within the major hydrophilic region (MHR) of hepatitis B surface antigen (HBsAg), which have been assumed to evade the immune surveillance and pose a challenge to the disease diagnosis. It is well documented that some commercial ELISA kits could detect the wild-type but not the mutant viruses. The high prevalence of HBV in China also impaired the application of nucleic acid testing (NAT) in the improvement of blood security. Among of 20,326 blood units in the Red Cross Transfusion Center of Nanjing from October 2008 to April 2009, 296 samples (1.46%, 296/20,326) were HBsAg positive in the 2 successive rounds of the ELISA test. In these HBsAg positive units, HBV S gene could be successfully amplified from 39 donors (13.18%, 39/296) in the nested-PCR. Sequence analysis revealed that 32 strains (82.1%, 32/39) belong to genotype B, 7 strains (17.9%, 7/39) to genotype C. Besides well known G145R, widely dispersed variations in the MHR of S region, were observed in 20 samples of all the strains sequenced. These mutations in the MHR of HBsAg may be associated with disease diagnosis and the risks for the blood transfusion safety.

Biography

Dr. Yonglin Yang has completed his PhD from Nanjing Medical University in 2011. He is the director of Department of Quality Control and Management from Nanjing Red Cross Blood Center. Also Dr. Yonglin Yang is a member of Chinese Medical Association. Recently he has published the article about HBV mutation in the Virology Journal.

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B cell epitope mapping of avian Hepatitis E virus capsid protein

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Avian Hepatitis E virus (HEV), which is a non-enveloped, positive-sense, single-stranded RNA virus, belongs to the genus Hepevirus which also includes swine and human HEVs. Although the entire genome shares only approximately 50% nucleotide sequence identities with human and swine HEVs, avian HEV is related genetically and antigenically to human and swine HEVs. The capsid protein of avian HEV, encoded by ORF2 gene, is very immunogenic and induces neutralization antibodies. Six major antigenic domains, I (aa 389-412), II (aa 481-492), III (aa 556-566), IV (aa 583-600), V (aa 339-382) and VI (aa 23-88), were predicted in the capsid protein of avian HEV. In domain I, the B-cell epitopes are located in the second half of the domain (aa 400-410) and one epitope is common to avian, human and swine HEVs. In domain II, one or more B-cell epitopes are located in aa 477–492 and are unique to avian HEV. In domain IV, one or more epitopes are shared between avian and human HEVs. In domain V, three epitopes are identified in which two epitopes are common to avian, human and swine HEVs. In addition, antibodies to domain V antigens are persistent in chickens experimentally challenged with avian HEV stock. However, antibodies to domain VI antigens are transient in the challenged chickens. Two neutralization epitopes located in aa476-513 and aa 513-570 are identified since the monoclonal antibodies recognized the two epitopes can neutralize the virus infectivity and capture the virus. Another two epitopes located in domain V and domain I may be neutralization epitopes because these two monoclonal antibodies recognized the two epitopes can also capture the virus particles.

Biography

En-Min Zhou has completed his Ph.D in 1993 from University of Manitoba, Canada and postdoctoral studies from Southwest Foundation for Biomedical Research in San Antonio, Texas. He has spent 21 years in Canada and USA served for Agriculture Canada and Iowa State University. He currently is the distinguished professor and Dean of College of Vet Med at Northwest A&F Univeristy. He has published more than 80 papers in reputed journals and serving as editorial board members of several repute journals.

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Enigma of HIV control

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It seems control of HIV infection spread is lag behind research for new treatments, especially in developing countries. Intravenous drug use is a major risk factor for HIV infection. Addiction control is a reasonable measure for HIV transmission prevention. Implementing treatments for opiate dependence such as: training drug dependence side effects in schools, omitting behavioral and environmental addiction predispositions, organizations to assist addicted, HIV-infected infected patients to change their lifestyles, and teaching prevention and health maintenance, promoting access to diagnosis and treatment, and to remain in treatment is necessary. More definite protocols for screening and surveillance of at-risk groups by implementing ethical and legal codes, and also better supervision on addiction and harm reduction policies may decrease number of new victims. Estimating addiction patterns and prevalence of HIV infection in different situations may help to model prevention protocols. Lack of routine testing of at-risk populations, data registry screening programs and intervention strategies had lead to confronting HIV- infected subjects lately when presenting with co-morbidities such as advanced liver failure, tuberculosis and opportunistic diseases. It seems in developing countries more policy guidelines for testing, counseling and treatment, and modeling HIV control according to at-risk population such as in the injecting drug users, male homosexual context is needed.

Regarding transmission of HIV infection from undiagnosed cases or by those neglecting treatment to community, it is necessary that prevention methods, receive of appropriate prophylaxis, practical use of recommendations, harm reduction policies, adverse drug side effects, treatment failure, and drug resistance followed and supervised regularly in each country.

Biography

F.Fallahian has completed internal medicine specialty and passed clinical ICU fellowship, graduated from Tehran University of Medical Sciences with a history of working in gastrointestinal and liver diseases research centers and published about 30 articles in this field in Iranian and international journals. Now working in Intensive Care Unit, Firuzgar Hospital, Tehran University of Medical Sciences, Vali asr Square, Aban St. Tehran, Iran.

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Evaluation of the efficacy of vaccination of partridge against H9N2 vaccine

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Low pathogenic avian influenza viruses (H9N2) is circulating in poultry industry of many Euroasian countries causing serious economical problems. In this study we investigated clinical signs, antibody response, viral shedding and efficacy of oil emulsion vaccines in chukar partridges. Seventy five chukar partridges (Alectoris chukar) were divided randomly in three groups of 1-Challenged, 2- Vaccinated and challenged 3-Control (non-vaccinated and non-challenged) groups 25 birds/group. In challenged and vaccinated groups birds were inoculated with 0.4 ml allantoic fluid containing 107 EID50/bird of tA/Chicken/Iran/772/1998(H9N2) avian influenza virus. Clinical signs, antibody response, viral shedding and vaccine efficacy were evaluated and compared among these groups. Clinical signs such as; coughing and sneezing with depression and decreased in feed and water consumption were observed in group one. Also in vaccinated and challenged group slight decrease of food and water consumption were observed. Both vaccinated and challenged groups showed maximum antibody titer at 9 DPI. At 1 DPI the virus was detected from all tissues in challenged group, however the virus wasn't detected from the spleen and cecal tonsil of group vaccinated and challenged group. Unvaccinated challenged groups showed longest period of viral shedding in the trachea and kidney.

Biography

Professor Hassan Nili has completed his Ph.D at Queensland University in Australia and he his study leave research at VLA in UK on highly pathogenic avian influenza viruses. He is currently the head of avian diseases research center of Shiraz University. He has been involved in Influenza research for more than 10 years.

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Workshop on High Throughput Screening and Assay Development

Instructor: G. Sitta Sittampalam
National Institutes for Health (NIH), USA

Chair: Harold C. Smith
University of Rochester Medical Center, USA

Virology-2012

Day: 3

Workshop: NIH Grantsmanship

Instructor: Fatah Kashanchi
George Mason University, USA

Virology-2012

5 (i) : Virology Immunology and Clinical Complications

Session Chair
Qianjun Li
University of Alabama, USA

Session Co-Chair
Lena Al Harthi
Rush University Medical Center, USA

Session Introduction

Title: **The role of tight junction proteins during Dengue virus entry**
Qianjun Li, University of Alabama, USA

Title: **Serological and molecular characterization and diagnosis of viruses expressing similar necrotic symptoms in blackgram and greengram**
Jyothirmai Madhavi K, Acharya NG Ranga Agricultural University, INDIA

Title: **Phenotypic and functional assessment of peripheral blood dendritic cells in HIV/HCV co-infected patients undergoing Interferon/Ribavirin combination therapy**
Mohit Sehgal, Drexel University College of Medicine, USA

Title: **The infectivity and pathogenicity of a foot-and-mouth disease virus persistent infection strain from oesophageal-pharyngeal fluid of a Chinese cattle in 2010**
Dong Li, Lanzhou Veterinary Research Institute, China

Title: **Searching the Human Herpes 6, 7 (PCR) in CSF of children admitt: Cross section studied in pediatric ward of rasoul hospital**
Samileh Noorbakhsh, University of Medical Sciences, Iran

Title: **Polio Vaccination in Nigeria: The 'good', the 'bad' and the 'ugly'**
Marycelin Mandu Baba, University of Maiduguri Teaching Hospital, Nigeria

Title: **A method to distinguish potentially infectious from inactivated human norovirus**
David H. Kingsley, Delaware State University, USA

Title: **What is the role of bacteria in AIDS?**
Vladimir Zajac, Slovak Academy of Sciences, Slovakia

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The role of tight junction proteins during Dengue virus entry

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With more than one-third of the world's population living in areas at risk of infection, dengue disease becomes a huge public health burden around the world. Considerable molecular, biochemical and structural virology information have been accumulated for Dengue virus (DENV), however, our understanding on its entry process remains ambiguous. We reported here that a group of cell surface proteins, including tight junction proteins claudin-1 and occludin, were involved in DENV entry process. In Huh 7.5 cells with claudin-1 or occludin knockdown, the amount of DENV entering into the cells was reduced. Consequently, the virus progeny productions were decreased and DENV-induced CPE/apoptosis were prevented. Furthermore, resuming the expression of claudin-1 in claudin-1-knockdown and occludin in occluding-knockdown cells facilitated DENV entry in respective cells. Using pulldown assay, we showed that claudin-1 interacted with DENV prM/M protein and occludin interacted with the E protein, respectively. Mutations on essential domains in claudin-1 and occludin disrupted such interactions. The critical domain and amino acids in claudin-1 were determined using various deletion mutations and point mutations, especially mutations on the extracellular loops (ECL) of claudin-1 and occludin. Since host and viral factors involving in virus entry are very promising therapeutic targets, and entry inhibitors may provide attractive therapeutics against DENV. Thus, the results reported will provide significant insights of DENV entry mechanisms and will lead, eventually, to the ability to describe completely the entry process and to moderate or inhibit it at will.

Biography

Dr. Qianjun Li received his Ph.D. degree at the age of 27 years at Beijing Agricultural University (1993), and had postdoctoral training at Carnegie Mellon University and University of Florida at Gainesville. He is currently an assistant professor at Division of Infectious Diseases, Department of Medicine, University of Alabama at Birmingham. Dr. Li has published more than 30 papers in reputed journals, and has served on the editorial board of several scientific journals, and on the advisory panels of academic and government institutions, including the USDA and NIH.

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Serological and molecular characterization and diagnosis of viruses expressing similar necrotic symptoms in blackgram and greengram

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In the recent years, viruses causing necrosis, transmitted by thrips has assumed epidemic proportion and became a serious production constraint in blackgram and greengram. Leaf curl disease caused by Peanut bud necrosis virus(PBNV) considered being a major threat. Recently, Tobacco streak virus(TSV) has also been reported to be a cause of leaf curl symptoms paving confusion in field diagnosis to assess the disease incidence. Although both the viruses cause necrosis and are transmitted by thrips, the method of transmission and virus vector relationships vary and hence need different approaches of management practices. Symptomatic leaf samples collected from various locations of Andhra Pradesh, subjected to DAC-ELISA against TSV and PBNV polyclonal antisera. The samples tested positive to PBNV (PBNV-BG, PBNV-GG) and the samples positive to TSV (TSV-BG, TSV-GG) expressed typical symptoms in infectivity tests. The nucleo-capsid protein(N) gene of PBNV-BG&GG and coat-protein(CP) gene of TSV-BG&GG were amplified by RT-PCR yielding a fragment of the expected size, ca.~830bp and ca.~700bp respectively. The determined nucleotide sequences of PBNV and TSV isolates were deposited at GenBank. The sequenced region in PBNV isolates contained a single ORF of 831 bases that could potentially code for a protein of 276 amino acids while TSV isolates contained a single ORF of 717 bases that could encode for a protein of 238 amino acids. Comparative sequence analysis revealed that TSV-BG shared hundred per-cent sequence homology with TSV-GG at nucleotide and amino acid levels, whereas PBNV-BG shared maximum sequence homology with PBNV-GG at nucleotide(99.7%) and amino acid(100%) levels.

Biography

Dr. K. Jyothirmai Madhavi has completed her Ph.D from Acharya N G Ranga Agricultural University, Andhra Pradesh, India and joined Dr. YSR Horticultural University as Assistant Professor(Plant Pathology). She has published seven research papers and one invited paper in peer reviewed journals. She has presented in XX National Conference of Indian Virological Society(IVS) on "Managing Emerging and Re-emerging Plant, Animal, Human and Aquatic Viral Diseases: One Health Perspective", VIROCON-2011 at National Research Centre for Equines(NRCE), Hisar, India. She also presented in "3rd Global Conference: Plant Pathology for Food Security" at Maharana Pratap University of Agriculture and Technology, Udaipur, India.

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Phenotypic and functional assessment of peripheral blood dendritic cells in HIV/HCV co-infected patients undergoing Interferon/Ribavirin combination therapy

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HIV/HCV co-infection represents a significant burden on global economy and public health. It is now widely accepted that HIV accelerates the course of HCV-related chronic liver disease. The current standard treatment for treating HCV infection in HIV/HCV co-infected patients is a combination of pegylated interferon (IFN) and an antiviral drug ribavirin (RBV). This treatment is successful in only 50% of the patients and is associated with significant side effects. Therefore, it becomes necessary to determine the predictive host factors of successful treatment response. Since dendritic cells (DCs) play an important role in orchestrating innate and adaptive immune response against pathogens, we hereby investigate DC-based markers of treatment response to the combination therapy in a cohort of HIV-1/HCV co-infected individuals including non-responders (NRs), sustained virological responders (SVRs) and relapsers. Using a recently developed 13-color polychromatic DC antibody cocktail, mononuclear cells from patients isolated at different time points (baseline, week 1, 2, 4, 24 and 48) during the course of therapy were analyzed for various markers of myeloid and plasmacytoid DCs. We studied the frequency of mDCs ($Lin\text{-}1^-/CD11c^+CD123^-$) and pDCs ($Lin\text{-}1^-/CD11c^-CD123^+$) as well as markers of DC activation (HLA-ABC, HLA-DR, and CD86) and adhesion (CD54 and CD62L). We also analyzed chemokine receptors (CCR5 and CCR7), HIV entry receptor CD4 and death receptor ligand (PDL)-1 on DCs. Statistical analyses of data revealed CD62L and CCR7 to be potential markers of early treatment response. Ongoing studies involve transcriptomics and proteomics analysis of differential response between SVRs and NRs, which is likely to contribute towards effective therapeutic management of HIV-1/HCV co-infection.

Biography

Mohit Sehgal is a 2nd year Ph.D. student in the laboratory of Dr. Pooja Jain who is an Associate Professor in the Department of Microbiology and Immunology, Drexel University College of Medicine (DUCOM), Philadelphia, USA. Mr. Sehgal is studying the role of dendritic cells during HIV-1/HCV co-infection in order to delineate the complex interplay of host-pathogen interaction during two chronic viral infections vis-à-vis IFN/Ribavirin combination therapy.

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The infectivity and pathogenicity of a foot-and-mouth disease virus persistent infection strain from oesophageal-pharyngeal fluid of a Chinese cattle in 2010

Dong Li[1], Hui-Fang Bao[1], Pu Sun[1], Qiang Zhou[1], Jun Hu[2], Xing-Wen Bai [1], Yuan-Fang Fu[1], Zeng-Jun Lu[1] and Zai-Xin Liu[1,2]
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Background: Foot-and mouth disease(FMD) is an acute, febrile, and contagious vesicular disease affecting cloven-hoofed animals. Some animals may become persistent infected carriers when they contact FMD virus(FMDV),and persistent infected animals are a dangerous factor to cause FMD outbreak.

Findings: 300 OP(oesophageal-pharyngeal) fluid samples were collected from cattle without clinic symptom after one month FMD circulated in 2010 in China. A FMDV strain was isolated when a positive OP sample was passed in BHK21 cell line.The strain,named O/CHN/2010/33-OP, was detected to be O/Myanmar/1998 lineage with VP1 DNA sequence comparison.In order to testify its infectivity, two cattle were challenged with OP fluid and three pigs were put into the same pen for direct contact infection.The result showed that one of the cattle and one of the pigs appeared FMD clinic symptoms respectively. Further more, two cattle(three pigs were also put into the same pen for direct contact infection) and three pigs were inoculated with O/CHN/2010/33-OP cell passaged strain. The result showed that one of the challenged pigs appeared FMD clinic symptoms. Two cattle and three pigs in the same pen did not appeared FMD clinic symptoms,but the sera antibody and their OP fluid of two cattle were positive. Meanwhile,the spinal cords of three pigs in the same pen with two cattle were positive detected with multiplex-RT-PCR.

Conclusion: The persistent infection strain O/CHN/2010/33-OP has infectivity and pathogenicity to cattle and pigs, and infected cattle may transmit the virus to pigs although its virulence was lower than the circulated strain O/CHN/Mya98/2010.

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Polio Vaccination in Nigeria: The 'good', the 'bad' and the 'ugly'

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Despite the decline in reported cases of polio by over 99%, Nigeria remains one of the most entrenched reservoirs of wild poliovirus in the world. The "good" the bad" and "the ugly" aspects of polio vaccination in Nigeria is discussed. The 'good' aspect centers around the decline in the number of wild poliovirus cases by over 95%, from 388 in 2009 to 56 in 2011; cVDPV 2 cases declined by 82% , from 154 in 2009 to 57 in 2011. The Immunity to polioviruses has improved in endemic States and areas with persistent polioviruses are better identified and targeted. New approaches to identifying settlements not on the micro plan and to promote community initiatives have been made. On the 'bad 'aspect, polio cases have increased from 21 in 2010 to 56 in 2011 with ongoing transmission of wild poliovirus type1, 3 and cVDPV2. Declined political oversight at critical juncture and non Implementation of emergency plans in key infected areas has been observed. Lastly, "the ugly" aspect focus on the aftermath of the boycott of polio vaccination in three northern States in 2003 due to a report that, the polio vaccine contained infertility drugs capable of sterilizing young girls, causes polio and spread HIV The boycott led to the spread of polio into twenty countries across Africa, the Middle East and South east Asia causing 80% of the world's cases of paralytic poliomyelitis. After resolving the crisis, some parents in the north are still resisting compliance with the polio vaccination.

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A method to distinguish potentially infectious from inactivated human norovirus

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Human norovirus strains cannot be propagated in the laboratory and current detection methods are based on RNA detection methods, such as RT-PCR. Unfortunately RNA-based methods cannot distinguish infectious virions from damaged virions unless the capsid has lost its integrity. In order to infect the host cell, a virus must first bind to its receptor. This fact has been exploited to develop a means of separating potentially infectious virus from inactive virus using virus receptor-like glycoproteins attached to magnetic beads. This extraction method when coupled with RT-PCR extraction should reduce the detection of inactive norovirus virions that are not a threat to public health. The utility of this method for testing of shellfish and other foods is currently being evaluated.

Biography

David H. Kingsley Ph.D. is a Research Virologist within the Food Safety and Intervention Technologies Research Unit of the USDA Agricultural Research Service. His research is focused on foodborne viruses such as norovirus and hepatitis A, particularly as they relate to bivalve shellfish. His primary research efforts are focused on improved detection methods for foodborne viruses contaminating shellfish and developing methods to inactivate or purge viruses from shellfish.

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What is the role of bacteria in AIDS?

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A cluster of evidences has accumulated to date indicating that the main site of HIV infection and CD4+ T cell loss is in the GIT and other mucosal tissue rather than in blood. Thus the pathogenesis of HIV infection is presumably centered on these mucosal viral "target" cells. The HIV-1 was also detected in bowel crypt cells and the lamina propria. Since these cells are in close vicinity to intestinal bacteria, promoted the idea that bacteria may also be involved in the pathogenesis of AIDS. It has also been expressly proven that various forms of HIV reservoirs persist in practically all patients receiving HAART. The recent studies suggest that the palette of viral reservoirs in human body is probably very much wider.

Bacterial DNA isolated from the intestinal tract of American and Slovak HIV/AIDS patients and DNA of bacteria and yeasts isolated from respiratory tract of Cambodian and Kenyan HIV positive children were tested for HIV-1 sequences by PCR using specific primers for gag, pol and env genes of HIV-1. The PCR products sythesized on template of these DNA, were found to be for more than 90% homologous to the corresponding HIV-1 sequences. In Western blotting analysis were in these bacteria identified HIV-like proteins using monoclonal antibodies against HIV-1 antigens p17, p24, gp41 and gp120. Molecular weight of detected proteins are mostly not in accordance with corresponding viral proteins. Differences between profile of detected bacterial proteins by using MAbs against HIV-1 antigens of American and Slovak on the one side and Kambodian and Kenyan patients on the other one, are very probably results of evolutionary process. The presence of HIV sequences in commensally bacteria of the patients may be explained as follows: 1) HIV was transferred into intestinal bacteria from human cells, in particular out of macrophages and lymphocytes; 2) intestinal bacteria are a natural host of HIV sequences in a form of virus or "virus like particles".

Biography

Vladimir Zajac has completed his PhD. in 1982 from the Cancer Research Institute of Slovak Academy of Sciences in Bratislava (Slovakia), where he was from 1996 the head of Department of Cancer Genetics. He joined the Medical Faculty of the Comenius University as Associate Professor of Genetics in 2007. He has published 56 papers mostly in reputed journals and he was editor of the book „Bacteria, viruses and parasites in AIDS process" (InTech).

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5 (ii) : Respiratory and Vector Borne Diseases

Session Chair
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Griffith University, Australia

Session Co-Chair
Reena Ghildyal
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Session Introduction

Title: Novel approaches to reducing inflammation following respiratory virus infections - A focus on respiratory syncytial virus
Suresh Mahalingam, Griffith University, Australia

Title: Identification of novel hit against mutant influenza A virus
Wenhui Hu, Chinese Academy of Sciences, China

Title: Endonuclease substrate selectivity characterized with full - Length PA of influenza A virus polymerase
Erin Noble, University of Rochester, USA

Title: Host targets of respiratory syncytical virus matrix protein
David Jans, Monash University, Australia

Title: Nucleocytoplasmic trafficking of the matrix protein of respiratory syncytial virus: Role in infection
Reena Ghildyal, University of Canberra, Australia

Title: Lactate Dehydrogenase level in nasal wash fluid as a novel marker for respiratory syncytial virus induced bronchiolitis
Sanaa, Zagazig University, Egypt

Title: Clinical factors predictive of pneumonia caused by 2009 H1N1 Flu
Kittisak Sawanyawisut, Khon Kaen University, Thailand

Title: DAS181 for the treatment of influenza and para influenza infections
Ronald Moss, NexBio, USA

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Novel approaches to reducing inflammation following respiratory virus infections - A focus on respiratory syncytial virus

Suresh Mahalingam
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Indiscriminate targeting of cytokines in inflammatory disease has been met with major limitations. We have discovered a means to manipulate key cytokine and chemokine responses to selectively target these responses against inflammation. We demonstrate that small receptor antagonist that target TNF signaling or small molecule inhibitor that target the chemokines MCPs are effective in ameliorating RSV disease in mice. Histological analysis of lung tissues showed a reduction in inflammatory infiltrate in infected mice treated with these inhibitors. Importantly, treatment of mice with these inhibitors does not compromise antiviral immunity. These results suggest that these small molecule inhibitors may be useful in treating RSV-induced inflammation in humans. These findings represent novel approaches to targeting inflammatory processes caused by RSV infection. The findings will be discussed.

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Identification of novel hit against mutant influenza A virus

Wenhui Hu
Guangzhou Institutes of Biomedicine and Health, Chinese Academy of Sciences, China

Although amantadine derivatives are the only M2 drugs for influenza virus A, their use is limited in the U.S. because of drug resistance. We initially identified multiple M2 inhibitors that were rapidly generated through focused screening of a small primary amine library that was designed using a scaffold-hopping strategy based on amantadine. Though these compounds are novel M2 inhibitors and are as active as amantadine, they have no any effect against adamantane-insensitive A/M2 mutants. We further explored the hit and made a series of Pinanamine derivatives, fortunately some of the new compounds were capable of inhibiting WT A/M2 and selected adamantane-resistant M2 mutants. Several imidazole and guanazole derivatives of pinanamine were found to inhibit WT A/M2 well and one of these compounds exhibits inhibition of A/M2-S31N mutan (IC50 = 28.7 μM). Our study may provide a new insight into the structural nature of drugs required to inhibit WT A/M2 and its mutants.

Biography

Dr. Hu completed his Ph.D. in Medicinal Chemistry from the Institute of Materia Medica, Chinese Academy of Medical Sciences in 2002. During 2003-2006, He did the post-doctoral research at the Center for drug discovery and chemical biology, Northwestern University Medical School in the US. He is the Principal Investigator of Guangzhou Institutes of Biomedicine and Health, Chinese Academy of Sciences where he is running multiple drug discovery projects, including anti-diabetic drug discovery, anti-alzheimer's disease drug discovery (anti-neuroinflammatory inhibitors) and anti-influenza A drug discovery (M2 inhibitors). He is the owner of several drug candidates and more than 20 publications in high level journals along with 10 PCT patents.

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Endonuclease substrate selectivity characterized with full - Length PA of influenza A virus polymerase

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The influenza A viral polymerase is a heterotrimer capable of both transcribing viral mRNAs and replicating the viral genome. To initiate synthesis of viral mRNA the virus uses a process known as "cap snatching" wherein the viral polymerase binds a host pre-mRNA and cleaves a short primer with a 5' end cap structure. Essential to this process is the enzymatic activity contained within the PA subunit. The N-terminal domain of PA has been demonstrated to have endonuclease activity in vitro and crystal structures of the PA N-terminal domain reveal a distinct active site. Here we sought to understand the biochemical nature of the PA endonuclease activity using, for the first time, the full-length PA protein. This full-length protein is active against both RNA and DNA in a cap-independent manner and can use several different divalent cations as cofactors. Different metal cofactors induce secondary structure changes, which correlate with cleavage patterns. Our in vitro assay was also able to demonstrate the minimal substrate size and sequence selectivity of the PA protein. Finally, we confirmed the observed endonuclease activity of the full length PA with a FRET based endonuclease assay, which is well suited for screening of novel anti-influenza agents.

Biography

Erin Noble completed her B.S. degree in Microbiology and Immunology in 2007 from the University of Rochester. She is in the process of completing her PhD research in the lab of Dr. Baek Kim at the University of Rochester. Her research focuses on understanding the biochemical nature of the influenza A virus polymerase complex and developing assays to characterize viral biochemical processes.

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Host targets of respiratory syncytical virus matrix protein

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Respiratory syncytial virus (RSV) is the major respiratory pathogen of infants and children worldwide, with no effective treatment or vaccine available. The RSV matrix protein (M) is critical to the virus, playing a key cytoplasmic role in virus assembly late in infection (1,2). Intriguingly, however, it is localized in the nucleus of infected cells early in infection through the action of the importin (IMP) β1 nuclear transporter; prevention of M-IMPβ1 interaction in mutant RSV reduces virus production over 20-fold, indicating that M nuclear localization is critical to the RSV life cycle (3). The role of M in the nucleus appears to be to inhibit host cell transcription, thereby dampening the host anti-viral response, but the mechanism thereof has not been defined. We set out to address this question using a 3-pronged screening approach to look for nuclear targets of M action, through yeast 2-hybrid screening and proteomic approaches, and subsequent assessment the importance of the cellular factor to RSV infection by siRNA knockdown. A range of potential targets of M were identified, and are beginning to be validated, our initial results indicating the power of our 3-pronged screening approaches.

(1) J. Virol. 82, 8863-8870 (2008); (2) Infectious Disorders-Drug Targets 12, 103-109 (2012); (3) J. Virol. 83, 5353-5362 (2009).

Biography

Prof. Jans completed his Ph.D at the age of 25 years at the Australian National University (Canberra) and postdoctoral studies at the Friedrich Miescher Institut (Basel, Switzerland) and Max Planck Institut fuer Biophysik (Frankfurt am Main, Germany). He is presently an NHMRC Senior Principal Research Fellow (SPRF1) and Head of the Nuclear Signalling Lab. at Monash University (Melbourne, Australia). His research over the last 16 years has focused on the regulation of transport into and out of the eukaryotic cell nucleus, and how this relates to viral disease, cancer and development, and how it may be exploited for drug delivery. He has >240 peer-reviewed publications in eminent journals (> 7000 citations; H-factor of 54), has served as committee member of the International Photodynamic Association (2005-2011), and currently serves as an editorial board member of Biochemical Journal (since 2006), Biochem Biophys Acta Mol Cell Res (since 2010) and Antiviral Res (since 2012).

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Nucleocytoplasmic trafficking of the matrix protein of respiratory syncytial virus: Role in infection

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Respiratory syncytial virus (RSV) is the major respiratory pathogen of infants and children worldwide, with no effective treatment or vaccine available. Within all paramyxoviruses, cytoplasmic matrix protein (M) plays a key role in assembly of new virions. Intriguingly, M localizes to the nucleus of infected cells early in infection where it inhibits host cell transcription, thus promoting viral transcription and pathogenesis. Nuclei of RSV-infected cells are deficient in transcription correlating with M nuclear localization and recombinant M is able to bind to DNA, RNA and inhibit in vitro transcription. Using in vitro and transfected cell systems, we have shown that M localizes in the nucleus through recognition of its nuclear localization signal (residues 155-172) by the importin β1 nuclear transporter. M's ability to shuttle to the cytoplasm is through the action of the nuclear export receptor Crm1 largely via a nuclear export signal within residues 196-210. M's nucleocytoplasmic transport is regulated, at least in part, by CK2 phosphorylation at two positions, S95 and T205; mutation of both residues to alanine results in a loss of regulated nuclear transport. That nucleocytoplasmic trafficking of M is critical to RSV infection is indicated by the fact that recombinant RSV with M mutated to either block nuclear entry or nuclear export is replication-deficient compared with wild-type virus.

Biography

Reena Ghildyal obtained her PhD in Life Science from Jawaharlal Nehru University, New Delhi, India. This was followed by postdoctoral fellowships at the State Department of Agriculture, and Macfarlane Burnet Institute in Melbourne, Australia. In 2004 she was offered the opportunity to establish a respiratory virology research group as part of a new Chinese Government initiative within Fudan University, Shanghai. On her return to Australia in 2007, she has continued her association with Fudan University and has been instrumental in establishing several bilateral research collaborations between Chinese and Australian scientists. In 2010 Reena was appointed Assistant Professor at the University of Canberra, establishing the viral laboratory.

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Lactate Dehydrogenase level in nasal wash fluid as a novel marker for respiratory syncytial virus induced bronchiolitis

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Respiratory syncytial virus bronchiolitis is an important cause of wheezy chest in infancy and can be lifethreatening.

Nasal wash lactate dehydrogenase (NWLDH) which is released from injured epithelial cells may be used for prediction of disease severity. The aim of this study was to assess the use of NWLDH versus serum LDH, IL-6 and TNF- α in the evaluation of RSV bronchiolitis severity in infants. A total of 55 infants older than 6 months old who presented with bronchiolitis were prospectively enrolled in the study. Nasal-wash samples were analyzed to detect RSV by polymerase chain reaction and quantify LDH concentration and serum samples to quantify IL-6, TNF-α and LDH concentrations. The median concentrations of serum LDH and NWLDH were significantly higher in infants with severe than were those with moderate respiratory disease (p=0.002, 0.0001 respectively) while no significant difference was observed according to IL-6 and TNF-α. However; SLDH, NWLDH and IL-6 levels presented significant positive correlations with disease severity and the length of hospital stay, also between NWLDH and IL-6 with duration of oxygen therapy. While; TNF-α presented a significant positive correlation with disease severity only. There was a significant positive correlation between NWLDH and S.LDH ($r = 0.7$, $p < 0.0001$). The measurement of LDH in nasal wash rather than serum LDH, IL-6 and TNF-α is more practical for monitoring the severity of RSV bronchiolitis in infants.

Keywords: Bronchiolitis, LDH, IL-6, TNF-α, infants, nasal wash.

Biography

Sanaa Mahmoud had taken Bachelor Degree at Faculty of Medicine, Zagazig University, Zagazig, Egypt (December 1997), Passing Master Degree on May 2002, Passing M.D. Degree on April 2007.

Sanaa Mahmoud is a lecturer in Pediatric Department, Faculty of Medicine, Zagazig University during the period from September 2007 till now.

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Clinical factors predictive of pneumonia caused by 2009 pandemic H1N1 influenza virus

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Pneumonia is the most common cause of death in 2009 H1N1 Flu (H1N1) infection. Clinical risk factors for pneumonia caused by this virus are still limited. We enrolled consecutive patients treated at H1N1 clinic, Thungsong hospital, Thailand from June to December 2009 who had positive PCR test for H1N1. Clinical features between patients diagnosed with and without pneumonia were studied. There were 441 patients with positive PCR test for H1N1. Of those, 51 patients had pneumonia (11.56%). Six clinical factors predictive for pneumonia including headache, myalgia, having underlying disease, body temperature of more than 39°C, absolute neutrophil count > 7,700 cells, and serum creatine phosphokinase (CPK) more than 200 U/L. The adjusted odds ratio (95% CI) for all six variables were 0.432 (0.193-0.967), 0.400 (0.174-0.922), 3.095 (1.435-6.675), 2.770 (1.326-5.786), 4.432 (1.944-10.101), and 2.232 (1.040-4.789), respectively. Clinical features may be a useful tool for clinicians to predict risk of pneumonia from H1N1.

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DAS181 for the treatment of influenza and para influenza infections

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DAS181, a novel inhaled sialidase fusion protein, has shown in vitro and in vivo activity against many subtypes and strains of influenza virus and parainfluenza virus by inactivating the virus binding receptors. Preclinical studies have demonstrated antiviral activity against multiple strains of influenza including 2009H1N1, H275Y resistant virus, and H5N1. The drug has also demonstrated preclinical antiviral activity against parinfluenza virus. Phase 1 clinical studies have been completed without safety concerns and the drug was well tolerated. A recent phase 2 in influenza-infected individuals demonstrated the safety of this approach as well as a statistically significant effect on decreasing viral load. A number of immunosuppressed transplant patients with potentially fatal parainfluenza infection have been studied under emergency IND's with good clinical outcomes. Results from clinical studies of DAS181 in both influenza and PIV patients will be presented. DAS181 represents a novel host-directed influenza and parainfluenza treatment approach. Studies are ongoing to confirm the safety and activity of this approach for pandemic preparedness and to meet unmet medical needs.

Biography

Dr. Moss trained in the Laboratory of Clinical Investigation at the National Institute of Allergy and Infectious Disease and has been involved in clinical trials for over twenty years. He is a practicing physician, boarded in Allergy/Immunology and Pediatrics. He has been involved in the execution of successful phase 1-3 trials in both pediatric and adult populations in the U.S., Europe, Asia, Africa, and South America. Dr. Moss is the author of over 70 peer reviewed publications.

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Intestinal parasitic infections in HIV-positive individuals on HAART and HAART naïve accessing healthcare in a Federal Medical Centre in Nigeria

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Intestinal parasitic infections (IPIs) in HIV-positive individuals have been widely reported as a significant cause of morbidity and mortality all over the world. However, IPIs in such individuals have not been studied in this part of Nigeria. This study was therefore a baseline survey of the prevalence of intestinal parasites among HIV positive individuals in Central Nigeria. A total of 200 consenting HIV positive individuals were recruited for the study. Of these, 100 were on Highly Active Antiretroviral Therapy (HAART) and 100 were HAART naïve. Stool specimens collected were analyzed for the presence of enteric parasites. The overall prevalence of infection was 88.5% with 11 types of parasites detected. IPIs that occurred with a prevalence of ≥ 20% include: Entamoebahistolytica, Ascarislumbricoides, Ancylostomaduodenales, TaeniasppandStrongylidesstercoralis. Helminth infections were more common (51.5%) than protozoan infections (37%) and there were more mixed (64.5%) than single (23%) infections. The prevalence of IPIs was not associated with HAART. This study reports the high burden of IPIs among HIV infected individuals in Central Nigeria. It is recommended that routine screening for intestinal parasites be included in the healthcare management of HIV patients.

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Prevalence of Toxoplasma, Cytomegalovirus(IgM), Rubella(IgM), Hepatitis C Co-infection and Bactermia in newly diagnosed and treatment naive HIV- infected patients attending Apin clinic at Lagos university teaching hospital- a pilot study

Adeleye I.A, Adeoye G.O, Bamiro S.O, Akanmu S, Adepoju A and Opara
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This study determined the prevalence of Toxoplasma,Cytomegalovirus(IgM),Rubella(IgM), Hepatitis C co-infection and bacteremia among newly diagnosed HIV positive patients attending APIN clinic at Lagos University Teaching Hospital.

Random peripheral blood samples were collected from 80(28 males and 52 females) patients who were newly confirmed to be HIV positive and were enrolled in the antiretroviral therapy clinic. The patients have not commenced their antiretroviral treatment although most have been treated for febrile symptoms and weakness. Sera samples were screened for anti Toxoplasma antibodies,Cytomegalovirus(IgM),Rubella(IgM) and Hepatitis C surface antigen using fourth generation ELISA kits. Seventy of the blood samples collected from 24(34.4%) males and 46(65.7%) females were cultured by standard methods for the isolation of blood steam pathogens. All the sera samples(100%) tested positive for Toxoplasma immunoglobulin G while 74(92.5%) were positive for Cytomegalovirus immunoglobulin M. Only one serum sample was positive for IgM against Rubella virus. Seven sera(8.75%) comprising of 3 males and 4 females were positive for Hepatitis C(HCV) antibodies. Five staphylococci comprising of 2 Staphylococcus warneri,1 Staphylococcus epidermidis , Staphylococcus xylosus, Staphylococcus cohnii cohni as well as 2 Pseudomonas aeruginosa were isolated from the blood cultures of the patients.

Although this is a pilot study, the extremely high prevalent rate of Toxoplasma and Cytomegalovirus IgM antibodies suggests a strong association between HIV, Toxoplasma and Cytomegalovirus infections in this environment and should be a major consideration in the initiation and choice of antiretroviral therphy. HCV co-infection rate is consistent with earlier studies conducted in this environment. Presence of bacteremia suggest depressed immune state of some of the patience.

Biography

Professor Adeleye completed his Ph.D some 21 years ago and has lectured in the Polytechnic and Universties in Nigeria for about three decades. He has up to 40 published articles and his current research is on Blood stream infections of retroviral naïve HIV patents.

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High DNA HTLV-1 proviral load among TSP/HAM patients: A potential marker for disease progression

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Institute of Infectious Diseases Emilio Ribas, IIER - Brazil

Background: Human T-cell lymphotropic virus type 1 (HTLV-1) infection can increase the risk of developing neurological disorders. This study evaluated the correlation between HTLV-1 DNA proviral load among HTLV-1-infected individuals with HTLV-1-associated myelopathy (HAM/TSP). Material and Methods: In the last 15 years, one cohort of HTLV-infected subjects has been followed in the HTLV-outpatient Clinic at the Institute of Infectious Diseases "Emilio Ribas" (IIER). The results of the first HTLV-1 proviral load were available in the database. Quantitative proviral DNA levels were detected by a real-time automated PCR method, using TaqMan probes for the pol gene. The albumin gene served as the internal genomic control, and MT2 cells were used as a positive control. The results are reported as copies/10000 PBMCs, and the detection limit was 10 copies. Results: From this cohort, 60 HAM/TSP patients were studied. In accordance to the DNA HTLV-1 proviral load results were divided in four intervals: < 10 copies/104 PBMC=; 12 (20%) patients; 11-50 copies/104 PBMC= 5 (8%); 101-500 copies/104 PBMC=; 22 (36%); and >501 copies/104 PBMC= 21 patients (35%) p≤0,001. We observed a significant increased in proviral load in patients with HAM/TSP, showing this parameter as a potential marker for disease progression.

Biography

Adriele S. Fontes was graduated in Biomedicine from the Integrated College Aparicio Carvalho, Porto Velho, Rondonia city. Actually is Master´s Student in Tropical Medicine and Health International at the IMTSP-USP.

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Study on the functional role of immunoglobulin E as surrogate marker for HIV/AIDS infection

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King Institute of Preventive Medicine and Research, India

Certain viral infections are known to produce specific IgE antibodies that significant changes in the level of total serum IgE may occur. Study attempts to associate the Level of IgE in HIV progression.

The study involves fifty HIV seropositive patients attending ART Centre, Department of Sexually Transmitted Disease, Rajaji Government Hospital, Madurai, India subjected for the present study. The individual involves 27 HIV/ AIDS Male patients, 23 HIV/ AIDS Female patients. The control sample comprises 15 HIV sero negatives. The samples were collected at the informed consent of the patients. Serum sample were collected and IgE was quantified using MAGIWELL IgE quantitative solid phase Enzyme- linked Immunosorbent assay (ELISA).

The study documents highest percentage of deviation from the control observed in Male HIV seropositives (43.7%) and age-wise influence documents highest percentage of deviation in the age group 15- 29 years (56%).

Serum IgE level in the study found to be elevated from the normal range documents the existence of imbalance between Th 1 and Th 2 and associated with T-cell dysfunction and a hypergammaglobulinemia. The results suggest that elevation of circulating IgE levels may be due to specific IgE directed to the HIV virus rather than as a result of a nonspecific phenomenon. It indicates that total IgE is increased during the early stages of disease and this elevation appears to be independent of CD4 counts and is not correlated with the levels of other immunoglobulins, suggesting an important role for IgE as a surrogate marker of disease progression
Keywords: Reaginic antibody, Hypersensitivity, Hypergammaglobulinemia.

Biography

Mr. S.U.Baalaji is pursuing his master's in Molecular Virology at King Institute of Preventive Medicine and Research and had done his bachelor's in Biochemistry at The American College affiliated to Madurai Kamaraj Univesity. During this course of time he had done a short term project on "Effect of Tamarindus indica L. extracts on DNA damage studies" and presented a paper on "Recombinant DNA technology in monoclonal antibodies" at the national level seminar organized by Kamaraj College of Engineering and Technology and a presentation on Botulinum Toxin. He is also an active member of the European association of Cancer Research

Ms. Vaishnavi.B is pursuing her master's in Molecular Virology at King Institute of Preventive Medicine and Research and had done her bachelor's in Biotechnology at SRM Arts and Science College affiliated to University of Madras. Presented a paper in National conference on biotechnology intervention for conservation and sustainable utilization of Bioresources (BICSUB) conducted by Sathyabama University. Actively participated in National Conference on "Molecular Perspectives and Therapeutic Strategies for Influenza" organized by King Institute of Preventive Medicine and Research and Influenza Foundation of India

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Dengue virus type 1 from field-caught vectors and humans in brazil: Phylogeny reveals different lineages of the American African genotype in 25 years

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Dengue viruses (DENV) replicate alternately on the mosquito vector (mainly Aedes aegypti) and human host. In Brazil, dengue became a major public health problem after DENV-1 introduction in 1986 in Rio de Janeiro and in 2009, this serotype re-emerged causing major epidemics in the country. Since then, a virological and entomological program was established for monitoring DENV in human sera and vectors and it has constituted an important tool for dengue epidemiology and vector-virus-host interactions studies. DENV-1 was identified by virus isolation and RT-PCR during the 1986, 2001 and 2010 entomological surveillances performed in Rio de Janeiro (RJ) and Roraima (RR) and the Real Time qRT-PCR detected 1.6x104 copies/mL of DENV-1 in the macerate of a single Ae. aegypti female naturally infected. The phylogeny demonstrated that DENV-1 isolated from both field-caught vector and humans belong to genotype V (Americas/Africa), although the co-circulation of two distinct lineages (lineages II and III) was detected. The use of molecular techniques combined to virus isolation showed to be important approaches for the surveillance and molecular characterization studies of DENV from field-caught vectors. The molecular characterization showed sequence differences lineage-specific, independently in which host the virus was isolated. Moreover, we also determined the viral titer in a single Ae. aegypti female naturally infected.

Biography

Marcia de Castro is a PhD researcher on the Transmissores de Hematozoarios Laboratory, a reference laboratory for dengue entomological surveillance, for the Brazilian Ministry of Health. Her expertise areas compromises Parasitology, focusing on the entomology and malacology of parasites and vectors, mainly on mosquitoes and dengue viruses surveillance by using molecular biology techniques.

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An improved method to detect bacterial endotoxin in vaccines using the LAL cartridge system

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The bacterial endotoxin test is required for all parenteral drugs including vaccines. Limulus Amebocyte Lysate (LAL) derived from the horseshoe crabs is used in the endotoxin test and a variety of LAL assay options are available. The kinetic turbidimetric assay (KTA) and the kinetic chromogenic assay (KCA) are popular quantitative methods. However, these methods require a standard curve using a Control Standard Endotoxin (CSE) in each test, which is influenced by the vortexing time of CSE and by technical ability of each analyst. In this study, we intended to check a suitability of automated cartridge system, based on a kinetic chromogenic LAL as a new method to detect endotoxin in vaccines. A significant feature of this new system is the use of an archived standard curve that provides pre-calibrated reference to the reference standard endotoxin. Variability associated with standard curves generated with liquid reagents and incubating microplate is eliminated. The method validation study was conducted on this new LAL cartridge system according to ICH guideline. All validation parameters including accuracy, precision, specificity and linearity were checked and satisfied the acceptance criteria. And we also conducted the comparative analysis between new LAL cartridge system and the existing methods, KTA and KCA. As a result, the LAL cartridge system is considered to be proper method to detect endotoxin in vaccines. Moreover this technique has several advantages like convenience, simplicity of use and speed of result output. Hence the LAL cartridge system will be able to be used for endotoxin test in vaccines.

Biography

Jong-Mi Lim has completed her master degree(M.D) from University of Seoul. She is working for Korea Food and Drug Administration as a scientific official.

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Preliminary evaluation of the immunoenhancement of Newcastle Disease (ND) vaccine formulated as a cationic liposome

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This study evaluates the enhancement of immune response of birds to ND vaccine encapsulated in 1,2-dioleoyl-3-trimethylammoniumpropane (DOTAP) -based liposomes. The vesicles of the liposomal ND vaccine were physically characterized for shape, particle size and zeta potential.

Sixty experimental birds were divided into unvaccinated group, liposomal ND vaccine group and live La Sota® vaccine group. Both liposomal ND vaccine and the live La Sota® vaccine groups were vaccinated orally at three weeks and six weeks of age. The mean antibody titres, total and differential white blood cell count, and blood chemistry respectively were assessed. Ten birds from each group were challenged by òrally administering 0.2 ml of the virulent Herts 33 strain at 9 weeks of age. From the results, vesicles of the liposomal ND vaccine were spherical and tightly packed. Mean size distribution was below 100 nm. The mean zeta potential was 24 mV. The unvaccinated group yielded no antibodies to ND virus. The log2 of mean antibody titre of the birds induced by liposomal ND vaccine after secondary immunization was 9.60 + 0.95 while that of the live La Sota® vaccine was 6.00 + 0.63. Nine of the ten challenged birds in the unvaccinated group died while none died from the liposomal ND vaccine group or the marketed La Sota® vaccine group. It could therefore, be inferred that encapsulating ND vaccine in DOTAP-based liposome significantly caused higher immunity in the experimental chickens than the marketed La Sota® vaccine.

Biography

The author is a teaching staff of the University of Nigeria, Nsukka. She obtained her Ph.D in 2011 and is applying for post doctoral position. She has been teaching for four years and is still an early investigator in the field of viral vaccines. She has attended two conferences in the Baltimore and Chicago in 2008 and 2009 respectively on vaccines development. In 2010, she was sponsored by the Royal Society, London to attend a discussion meeting on New Vaccines for global Health. These conferences have broadened her vision and scope in the field. She has also been exposed to international scientists from all over the world and cross-fertilized ideas that would help her in her near and future targets. Her work has been accepted and published in local and international journals on adjuvanticity of liposomes and niosomes in vaccine delivery.

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Use of a statistical algorithm to classify influenza infection by lung and plasma cytokine and chemokine production profiles

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Influenza epidemics result in approximately 3 - 5 million cases of severe disease and 250,000 to 500,000 deaths annually. Rapid classification of an influenza infection is of utmost importance in determining the proper treatment regimen. In this study, mice were infected with one of three strains of influenza, 2009 swine-origin influenza A (H1N1) A/California/04/09, seasonal H1N1 A/Texas/36/91, and the highly pathogenic avian (H5N1) A/Vietnam/1203/0 or vehicle control. Levels of thirty seven cytokines and chemokines in lung (6, 12, 24, 72 and 96 hr) and plasma (24 and 96 hr) were measured for 4 days following challenge and analyzed statistically using a support vector machine, a statistical concept using supervised learning methods for classification and regression analysis. Using this method, at a given time point post-challenge, only 2 or 3 cytokines in lung or plasma were needed to predict the influenza type with 100% accuracy at a given time post-infection. Since the exact time of influenza infection is seldom known, the ability of the support vector machine procedure to classify the type of influenza infection independent of the time post-infection was also tested. Combinations of 10 lung or 14 plasma cytokines/chemokines were able to provide a 100% classification accuracy of the influenza type over a 96 hour period post-infection.

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Cell specific gene targeting to the CNS using engineered lentiviruses

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Viral-mediated gene targeting to specific cells and organs is a major challenge for establishment of an efficient gene therapy regimen. In this study we manage to develop a gene delivery platform into cells using lentiviral vectors that are pseudotyped with a sindbis mutated envelope and a single chain variable fragment (scFv). It combines unique features of self-inactivated lentiviruses that promote stable gene delivery into non-dividing cells and efficient display of single-chain variable region human fragments (scFv) or soluble IgG on the surface of viral particles. In vitro, cells that express two versions of the receptor-binding domain of the SARS CoV spike glycoprotein were targeted by engineered sindbis pseudotyped lentiviruses that incorporate specific scFvFc attachment moieties. Despite high similarity of the two S1 antigens, gene transfer was obtained with low background of transduction levels, indicating high affinity of the scFvFc to their cognate antigen. Additionally, in vitro targeted gene expression to primary astrocytes was also demonstrated, using engineered lentiviruses that incorporate GLAST IgG. In vivo, lentiviral targeting of astrocytes and oligodentrocyes progenitor cells (OPCs) that express the chondroitin sulfate proteoglycan, NG2 was obtained using viral particles that display an anti-GLAST and anti-NG2 IgG, respectively. Overall, these results demonstrate efficacy of lentiviral vectors as a gene delivery platform. Such a system could potentially be used to mark specific cells populations enabling efficient fating and imaging studies during CNS development, as well as enhance the understanding of the molecular mechanisms that mediate cell communication in healthy and diseased brain.

Biography

Michael Fassler received his M.Sc degree in 2010 from the Department of Virology, Faculty of Health Science, Ben-Gurion University of the Negev, Beer-Sheva, Israel. He is currently completing his Ph.D. degree in the Department of Virology, Faculty of Health Science, and in the Department of Physiology and Neurobilogy, Ben-Gurion University of the Negev. His research work involves developing a gene-targeting platform using lentiviruses that can mediate specific gene delivery into cells of the CNS.

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The latest outbreaks of Lassa Fever Virus in Nigeria

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Lassa fever virus is the microbial pathogen associated with the outbreaks of Lasser fever, a zoonoses disease. The virus is arenavirus, a genus in arenaviridae family. The enveloped virus with RNA genome causes persistent, asymptomatic infection in rodent vector, Mastomys natalensis but sever symptoms in an infected person. Humans contract the disease through inhalation of contaminated air droplets, blood contact with infected individuals or materials, consumption of food contaminated with excretions from infected rodents and eating uncooked or under cooked infected rat meat. The infection in man is characterized by pathologies of gastrointestinal and respiratory tracts, conjunctivitis, mucosal bleeding, cardiovascular and neurological disorders. Lassa fever is endemic in West African countries including Liberia, Sierra Leone and Guinea. The infection has been reported in Senegal, Mali, Central African Republic and Congo Democratic Republic. Through international travels, the disease has been exported to United States of America, Canada, United Kingdom, Israel, Japan and Netherlands. Sub Sahara Africa is natural habitat of the vector rodents. It has been reported that 300,000 to 500,000 cases of Lassa fever out breaks with 5000 deaths occur annually in the sub region. In Nigeria including Jos 1970, Zonkwua from 1974 to 1977, Abo Mbaise and Owerri, 1985, Epkoma, 1990 to 1992, Lafiya 1992 and Abakaliki 2005, 2007, 2009 and 2011. The 2012, Lassa Haemorrhagic fever outbreak in 12 States of Nigeria claimed not fewer than 51persons lost their lives from 8,500 cases reported. Enzyme Linked Immunosorbent assay (ELISA), Immunofluoresence assay (IFA) and Polymerase Chain Reaction (PCR) techniques were used to detect viral antigens, its nucleic acid or specific antibody from pathological specimens. Ribavirine was the most effective drug used for treatment of Lasssa fever cases. Isolation of patients, barrier nursing, contact tracing, control of the vectors and adequate disposal of infectious wastes were effectively employed to prevent and control the outbreaks.

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Viremia, immune status and demographic factors associated with disease severity during two epidemics of DENV-2 in Rio de Janeiro, Brazil

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The state of Rio de Janeiro was marked by extensive dengue epidemics, resulting from introduction of DENV-1 in 1986, DENV-2 in 1990, DENV-3 in 2000 and DENV-4 in 2011. Since the introduction of DENV-2 American/Asian genotype, two additional outbreaks occurred in 1990 and 2008. The 2008 epidemic was considered the greatest in magnitude in Brazil in number of cases, disease severity and high case-fatality rates. Considering the distinct epidemiological features of 1990 and 2008 DENV-2 epidemics, we investigated virological, immunological and demographic factors as a possible determinant to the pathogenic pattern of 2008 epidemic. The level of plasma dengue viral load was assessed in 102 DENV-2 cases from 1990 and 2008 epidemics using qRT-PCR. Results were correlated with the following variables: disease severity, days of illness, age, gender and immune response. In our cohort, no statistical correlation with level of viremia versus age, days of illness and immune status was found in samples from both epidemics. However, plasma viral load of cases from 2008 (7,10x1006 RNA/mL) were higher than those of 1990 (4,70x1004 RNA/mL), p = 0.001. Sequencing analysis of samples from both epidemics confirmed the American/Asian genotype in samples from 1990 and 2008 and identified a new lineage of DENV-2 in 2008. This study demonstrated that in 2008 epidemic high levels of virus were associated with disease severity. The detection of a new lineage of DENV-2 in 2008 deserves more studies to investigate whether this lineage is more virulent and have contributed to the pathogenic profile of 2008 epidemic.

Biography

Priscila Nunes completed her Mastership in 2012 at the age of 25 and currently is a PhD student at the Flavivirus Laboratory, Oswaldo Cruz Institute, Regional Reference Laboratory for Dengue and Yellow Fever Diagnosis, for the Brazilian Ministry of Health. She has experience on molecular biology techniques and currently works on dengue viruses detection by implementing new methodologies.

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Differential proteomic analysis of purified dengue virus obtained from HepG2 cells infected with varying multiplicity of infection

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With the prevalence of antiviral therapy in the developed world, many HIV-1-infected people die of diseases other than AIDS. One of the emerging major causes is cardiovascular disease, leading to the prediction that the majority of HIV-1 patients are expected to develop cardiovascular complications.Endothelial dysfunction is thought to be a key event in the development of cardiovascular diseases, particularly atherosclerosis. Assays testing the effect of HIV-1 on

endothelial activation shows that direct contact with HIV-1 infected T cells enhance endothelial cell activation to a greater extent than HIV-1 alone, suggesting an intracellular HIV-1 protein is responsible for endothelial activation. The HIV-1 viral protein Nef, which is responsible for T cell activation and maintenance of high viral loads in vivo, has been shown to mediate its own transfer to bystander cells. We demonstrate here for the first time that Nef induces nanotube-like conduits connecting T cells and endothelial cells. We also show that Nef is transferred from T cells to endothelial cells via these nanotubes, and is necessary and sufficient for endothelial cell activation. Moreover, we show that SIV-infected macaques exhibit endothelial Nef expression in coronary arteries. Nef expression in endothelial cells causes endothelial apoptosis, ROS and MCP-1 production. Interestingly, a Nef SH3 binding site mutant abolishes Nef-induced apoptosis and ROS formation and reduces MCP-1 production in endothelial cells, suggesting that the Nef SH3 binding site is critical for Nef effects on endothelial cells. Nef induces apoptosis of endothelial cells through an NADPH oxidase- and ROS-dependent mechanism, while Nef-induced MCP-1 production is NF-kB dependent. Taken together, these data suggest that Nef can mediate its transfer from T cells to endothelial cells through nanotubes to enhance endothelial dysfunction in vivo. Thus, Nef is a promising new therapeutic target for reducing the risk for cardiovascular disease in the HIV-1 positive population.

Biography

Ting Wang is a third year Ph.D student from Indiana University School of Medicine.

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Transcription interference networks coordinate the expression of pseudorabies virus genes

Zsolt Boldogkoi and Dora Tombacz
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The genomic structure of herpesviruses shows a modular organization whose basic units are the convergently oriented nested gene clusters each with coterminal 3'-ends. In this study, we report the detection of a genome-wide expression of antisense non-coding RNAs from the genome of an alpha-herpesvirus called pseudorabies virus. We put forward the Transcription Interference Network (TIN) hypothesis in an attempt to explain the genomic design and existence of the antisense RNAs in a common interpretation framework. The TIN hypothesis suggests the existence of a novel genetic regulatory layer, which controls the cascade of herpesvirus gene expression at the level of the transcription. According to our model, the genes and gene clusters mutually inhibit each other's transcription through the collision of their transcriptional machineries at the various overlapping transcription units. The TIN might represent a mechanism, which plays a central role in the programmed step-by-step switches of transcription between kinetic classes and subclasses of viral genes. The proposed model may be not restricted to the herpesviruses, but might explain the mechanism of an important regulatory system existing in other organisms belonging to various phyla.

Biography

Zsolt Boldogkői received his Ph.D. degree (1999) in molecular biology from Szent Istvan University at Gödöllő and had post-doctoral training at University of Bonn. He received his DSc degree (2008) at University of Szeged. His primary field of interest is the molecular biology of herpes viruses with special emphasis on the regulation of gene expression analysis and utilization of herpesviruses as tools in various fields of biology including neurobiology and cardiology. He has published more than 60 papers in reputed journals. Currently, Zsolt Boldogkői is the head of Department of Medical Biology at Faculty of Medicine of University of Szeged.

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MicroRNAs expression of Ascocenda Orchid under stress of cymbidium mosaic virus infection

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MicroRNAs (miRNAs) are the endogenous small non coding RNA (~21 nt), which play an important role to regulate gene expression in growth development and defense mechanism systems. Although a number of orchid species are always susceptible to Cymbidium mosaic virus (CymMV) infection, there is still no data available for CymMV defense mechanism in orchids regarding to miRNAs. To get insight into miRNAs profile of Ascocenda orchid infected with CymMV, two miRNA libraries were constructed from non-infected (AscoF) and CymMV infected (AscoV) Ascocenda and were generated by high through-put sequencing. With the availability of Illumina sequencer, a total of 8,811,192 clean reads consisting of 4,325,140 unique sequences ranging in size between 18-30 nt were provided from AscoF and AscoV libraries. After bioinformatics prediction and molecular analysis of Ascocenda miRNA libraries, twenty three conserved miRNA families and one precursor secondary structure were discovered. Moreover, miRNA target identification and classification showed 100 candidate target transcripts belong to several gene families with diverse biological functions. Obviously, the AscoF and AscoV presented different pattern of miRNA expression which related to growth development and defense mechanism systems in particular miR156, miR162, miR164, miR172, miR528 and miR529. In this report, we have identified by first time the conserved miRNAs presenting in Ascocenda orchid comparing with published Arabidopsis and rice miRNA libraries. Understanding from these experiments will useful to provide the growing database of orchid miRNAs, which can be the tool to generate genetic improvement of viral infection tolerance.

Biography

Udomporn PETCHTHAI obtained her Bachelor's Degree in Biology from Kasetsart University (KU) in 2007. Since 2008 she has received a scholarship "The Royal Golden Jubilee PH.D Program" from Thailand Research Fund (TRF) to fulfill her dissertation. Her research interest covers RNAi technology in orchid plants. She gained one year research experience at University of California at Riverside (UCR) in Prof. Shou-Wei Ding's laboratory to study about RNAi mechanism in Arabidopsis and constructing cloning of orchid miRNA libraries.

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Presence of HPV in saliva samples of HIV-1-infected subjects

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Human Papillomaviruses are the most prevalent sexually transmitted diseases with more than 200 HPV types have been described. Immunedefiency individuals have a higher risk for HPV acquisition, such as HIV-infected subjects. The aim of this study was determine the prevalence and persistence in the oral tract of HIV-1-infected men subjects who have been following up at the out clinic HIV - HC/FMUSP in São Paulo city. From november 2009 to March 2011, 28 saliva samples were analyzed, three had HPV DNA, corresponding to a prevalence of 10%. In July 2011, the three positive samples were re-tested and low risk HPV types (62, 40 and 11) were disclosed. In january 2012, among three HPV-positive subjects, one remaing positive after three years of follow up. Thus, we concluded that HPV persistence was seen in 1/3 of the patients studied. The HPV monitoring is important since these patients may have potential for oral transmission.

Biography

Karen Gaester was graduated in Biomedicine from the UNINOVE, during her under-graduation she had scholarship from Fapesp, and currently she is a Master´s Student in Tropical Medicine and Health International at the IMTSP-USP.

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Enhanced microarray-based detection of human enteric viruses using cDNA targets

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Human enteric viruses are commonly recognized principal agents for foodborne and waterborne diseases world-wide. They are inherently group of viruses that usually confer similar or overlapping clinical symptoms and pose a challenge for correct diagnosis. DNA microarray technology has emerged as a promising tool for effective broad-spectrum detection of viral pathogens including enteric viruses. However, one of the critical limitations of this powerful technology for pathogen detection is that a relative large amount of sample genetic materials is required for microarray analysis; therefore, their detection power can be limited by the amount of genetic materials available. Herein, we examined the suitability of a linear isothermal amplification method for the application of microarray-based detection and identification of enteric viruses. This method was able to amplify target RNA and produce single stranded cDNA as the end product in approximately four hours from nanogram quantities of input total RNA. We performed a series of tests using starting amounts of viral RNA ranging from 0.06 ng to 22 ng to assess amplification yield, analytical sensitivity and reproducibility, as well as fidelity. We demonstrated that as little as 0.06 ng of viral RNA could produce enough material using the amplification protocol for successful identification by microarrays without compromising detection specificity. Pairwise comparison of technical replicates hybridized to microarrays by regression analysis showed excellent consistency in appropriate sensitivity range. We also showed that the use of amplified material offered increased microarray detection accuracy over complementary RNA generated by traditional in vitro transcription (IVT) amplification method. The improved performance appeared to be associated with reducing microarray cross-hybridization using cDNA. Our results suggest that this method is a useful alternative approach for preparing targets from limiting amounts of material in a rapid, sensitive and unbiased manner for microarray analysis.

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Association of HTLV-1 and HCV: Epidemiological and risks of exposure

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Since hepatitis C virus (HCV) and human T-cell lymphotropic virus type 1 (HTLV-1) share their transmission means, the occurrence of co-infections is expected in populations at higher risk of sexual or blood-borne viral acquisition. The aim of this study is to describe the epidemiology, and risks of exposure associated with co-infection HTLV-1 among HTLV-1-infected subjects who have been following up at the HTLV-out Clinic of Institute of Infectious "Emilio Ribas" (IIER) in Sao Paulo city. From August 2010 to March 2012, clinical charts 130 HCV-infected from Hepatology out-clinic and was noted that 2 cases (1,5%) and among 442 HTLV-1-infected subjects, of total patients, 122 (27,6%) were co-infected with HIV and 38 (8,6%) were also co-infected with HCV co- infected . We observed that the major route of transmission in patients coinfected with HTLV-1/HCV (n=40) was the use of injected drugs 37.5%. Thus, we conclude that the increased risk of coinfeccção HTLV-1 / HCV is associated with intravenous drug use.

Biography

Tatiane Assone is graduated in Biomedicine from the University Methodist of Sao Paulo, has a MBA in Health Executive from Foundation Getulio Vargas, and is Master´s Student in Tropical Medicine and Health International at the IMTSP-USP.

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Differential proteomic analysis of purified dengue virus obtained from HepG2 cells infected with varying multiplicity of infection

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Dengue is the most common mosquito-born viral disease of public health significance. Patients can develop life threatening dengue hemorrhagic fever or dengue shock syndrome (DHF/DSS) related to the dysfunction of endothelial cells and platelets. Therapy is symptomatic and is designated to control the clinical manifestation of hemorrhages and shock. Prognosis of disease severity is essential to adjust patient management. The pathogenesis of the DHF/DSS has not been fully elucidated but higher viral load has been identified as a risk factor. We made the hypothesis that the composition of host proteins associated to virions depends on the viral load and is linked to the pathogenicity. The proteomes of highly purified virions from HepG2 cells infected with dengue virus (DV) at different Multiplicity Of Infection (MOI) were compared. Five days after infection, virions were purified from the culture supernatants by ultracentrifugation and water-insoluble polyelectrolyte-based technique. The purified particles were controlled by immunoblots and quantitative RT-PCR. After in-gel hydrolysis, peptides were analysed by nano-liquid chromatography coupled to ion trap mass spectrometer and identified on data libraries. The level of viral NS1 protein excreted in the culture supernatant was shown to be proportional to the viral input. The quantity of virus released also increased. Dengue virus structural proteins and several cellular proteins were specifically identified on purified DV. Some of them were only present in samples corresponding to the infection with higher MOI. The impact of these proteins on disease outcome is discussed, as well as their potential use as biomarkers for dengue severity prognosis.

Biography

Romain Fragnoud received his M.Sc. degree in Biochemistry (2009) from the Faculty of Sciences, University Claude Bernard Lyon 1, Lyon, France He worked on the interaction between the core protein of hepatitis C virus and the lipids at the Biology and Chemistry of Proteins Institute (IBCP) Lyon, France, during his internship graduation. He is currently completing his Ph.D. degree on dengue virus in the biomarkers Department of bioMerieux SA, Marcy L'Etoile, France, in collaboration with the Emergent Pathogen Laboratory, Fondation Merieux, Lyon, France and the University Claude Bernard Lyon 1, Lyon, France. His work focuses on the identification of viral elements linked to dengue severity.

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Characteristics of viruses derived from nude mice with persistent measles infection: An animal model of subacute sclerosing panencephalitis

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Measles virus (MV) isolates from patients with subacute sclerosing panencephalitis (SSPE) differ virologically from wild-type MV.SSPE viruses lack cell-free virus formation ability and exhibit strong neurovirulence. However, there have been few reports on animal models of SSPE.

MV Edmonston strain was inoculated into the subarachnoid space of 4-week-old BALB/c nude mice. All nude mice displayed weight loss and required euthanasia, with a mean survival duration of 73.2 days. The viral load in the brain showed a 4–400-fold increase compared with the initial inoculated quantity, and infection was confirmed by immunostaining. Gene sequencing of viruses from the brain homogenate showed amino acid mutations occurred more frequently in matrix (M) proteins than other MV proteins. Start and stop codon defects were detected only in the M gene. The most common mutation was a uridine-to-cytosine transition. The virus derived from persistent infection in the brain exhibit lower cell-free virus formation ability than did the Edmonston strain. When the nude mice were challenged with 200 plaque-forming units (PFU) of MV isolates, the mean survival duration was 34.7 days, which was significantly shorter than that for mice challenged with 4×104 PFU of MV Edmonston strain ($P < 0.01$), while the mice inoculated with 200 PFU of MV Edmonston strain did not show any weight loss or death.

This study indicated that nude mice inoculated with MV exhibited typical characteristics of SSPE. This model may prove useful for the elucidation of the pathogenic mechanism of SSPE and the development of potential therapeutics.

Biography

Yusaku Abe obtained a medical license in Japan and has completed PhD course in Fukushima Medical University of Medicine. He is currently a faculty member of the Department of Pediatrics, Fukushima Medical University.

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Adapted host infectivity of bovine viral diarrhea virus quasispecies isolated from a persistently infected rabbit kidney cell line

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The rabbit kidney cell line, ATCC RK-13, has been reported to be contaminated with a noncytopathogenic (non-cp) bovine viral diarrhea virus (BVDV). BVDV`s ability to enter and replicate, is host species cell restricted, under in vitro conditions. Therefore, the purpose of this study was to investigate the adaptation of a BVDV contaminated rabbit origin cell line (RK-13). Persistent infection was confirmed by stability of virus titres (104.6±0.5 TCID50/ml) and pestivirus protein (NS3) positive cells (71.9±3.12%), over 6 subsequent passages, after detection. Based on exaltation of Newcastle disease virus (END) phenomenon, two biotypes of noncp BVDV were isolated, from contaminated virus (parent strain), as being END phenomenon positive (E+) and negative (E-), and designated RK13/E+ and RK13/E- viruses as quasispecies.

Each virus (/E+ and /E-) demonstrated 1) differing levels of reproducibility in bovine origin cells (MDBK and bovine testis cells), 2) similar antigenicity against BVDV-1 antisera, and 3) identical 5'-UTR nucleotide sequences. The inoculation of rabbit cells (RK) by RK-13 parent, RK-13/E+ and /E-, No.12/E+ and /E-, Nose/E+ and /E-, and KZ91/E+ strains, all at a MOI of 0.01, in parallel, revealed that only RK13 derived strains had growth ability in RK cells, however with different levels of reproducibility. Moreover, RK-13 viruses can grow in RK cells in either a transient or persistent infection.

Overall, these findings provide evidence that the BVDV contaminated RK-13 cell line, consists of at least two types of virus as quasispecies, which have adapted to rabbit origin cells. Further studies on these virus quasispecies may have potential benefit for the development of a live attenuated BVDV vaccine.

Biography

Mahmod Muhsen completed his Master's degree in Veterinary Science, at the age of 27 years, in Al-baath University, Homs, Syria. He had worked in the Central Laboratory for Research and Disease Diagnosis, in the faculty of Veterinary Medicine, of Al-baath University, for 6 years, and became an administrative director, within his last 2 years. He is currently pursuing doctoral studies, in virology, at Nippon Veterinary and Life Science University, in Japan, with an expected date of completion, of his PhD degree, of September 2012.

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Combinatorial antimicrobial peptides exhibit enhanced virucidal properties against vaccinia virus

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Use of antimicrobial peptides may provide an alternative to existing antibiotics and improve transfusion product safety. The present study identifies the use of four novel synthetic antimicrobial peptides named PD1-PD4 as potential anti-viral agents against a large enveloped DNA virus, the Vaccinia Virus (VV). We have previously shown PD3 and PD4 peptides to be effective anti-vaccinia virus agents. In our current work, we evaluated the combinatorial effects of PD1-PD4 on vaccinia virus infection in cell culture. The PD1-PD4 peptides were tested on plasma samples spiked with 10-fold dilutions of the wild-type laboratory strain of Vaccinia Virus (WR strain). Each spiked sample was preincubated individually with a single peptide (PD1-PD4) or in various combinations for 1 hour at 37^{0}C. Following incubation, the inoculum was added on to a monolayer of BS-C-1 cells and virus titers estimated at 24, 48 and 72 hr time points. Spiked sample without any peptide was included as control. Our analysis revealed that of the peptide combinations tested, PD2+PD3, PD3+PD4 and PD2+PD3+PD4 combination were most potent against vaccinia virus and resulted in the largest reduction of viral titers in the plasma. PD3+PD4 and PD2+PD3+PD4 peptide combinations demonstrated the highest anti-vaccinia activity by bringing about a 3-log reduction in viral titers. PD2+PD3 peptide treatment resulted in a 2-log reduction in viral titers. The present study clearly illustrates the usefulness of selective combinatorial antiviral small peptides for reducing VV burden in biological samples such as human plasma.

Biography

Dr. Shilpakala Sainath Rao is currently a Postdoctoral Fellow at the Center for Biologics Evaluation and Research, FDA. She received her Ph.D. (Microbial Genetics) in 2009 and her field of interest is antimicrobial agents. She has served as reviewer for journals like BMC Microbiology, BMC Research Notes, Foodborne Pathogens and Disease, and also served as judge for the National Institutes of Health FARE travel awards. She has received ASM Travel Award (2007) and the George McCraken Infectious Diseases Fellow Award (ICAAC, 2011). She is a member of American Society of Microbiology (ASM) and American Society of Hematology (ASH).

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Detection of dengue serotypes/genotypes by molecular methods and development of rapid diagnostics

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Dengue virus (DENV) belongs to the family Flaviviridae, genus Flavivirus, and is transmitted to humans by Aedes mosquitoes, mainly Aedes aegypti. There are 4 different serotypes of DENV, i.e; DENV1, DENV2, DENV3, DENV4 1. DENV infection is a major cause of disease in tropical and subtropical areas, with an estimated 50 million infections occurring each year and more than 2.5 billion people being at risk of infection. The research work focuses on the development of diagnostic method for detecting different dengue serotypes/genotypes and to correlate the serotype/genotype with disease severity. The standarised PCR method will be used for detection of dengue serotypes and those results will be used for the correlating will be clinical parameters. Moreover the new techniques "loop mediated amplification techniques will also be used to check the genotypes of the dengue. The new rapid diagnostic method will be developed based on the agglutination method to identify different dengue serotypes. After identifying different serotypes the , various clinical factors along with nitric oxide will be determined for the diagnosing the disease severity and for the early diagnosis of Dengue Haemorrhagic fever or dengue shock syndrome.

Dengue is diagnosed by isolation of the virus, either serologically, or by molecular diagnostic methods. Although several commercial kits for the diagnosis of dengue are available, concerns have arisen with respect to the performance characteristics of these kits. When such tests require the identification of the virus or the viral genome, they are expensive and require specialized laboratories. Affordable commercial kits of adequate sensitivity and specificity that are able to diagnose dengue infection during the acute stage have not been developed. Therefore considering the current problems in the diagnostics the main priorities are focused in the research project are early determination of acute dengue virus infection during the febrile phase , distinguishes between dengue and other flaviviruses , cost effective, easy to use at all levels of the health system, if possible, provides an early marker of severe disease, distinguishes between first and subsequent infections. Therefore, in order to develop this kind of ideal diagnostic kit we can evaluate the new technology in context of existing technology platform. This will shorten the development time for the introduction of new and improved diagnostic test of dengue.

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A new broad spectrum virus diagnostic microarray using random and taxon-specific probes

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Objectives: Necessity for rapid identification of biological agents has grown dramatically as it has become possible to engineer highly infectious pathogens in almost any well-equipped laboratory. Diagnostic microarrays have been successfully used for identification of pathogenic microorganisms. These arrays are mostly limited to a small number of organisms because a priori knowledge of genetic information is required for probe design which is not always available. To bypass this problem, we developed an alternative approach using high density chips composed of short probes with random DNA sequences. Based on a comparison of the hybridization patterns to a reference database, this chip showed reliable identification of bacteria down to the pathovar level. Preliminary assays with a small set of viruses also produced species-specific hybridization patterns. We are in the process of further developing this approach using a combination of approximately 10'000 random probes together with approximately 12'000 newly designed taxon-specific probes. The taxon-specific probes target different taxonomic levels and hence the random probe patterns may be used to differentiate within a narrow taxonomic range, enabling unprecedented differentiation levels.

Results: A script was written in order to select the optimal taxon-specific probes. Using this script on our so far elaborated sequence data, together with online available sequences, 40 probes per species have been designed. They are characterized with respect to their hybridization specificity at the family-, genus- or species-level. These probes will now be used to develop a robust and sensitive virus diagnostic tool covering a broad spectrum of over 260 virus species.

Biography

Pierre Schneeberger has completed his Master degree in June 2011 at the age of 24 years from the University of Strasbourg, France. He started his PhD at the Swiss Tropical and Public Health Institute in October 2011 with a project on viral diagnostics and viral evolution studies.

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Investigation of resistance mutations in the HBV DNA samples that isolated from patients with chronic Hepatitis to Nucleoside/Nucleotide analogues by INNO LIPA HBV DR and ultra deep pyrosequencing methods

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There are about 400 billion of people infected by Hepatitis B virus (HBV). 15-40% percent of chronic hepatitis B (CHB) patients lost their life due to mortal liver diseases such as liver deficiency, cirrhosis and hepatocellular carcinoma. Thus, HBV infections have to be treated carefully. Although nucleoside/nucleotide analogues are being used successfully, resulting antiviral resistance due to mutations is the most important reason for failing of the treatment.

INNO LIPA HBV DR test is often being used for detecting antiviral resistance mutations which are developing in CHB patients. Although well known that with this method it is possible to detect only the variants have less than 5% of viral population, the detection of known mutations is considered as the disadvantages of the method. Ultra Deep Pyro Sequencing method which is one of the next generation sequencing methods (UDPS), is a method has a high working capacity that can detect even the variants with about 1% percent in a short time.

In this study antiviral resistance mutations in CHB patients are investigated using INNO LIPA HBV DR and UDPS methods. The research related antiviral resistance mutations using INNO LIPA HBV DR method is done within routine applications in the Molecular Biology laboratory of the University of Istanbul, Medical Faculty of Istanbul, Department of Virology and Fundamental Immunology Branch. Analysis of the mutations with UDPS is done in Whole Genomic Laboratory of University of Istanbul, Medical Research Institute. 23 serum samples, which are belong to 9 naive and 14 treated CHB patients, are included in this study.

While resistance mutations could not detect in serum samples from naive patients by INNO LIPA HBV DR method, compensation mutations which caused lamuvidine resistance in 3 samples are detected using UDPS method. In treated patients (drug-experienced patients) 19 mutations in 8 samples are detected by INNO LIPA HBV DR (the frequency range; 100.0%-10%) and 29 mutations in 12 samples by UDPS (the frequency range; 100.0%-1.1%). All the mutations detected by INNO LIPA HBV DR test are also detected by UDPS. There are no mutations which are detected by INNO LIPA HBV DR but not by UDPS. The average frequency of 10 mutations which could not detected by INNO LIPA HBV DR is obtained as 14.7%.

In conclusion the common used method INNO LIPA HBV DR is identified as a sensitive and easy applicable method. However, it is considered that the detection of genotypic resistance in early stage by UDPS will provide important contribution to the direction the treatment.

This study is supported by the University of Istanbul Scientific Research Unit. Project No: 10282.

Biography

Sevim Mese has completed her Medical Faculty at the age of 23 years from Dicle University. She has worked in Health Center as a general practitioner from 1995 to 2003. Then, she has specialized on Microbiology at Medical Microbiology Department, Medical Faculty, University of Dicle from 2005 to 2008. After completing her specialising she has worked in Hospital of Batman as a Specialist of Microbiology for one year. Then, she has done a second specialisation on Virology at the University of Istanbul, Istanbul Medical Faculty, Department of Virology an Fundamental Immunology for two years. Now, she works at the same department as a virologist. She has published about 20 papers in reputed journals.

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Cellular protein HAX1 interacts with influenza a virus polymerase PA subunit and impedes its nuclear translocation

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Transcription and replication of the influenza A virus RNA genome occurs in the nucleus through the viral RNA-dependent RNA polymerase consisting of PB1, PB2, and PA. Cellular factors that associate with the viral polymerase complex play important roles in these processes. To look for cellular factors that could associate with influenza A virus PA protein, we have carried out a yeast two-hybrid screen using a HeLa cell cDNA library. We identified six cellular proteins that may interact with PA. In this report, we focused our study on one of the new PA-interacting proteins HAX1, a protein with anti-apoptotic function. By using Glutathione S-transferase pull-down and coimmunoprecipitation assays, we demonstrated that HAX1 specifically interacted with PA in vitro and in vivo, and that HAX1 interacted with the nuclear localization signal domain of PA. Nuclear transport of PA was increased in HAX1-knockdown cells and this phenotype could be reversed by reexpression of HAX1, indicating that HAX1 can impede nuclear transport of PA. As a consequence, knockdown of HAX1 resulted in a significant increase on virus yield and polymerase activity in a minigenome assay and this phenotype could be reversed by reexpression of HAX1, indicating that HAX1 can inhibit influenza A virus propagation. Together, these results not only provide an insight about the mechanism underlying nuclear transport of PA but also identify an intrinsic host factor that restricts influenza A virus infection.

Biography

Dr. Won-Bo Wang has completed his Ph.D at Purdue University and did his postdoctoral studies at Dana-Farber Cancer Institute, Harvard Medical School. He currently is a professor at College of Medicine, National Taiwan University. His studies focus on influenza A virus and DNA tumor viruses. Wei-Bin Hsu is a Ph. D student in Dr. Won-Bo Wang' s lab.

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CD44 participates in the IP-10 production in cells infected with HCV

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The mechanisms of induction of liver injury during chronic infection with hepatitis C virus (HCV) are not well understood. Interferon (IFN)-γ-inducible protein 10 (IP-10), a member of the CXC chemokine family, is expressed in the liver of chronic hepatitis C (CHC) patients and selectively recruits activated T cells to the sites of inflammation. Recently, it was shown that reduction of IP-10 expression in CHC patients was closely associated with the outcome of antiviral therapy. In this study, we examined the role of the Toll-like receptor (TLR) pathway on the IP-10 production in cells replicating HCV. Among the CXC chemokines, expression of IP-10 was specifically increased in cells replicating HCV through activation of the TLR2-dependent signaling pathway. The enhancement of IP-10 production upon stimulation with TLR2 ligands in cells replicating HCV induced CD44 expression. CD44 is a broadly distributed type I transmembrane glycoprotein and a receptor for glycosaminoglycan hyaluronan (HA). In CHC patients, expression of HA in sera has been shown to increase in accord with the progression of liver fibrosis, and HA also works as a ligand for TLR2. In the present study, IP-10 production upon HA stimulation was dependent on the expression of TLR2 and CD44, and a direct association between TLR2 and CD44 was observed. These results suggest that endogenous expression of HA in hepatocytes in CHC patients participates in IP-10 production through an engagement of TLR2 and CD44.

Reference: Takayuki Abe, and et al. 2012. CD44 participates in IP-10 induction in cells in which hepatitis C virus RNA is replicating, through an interaction with Toll-like receptor 2 and hyaluronan. J.Virol. 86. : 6159-6170.

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Human herpes virus 8 viral interferon regulatory factor 1 protein binds STING to Inhibit the host interferon response

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Kaposi's sarcoma-associated herpesvirus (KSHV) has evolved numerous mechanisms to combat the host immune response, one of which is by hijacking cellular regulatory genes for incorporation into its own genome. KSHV can also exist in the cell latently as an episome is strongly oncogenic. Viral interferon regulatory factor 1 (vIRF1), encoded by ORF K9, is the viral homolog of the cellular IRF family and has been shown to disrupt innate immune response signaling by interfering with upstream adaptor molecules essential to the initiation of the IFN response and the ability of transcription factors to bind to and induce IFN. In this study, we show for the first time the ability of any viral protein to bind to and inhibit STING (stimulator of IFN genes), the DNA sensor and innate immune response initiator, upon viral infection. STING is activated upon KSHV infection and is able to curb viral replication by inducing IFNβ. vIRF1 has the capacity to bind STING and abrogate the IFN response, likely at an upstream node of the pathway prior to IRF3 activation. Together, this study illustrates another mechanism by which a usurped KSHV gene can impede the host's ability to mount a complete innate immune response and ward off viral pathogenesis.

Biography

Alan Goodman received his B.S. in Biomedical Engineering from the Johns Hopkins University and completed his Ph.D. in Bioengineering at the University of Washington. He received a postdoctoral fellowship from the CSIC to train in Madrid, Spain for a year and a half and is currently in his second year of training under a T32 institutional grant at the University of Miami. He has published 15 papers

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Inhibition of HIV-1 in Sickle Cell Disease

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Sickle Cell Disease (SCD) is a hereditary disorder that affects about 100,000 people in USA primarily of African descent. Transfused SCD patients seem to have lower risk for HIV-1 infection and increased number of HIV-1 long-term non-progressors suggesting that SCD may prevent HIV-1 infection. We previously showed that HIV-1 transcription is inhibited by the expression of ferroportin, an iron export protein. HIV-1 infection is also blocked in macrophages and T-cells treated with hemin through the induction of heme oxygenase -1 (HO-1). In the present study we analyzed HIV-1 replication in PBMCs isolated from SCD patients, and compared it to the expression of HO-1 and ferroportin mRNAs. We also analyzed HIV-1 replication in THP-1 cells that were treated with hemin and hepcidin. HIV-1 replication was significantly reduced in SCD derived PBMCs in comparison to normal controls. Real-time PCR analysis showed induction of HO-1 and ferroportin mRNA in SCD derived PBMCs. Treatment of cultured monocytes (THP-1) with hepcidin, a ferroportin-binding peptide that leads to its degradation, increased HIV-1 replication in the cells treated with hemin. HIV-1 inhibition in the setting of SCD may include induction of HO-1 and expression of ferroportin which could be triggered by the hemolysis and the availability of heme. Expression of HO-1 and ferroportin will lead to the reduction of cellular iron and inhibition of HIV-1 transcription and replication. Low toxicity of hemin and its availability as an FDA approved drug suggests its potential usefulness as future anti-retroviral therapeutics.

Biography

Sergei Nekhai has a Ph.D. degree in biophysics from St. Petersburg Nuclear Physics Institute, Russia. He did his postdoctoral training at the George Washington University. He serves as co-Director for Howard University's Center for Sickle Cell Disease and Director of Proteomics. He studies HIV-1 transcription, pulmonary hypertension in sickle cell disease and iron metabolism with the focus on ferroportin mutations that might pose a risk for iron overload and worsen HIV-1 infection in African-Americans. He has more than 50 publications in basic virology and hematology journals and serves as a PI and co-PI on several NIH-funded grants.

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Retargeting vesicular stomatitis virus oncolytic vectors to treat Adult T-cell Leukaemia (ATL)

Dillon Betancourt, A. Morales, D. Gutman, J.C. Ramos and G. N. Barber
University of Miami Miller School of Medicine, USA

Vesicular Stomatitis Virus (VSV) is a powerful oncolytic agent that is being considered for use in clinical trials to treat malignant disease. While it's known that naturally occurring infections are typically asymptomatic in humans, VSV has demonstrated neurotoxicity in rodents if administered at high doses intravenously. To circumvent this and to target specific types of malignant disease, we've generated VSV that has the viral G glycoprotein (VSV-G) substituted for HIV gp160. We postulated that this recombinant VSV, (referred to as VSV-gp160G) should retain its potent oncolytic properties while being restricted to cells expressing CD4, the receptor for HIV gp160. For example, Adult T cell Leukemia (ATL) is a malignancy of mature CD4+ cells caused by prior infection with Human T-lymphotropic virus type-I (HTLV-I) and is susceptible to VSV infection ex vivo. However, the immunocompromised phenotype of HTLV-I carriers causes toxicity concerns when considering VSV to treat ATL. By restricting the tropism of VSV, we considered that VSV-gp160G could act as a specific oncolytic agent against ATL. VSV-gp160G was generated and evaluated for oncolytic potential and safety using non-CD4 expressing cells, as well as Hela (CD4 expressing and non expressing) cells and ATL lines. VSV-gp160G's ability to replicate and induce apoptosis against ATL cell lines was determined and also compared against wild type VSV. Additionally, both strains of VSV were used to infect CD4- and CD4+ primary cells. Our results indicated that VSV-gp160G is selectively oncolytic for transformed CD4+ cell types and that infectivity depends upon CD4 and gp120 interaction.

Biography

Dillon Betancourt is in the process of completing his PhD at the University of Miami Miller School of Medicine. He is studying Microbiology and Immunology under the advisement of Dr. Glen Barber. His research focuses on retargeting a recombinant Vesicular Stomatitis Virus to treat Adult T-cell Leukemia. Additionally, he is involved in a collaboration to incorporate Mycobacterium tuberculosis antigens into a VSV construct.

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Arbovirus co-infections in suspected febrile malaria and typhoid patients: A worrisome situation in Nigeria

Maryceln Baba[1], Christopher H. Logue[2], Bamidele Oderinde[1], Hauwa Abdulmaleek[1], Joshua Williams[1], Allessandro Marcello[3], Pierlanfranco Dagaro[4] and Roger Hewson[2]

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Clinically symptoms of malaria, typhoid and arboviruses are indistinguishable. In Nigeria, febrile patients only visit a health care facility if the fever persists after self medication with two or three anti-malaria treatments. 310 samples from suspected malaria/typhoid patients were tested initially for P. falciparum by microscopy, S.typhimurium by widal but Chikungunya , Yellow Fever, Dengue and West Nile viruses by plaque reduction neutralization test. 25 (8%) tested negative for the six pathogens, suggesting other arboviruses not tested may also be circulating. Of those that showed ≥ 90-95% virus neutralization, 196 (69%) had neutralizing antibodies against DENV, 148 (52%) against CHIKV, 72 (25%) against WNV and 25 (9%) against YFV. Within each of these groups, subsets had neutralizing antibodies to each of DENV (95/33%), CHIKV (25/9%) WNV (11/4%), and YFV (6/2%). 135 (44%) of 310 sera tested positive for > 1 virus. Of these, co-infection with DEN/CHIK viruses was most common (44%), followed by DENV/CHIKV/ WNV (23%), CHIKV/WNV and CHIK/YFV (11%). Of the 135 sera co-infected with >1 arbovirus, DENV/WNV co-infection was observed in 8% while WNV/CHIKV & YFV and WNV/YFV occurred only in 2% and 0.7%, respectively. A smaller than expected number of patients with positive diagnoses for typhoid / CHIKV (P=0.005) and DENV/YFV (P<0.001) was observed. Whereas a higher than expected number had neutralization antibodies for WNV/ CHIKV (P<0.001) and YFV/ CHIKV (P<0.001).The results suggest that misdiagnosis and under-reporting of arbovirus co-infections as malaria infections, is a serious underlying public health concern in Nigeria.

Biography

Professor Marycelin Mandu Baba is a Professor of human Virology with University of Maiduguri, Nigeria. She started the Department of Medical Laboratory Science as the head and only staff in 2003/2004 academic session. Now she has more than 30 staff and has graduated three set of graduates with each set specializing in different disciples such as in Chemical Pathology, Histopathology, Haematology and Medical Microbiology. She is also, the Director of WHO National/ ITD polio Laboratory, at Maiduguri, Borno State, Nigeria. She's actively involved in teaching and research and has published more than 40 papers in reputed local and international journals.

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The role of influenza virus polymerase stability in vaccine safety

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Influenza viruses infect millions of persons each year and are directly responsible for between 3,000 and 49,000 deaths annually in the United States. This morbidity and mortality has led to vaccination efforts against influenza A and B. Due to the low effectiveness of the triple inactivated virus (TIV), a live attenuated influenza virus (LAIV) was successfully developed to increased efficacy. The current LAIV has been increasing in popularity amongst health care providers due to both its less challenging route of administration and greater protection in persons age 1-49[1] and has recently been recommended as the primary vaccination strategy in this age group. Therefore, the mechanism underlying the stability of this attenuation is of great importance and not fully understood.

This virus has been attenuated through cold adaptation and subsequent work has determined the attenuating segments. These are contained in the polymerase (PB1, PB2, PA) and nucleoprotein segments. It has been shown through viral recombination that while the introduction of the attenuating PB2 segment into a wild type background could convey temperature sensitivity, rescue mutations in PA could restore the ability to grow at elevated temperatures[2]. These revertant viruses are of great interest as they provide an insight into the mechanism of attenuation by providing genetically similar viruses with vastly differing phenotypes and are medically significant as they provide an insight into vaccine safety.

We were gifted with these viral isolates from the lab of John Treanor and have isolated plaque purified viruses possessing impaired growth at 39°C as well as genetically similar revertant viruses who do not display reduced growth at 39°C. Further characterization of the responsible mechanism will include fully sequencing the viral genome of both the background and revertant isolates, performing a minigenome assay to determine polymerase function at various temperatures and biochemical analysis of trimeric stability in vitro.

Biography

Andrew completed his bachelors in Biology/Chemistry from Southern Nazarene University in 2009 and is currently a MD/PhD student at the University of Rochester in the lab of Dr. Baek Kim working on the mechanism of vaccine attenuation present in the live attenuated influenza vaccine.

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Alternative prevention of infectious diseases

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Infectious microorganisms are traditionally subdivided into normal, pathogenic, and opportunistic. This classification disregards that sometimes normal microorganisms cause diseases, or pathogenic microorganisms coexist with a healthy carrier. In other words, all microorganisms in a host body are opportunistic pathogens. This is not the only similarity between "normal" and "pathogenic" microfloras. In essence, there is no fundamental difference between them: both are infectious, and both may either cause diseases or persist in healthy carriers. Normal microflora is the most infectious: it infects all representatives of the host species soon after birth. This is literally an endless epidemic. In historical perspective, cholera and plague epidemics are no rivals to normal microflora as regards the number of victims. Normal microflora is infectious because the host body needs it; i.e., its infectiousness is an active property of the host: we infect ourselves with what we need. Now since "normal" and "pathogenic" microfloras are essentially similar in pathogenicity, infectiousness, and the healthy carrier state, it is conceivable that the susceptibility to "pathogenic" infectious microflora is also active. The infectiousness of pathological prions is strong evidence for active susceptibility [1]. Passive susceptibility, however, does exist; examples are the susceptibility to tetanus, botulism, and gas gangrene. The host body does not actively "attract" their pathogens; it is merely an accidental nutrient medium for them; hence, these infectious are not contagious. It should be emphasized that active susceptibility is due to the host's demand for the products of certain microorganisms' genes rather than the needs of the microorganisms themselves. This clearly shows the prospects for alternative prevention of infectious diseases: transplantation of the required microbial genes into the genome of the susceptible species should result in natural insusceptibility to the microorganism that normally carries them. An application for the patent on this approach [2] has been published by WIPO.

1. A.P.Malyshkin. Infection: A Hypothesis on Active Susceptibility and Species Immunity with Implications for AIDS Prevention. J. Immunobiology, 215 (2010), 894–897

2. A.P.Malyshkin. Method for prevention of infectious diseases of plants, animals and humans. WO2011084090

Biography

Alexander P. Malyshkin, male, microbiologist, graduated from Orenburg State Medical Academy in 1979 and worked for this academy as a researcher. After defending his Candidate of Science (Med.) dissertation (PhD thesis), he headed the Division of Laboratory Diagnosis of Orenburg Regional Tuberculosis Dispensary for some time. Dr. Malyshkin's field of research includes microbiology, immunology, and issues of infectious diseases and their prevention. He is the author of the active susceptibility hypothesis and a fundamentally new approach to the prevention of infectious diseases of plants, animals, and humans (including the HIV infection) based on it. The main recent work (now in press) is the chapter on the prevention of infectious diseases in the book Aquatic Plants and Plant Diseases (to be published by Nova Science). Dr. Malyshkin is exploring the possibility of collaboration in further developing and implementing his novel approach to disease prevention, which could be used, in particular, for breeding infection-resistant animal and plant varieties.

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The profile humoral immunoreactivity in survivors of ebolavirus Sudan

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Ebolavirus is a member of the family Filoviridae and the cause of Ebola hemorrhagic fever (EHF). Since it was first recognized in 1976, more than 18 outbreaks have occurred involving more than 2000 human cases, 1400 deaths, and with a case fatality rate that ranged from 30-90%. During the last decade, much research focused on the human immune response to ebolavirus to investigate the role of humoral immunity to different viral epitopes. Although there have been great efforts in this regard, the profile of specific humoral immunoreactivity to viral proteins during viral infection and their role in immunity and/or recovery from disease is still poorly understood. As such, we performed a study to characterize the human humoral immune response to the individual viral proteins of ebolavirus Sudan (strain Gulu). Our studies compared the profile of humoral immunoreactivity in serum of survivors versus non survivors of ebolavirus infection, to identify patterns of specific viral protein and epitope recognition that might correlate with a positive disease outcome. The results of our work thus far have supported previous published data and contributed to further understanding the humoral immune response, and its recognition profile to specific viral targets, following ebolavirus infection in human survivors.

Biography

Ariel Sobarzo received his B.Med.L.Sc. degree in 2005 and M.Sc. degree in 2007 with excellence from the Department of Virology, Faculty of Health Science, Ben-Gurion University of the Negev, Beer-Sheva, Israel. He is currently completing his Ph.D. degree in the Department of Virology, Faculty of Health Science, and in the Department of Biotechnology Engineering, Ben-Gurion University of the Negev. His research work involves an international collaboration for a sera-screening study to identify epitopes of the Ebola Sudan virus that are recognized by the immune response and correlate with disease outcome. These epitopes will hopefully be used for future development of diagnostics and therapeutics.

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The hydroelectrics on the Madeira River and the incidence of dengue hemorrhagic fever in Porto Velho

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With the current level of the humanity scientific knowledge, it is indisputable the relationship between the environment, space, and health. The various problems related to health and the environment in the State of Rondônia, in particular in the city of Porto Velho, are not new and it seems there is no provision to the end. The construction of large hydroelectric projects developed in the region, again, contributes to this statistic leaving the population vulnerable to the emergence of epidemics and endeminas. Thus, this study aims to analyze the pattern of geographical distribution of epidemiological manifestations of dengue hemorrhagic fever in the city of Porto Velho in the period preceding the construction of hydroelectric plants by the year 2009, and the relationship with the appearance of old and new manifestations of the disease. Data collection was conducted through the information available in the program DATASUS and the results indicate a significant increase in reported cases of dengue hemorrhagic fever between the years 2007 (0 registered cases), 2008 (6 cases reported) and year 2009 (42 reported cases) in health care centers in the city. Thus it is evident that the environmental changes influence the epidemiology of this disease manifestation. Moreover, it is noteworthy that the intervention on the incidence of this disease will only be achieved by controlling the mosquito vector population through targeted measures in the domestic sanitation and education of the human population in order to eliminate or prevent the formation of mosquito breeding sites.

Biography

Daniel Delani is graduated in Biological Sciences from the Faculty of Education of Porto Velho and has postgraduate in Methodology for Higher Education and Curricular Innovations and in Environmental Management. He is currently Assistant Professor I of the Federal University of Rondonia.

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Molecular detection and epidemiology of enteroviruses in Korea, 2001-2010

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Enteroviruses (EVs) are among the most common viruses infecting humans. The genus Enterovirus (family Picornaviridae) includes 4 species of human EV (A, B, C, and D). Most clinical manifestations are asymptomatic, but these viruses are implicated in a wide variety of clinical syndromes, ranging from minor febrile illness to severe, potentially fatal conditions such as neuromuscular complaints.

In this study, we have analyzed the epidemic patterns of enteroviruses in Korea during recent decade, 2001-2010 and we have detected 4,384 EVs from 14,483 patients during 2001-2010 in Korea and most common clinical manifestation is aseptic meningitis (n=4,380), and followed by HFMD/Herpangina (n=1,616). Among enterovirus-positive samples, 2,875 EV serotypes were determined by viral VP1 semi-nested RT-PCR. Most strains belonged to HEV-B (n=1,935, 70.0%) followed by HEV-A (n=816, 28.7%) and HEV-C (n=124, 4.3%). Predominant enterovirus type was EV71 (n=414, 14.4%) followed by E30 (n=379, 13.2%) and E6 (n=245, 8.5%).

EVs are an important cause of aseptic meningitis and encephalitis, and HFMD. The understanding of the recent 10 year epidemic pattern of circulating enteroviruses in Korea may be helpful in predicting the outcome of aseptic meningitis, encephalitis, and HFMD and development of antiviral treatments and vaccine.

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Dengue in Brazil: Epidemiology and diagnosis over 25 years by a regional reference laboratory

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Dengue is considered the most important mosquito-borne viral disease in humans who live in the tropical and subtropical areas of the world. High dengue activity in Brazil during the past 26 years is evidenced by the large number of cases. DENV-1, DENV-2 and DENV-3 were introduced in Rio de Janeiro, in 1986, 1990 and 2000, respectively. DENV-4 has recently been isolated after 28 years. The introduction of DENV-2 in 1990 caused the first cases of DHF and DSS. DENV-3 introduction led to severe epidemics in 2002 with DHF cases and deaths. In 2007–2008, the country experienced the most severe dengue epidemic in terms of morbidity and mortality and severe cases in children. Genomic analysis on DENV-2 identified the introduction of a distinct lineage of the Asian/American genotype. In 2009 and 2010, DENV-1 reemerged and the phylogeny also demonstrated the circulation of distinct lineages. Since 1986, laboratorial diagnosis has played an important role in the disease surveillance. Virus isolation and IgM ELISA were first used. With the introduction with DENV-2 in 1990, immune response characterization was performed by an IgG-ELISA. In the 90´s, molecular techniques such as RT-PCR and sequencing were used for nucleic acid detection and characterization. Real –time PCR showed to be essential for dengue fatal cases confirmation and NS1 tests have been used for the early diagnosis of dengue infections. Our experience has shown that the implementation of new diagnosis techniques over the years constituted an important and reliable tool for the disease surveillance in Brazil.

Biography

Flavia dos Santos has completed her PhD at the age of 29 from the Oswaldo Cruz Institute, Brazil and her postdoctoral fellowship in 2007 from the University of California, Berkeley, USA. She is currently a Brazilian Ministry of Health employee. For the past 17 years she has been working on the Regional Reference Laboratory for Dengue and Yellow Fever Diagnosis and has published about 30 papers.

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Torque Teno Virus (TTV) infection in hemodialysis patients

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Present study describes the prevalence and association of torque teno virus (TTV) infection with blood transmitted viral hepatitis including hepatitis B virus (HBV) and hepatitis C virus (HCV) infections in patients with chronic renal failure (CRF) on maintenance hemodialysis. TTV infection was diagnosed with detection of TTV-DNA in serum using polymerase chain reaction (PCR) technique. TTV-DNA was estimated in a total number of hundred patients with CRF simultaneously with 100 voluntary blood donors as controls. The markers of HBV and HCV were also tested in sera samples of these patients. TTV-DNA was detected in 39 of 100 patients (39%) with CRF and 27 of 100 (27%) healthy control. Analysis of results demonstrated HBsAg, IgM anti-HBc, anti-HCV and HCV core antigen in 5.0%, 3.0%, 6.0% and 4.0% patients, respectively. This study could not show any association of TTV with HBV and HCV infections for the transmission pattern or any impact on severity of diseases caused by these viruses in CRF patients. TTV also could not show any association with demographic characteristics of patients, duration of dialysis, number of blood transfusions and renal / liver function of the patients. As such, this study concludes that TTV appears a benign pathogen, showing no sign of renal / liver damage or any change in severity of diseases caused by blood borne hepatitis viruses.

Biography

Kishore mandal has completed his MD in Laboratory Medicine from All India Institute of Medical Sciences, New Delhi, India.

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Molecular characterization and phylogenetic analysis of hemagglutinin and neuraminidase of human H3N2 influenza A viruses in China from 2007 to 2011

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Annual H3N2 subtype influenza outbreaks in Guangdong, China are a severe public health issue and require ongoing monitoring of emerging viral variants. The variation and evolution of hemagglutinin (HA) and neuraminidase (NA) genes of influenza isolates from Guangdong and others from GenBank were analyzed using Lasergene 7.1 and Mega 5.03, and serologic analysis of antigens was determined by hemagglutination inhibition (HI). Susceptibility to drugs were correlated with genetic mutations. Phylogenetic analysis and alignments of HA and NA genes were performed on 18 Guangdong isolates and 26 global reference strains. The nonsynonymous (dN) evolutionary rate of the HA1 was 3.13 times that of HA2. Compared with the A/Perth/10/2009 vaccine HA gene, homologies with Guangdong isolates in 2009 reached 98.8–99.7% and in 2010 reached 98.0–98.4%. Amino acid substitutions were found in five epitopes of HA1 from Guangdong isolates between 2007–2011, especially in epitopes B (N160K) and D (K174R/N). The K189E/N/Q and T228A mutations in the receptor-binding site (RBS) occurred in the 2010 strains, which impacted the antigenicity of HA1. The antigenicity of the epidemic H3N2 isolates in 2010 was somewhat different from A/Perth/10/2009. The Guangdong H3N2 isolates were determined to be oseltamivir-resistant with IC50 of 0.396 ± 0.085 nmol/L (n = 17) and zanamivir-resistant with IC_{50} of 0.477 ± 0.149 nmol/L (n = 18). Variations were present in epitopes B and D, two sites in the RBS and two glycosylation sites in the Guangdong H3N2 HA1 gene. The majority of the Guangdong H3N2 isolates were sensitive to oseltamivir and zanamivir. Compared to the World Health Organization (WHO) 2011 vaccine strains, Guangdong H3N2 strains were genetically and antigenically varied slightly.

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Comparative evaluation of three regimens for the treatment of chronic hepatitis B: Tenofovir, Entecavir and combination of Lamivudine and Adefovir

Jayakumar R and Singh S
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Chronic hepatitis B is a disease of concern due to its life threatening complications like cirrhosis, and hepatocellular carcinoma (HCC) in 20-40% of patients. There are about 400 million people affected worldwide with HBV and over 300,000 die every year from HBV related diseases. Oral antivirals like lamivudine, adefovir, entecavir and tenofovir are commonly used to treat chronic hepatitis B. In this study, we tried to evaluate the comparative efficacy of these drugs alone and in combination. Chronic hepatitis B patients with HBV DNA more than 104Copies/mL irrespective of their HBeAg status (n=60) were enrolled in a prospective study. 21, 20 and 19 patients were treated with lamivudine (100mg/day) plus adefovir (10mg/day) combination entecavir monotherapy (0.5mg/day) and tenofovir monotherapy(300mg/day) respectively and followed up for 24 weeks with their virological, serological and biochemical markers measured at 12 and 24 weeks.After 24 weeks of treatment, there was no significant difference between the three groups in suppressing HBV DNA to undetectable levels. The median decrease in HBV DNA levels from baseline was better with tenofovir and entecavir monotherapies than lamivudine and adefovir combination which was statistically significant. There was no significant difference between the three groups in HBsAg and HBeAg seroconversion and normalization of biochemical parameters. Entecavir and tenofovir monotherapy were found to be more effective than lamivudine plus adefovir combination in reducing the HBV DNA levels. However, lamivudine plus adefovir combination was not too inferior especially when cost of treatment was taken into consideration.

Biography

Dr. Rajeswari Jayakumar is pursuing her MD in Laboratory medicine from All India Institute of Medical Sciences and she is working under the guidance of Dr. Sarman Singh on Hepatitis B.

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First report of a begomovirus infecting *Crassocephalum crepidioides* (Benth.) S Moore in India

Renuka Sharma and Yogesh Kumar
Amity University, India

Vein yellowing disease was observed on weed Crassocephalum crepidioides in northern India. Ageratum enation virus (AEV) along with an alphasatellite was found to be associated with the weed. Leaf samples of the symptomatic plants were collected and total DNA was extracted. Samples were subjected to rolling circle amplification followed by restriction digestion with a number of restriction enzymes to identify an enzyme having a single site in the genomic components. Complete genome of the begomovirus and alphasatellite were cloned and sequenced using primer walking. The isolate had 99% identity with an isolate of AEV reported from Zinnia elegans from this region. AEV and alphasatellite sequences from C. crepidioides were submitted to Genbank database under accession numbers FN794201 and FN794202 respectively. To the best of our knowledge, this report is the first of any begomovirus infection in C. crepidioides in India and the first on AEV infecting C. crepidioides worldwide.

Biography

Renuka Sharma is pursuing Ph.D from Amity university. She did her graduation in Biotechnology from Guru Nanak Dev University, Amritsar. She did her masters in biotechnology from LPU,Jalandhar. She did her thesis work at Directorate of Wheat Research, Karnal with Dr. Pradeep Sharma.

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The E627K mutation in the PB2 subunit of influenza A polymerase affects promoter binding at higher temperatures

Shilpa Aggarwal and Baek Kim
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Most avian influenza A viruses, which preferentially replicate at the high temperatures found in the digestive tract of birds, have a glutamic acid at residue 627 of the viral RNA polymerase PB2 subunit (Glu-627). In contrast, human viruses optimally replicate at the low temperatures observed in the human respiratory tract and have a lysine (Lys-627) at this position. The mechanism of action for this mutation is still not understood, although interaction with host factors has been proposed to play a major role. Previously, we have shown that this PB2 mutation may alter the temperature-dependent enzymatic activity of the viral polymerase. Moreover, our steady-state kinetics data revealed that the E627K mutation elevates apparent K(cat) at low temperatures with little effect on K(m), suggesting that the E627K mutation alters the biochemical steps involved in enzyme catalysis rather than interaction with the incoming NTP. To further explore this, we tested the structural integrity of the heterotrimeric polymerase complex at higher temperatures. We also looked at binding of the polymerase with 5' and 3' vRNA conserved promoter sequences at different temperatures. Our data suggests that the polymerase with Lys-PB2 shows increased binding to the 3' vRNA at lower temperatures with no difference in binding to the 5' vRNA. Also, we did not see a difference in the structural stability of the polymerase at the different temperatures tested.

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The effects of the deletion of VHS and EP0 gene on global gene expression of pseudorabies virus

Dora Tombacz and Zsolt Boldogkoi
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Pseudorabies virus (PRV) is a useful model organism for the study of molecular pathogenesis of herpesviruses. This virus has 70 protein coding genes, which belong in three major temporal classes: the immediate-early (IE), early (E) and late (L) classes (the fourth class is the early/late (E/L)). We analyzed the effects of the deletions of virion host shut-off (VHS) and early protein 0 (EP0) genes on the expression kinetics of PRV genes by real-time reverse transcription-PCR. Our data demonstrate that both genes selectively affect the E gene expression of the virus. Our results show that the VHS protein is a coordinator of global gene expression in PRV. This study revealed that in the early stage of viral infection tegument VHS proteins affect the amount of viral transcripts without bias toward any kinetic class of viral transcripts. Later, de novo VHS exerts a differential negative effect on the level of E transcripts. We also found that the effect on the level of L transcripts is slight, and there moderate lowering effect on the level of E/L transcripts. Our data suggest that a major function of the VHS protein is to assist in the switch from the early to late period of infection by selective inhibition of the E transcripts. EP0 exert a selective negative effect on the E transcripts in the early infection period and a general, alternating effect on the amounts of transcripts of each kinetic class of PRV genes in the late phase of infection.

Biography

Dóra Tombácz is MSc in Biology (Faculty of Sciences, University of Szeged - 2006) and PhD in Medical Sciences (Faculty of Medicine, University of Szeged - 2010). She works in the Department of Medical Biology at the Faculty of Medicine at University of Szeged in the Boldogkői's group. Their primary field of interest is analysis of herpesvirus gene expression and utilization of herpesviruses as tools in various fields of biology including neurobiology and cardiology.

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Anti-influenza virus activity of short synthetic Antimicrobial Peptides (AMPs)

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We have recently demonstrated antimicrobial activity for nine selected peptides against bacteria relevant to transfusion medicine and the WR strain of vaccinia virus. These AMPs include thrombin-induced human platelet-derived AMPs (named PD1-4) and Arginine-Tryptophan (RW) repeat AMPs (RW1-5). Based on our research, we theorized that these AMPs could also exert their antimicrobial activity on a number of medically important enveloped viruses such as Influenza A Virus. The nine peptides (PD1-PD4 and RW1-RW5) were tested on influenza A virus, H1N1 (2009-pandemic vaccine strain) grown in Madin-Derby Canine Kidney (MDCK) cells. These peptides were evaluated for antiviral activity by pre-treating the virus with peptides before subjecting the cells to viral infection. Inhibition of virus replication was measured by reduction of virus titer in the presence or absence of the peptide by 50% Tissue Culture Infectivity Dose (TCID50) assay in a 96-well format. RW4 and RW5 led to 10-100 fold and 100-1000 fold reduction of viral titers respectively, when compared to the untreated control virus titers. RW4 and RW5 were further evaluated for their minimal inhibitory concentration (MIC) by serial dilutions (10μM, 5μM, 2.5μM, and 1.25μM) and incubation with a constant concentration of the virus (4.0 Log10 TCID50/0.1 ml). The RW5 peptide exhibited significant antiviral activity at the lowest concentration analyzed (1.25μM) resulting in approximately 50-fold reduction of H1N1 viral titers. Based on these results, the current study clearly demonstrates the antiviral potential of small synthetic AMPs against Influenza A Virus.

Biography

Dr. Krishna Mohan V. Ketha completed his Ph.D. from University of Madras, India and Postdoctoral studies at the Center for Biologics Research & Evaluation (CBER), FDA, USA. Currently he is a Staff Scientist in CBER, FDA performing both Research and Regulatory Review with more than 10 years of Regulatory experience. He has published more than 25 papers in reputed journals and book chapters. He serves as an Editorial Board Member and Advisory Board Member of peer-reviewed journals.

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Changes in lipid profiles and BMI in HIV/AIDS patients on antiretroviral and anti-Tuberculosis therapies in FAKO division

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Introduction: HIV/TB co-infection remains a major problem in Cameroon Today. This study was aimed at evaluating the changes in lipid profile and BMI in HIV/AIDS patients on antiretroviral (ARV) and anti-tuberculosis (ATT) therapies.

Methods: Participants were recruited from the HIV and TB treatment centres in Limbe and Buea regional hospitals. Blood samples were collected and questionnaires administered to get the necessary study parameters. Participants were assembled into six groups: normal controls, HIV positive not on ARV (naïve), HIV positive on ARV, tuberculosis only on ATT, co-infected on ATT only and co-infected on ATT and ARV. Lipid profiles and BMI of each group were compared to those of the normal controls as well as comparison between groups. CD4+ counts were also compared between the HIV positive groups and a correlation analyses performed between CD4+ and the test parameters in all HIV positives on treatment.

Results: Four hundred and eighty adults (15-77 years) were recruited into the study. Comparative analyses revealed significantly low HDLc levels below normal in each of the groups compared to the normal controls ($P<0.05$) with varied changes in the TRIG, TC, LDL and BMI within the normal range. Comparison if the HIV positives to naïve revealed significantly higher HDLc and TC and a comparison of co-infected groups to TB only revealed significantly lower TC and HDLc in the co-infected on ATT only. The CD4+ counts of the two co-infected groups were significantly lower compared to naïve. Correlation analyses revealed a significant positive correlation between CD4+ counts and BMI in HIV positives on treatment.

Conclusion: From the analyses, Tuberculosis was observed to worsen the immune suppression caused by HIV and enhances HIV-induced lipid profile changes. Also, ARV alone and coupled to ATT induces complex lipid profile and BMI alterations suggesting the need for routine laboratory and dietary monitoring.

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Isolation of Candida species in oral cavity of HIV/AIDS pediatric patients from the north border of Mexico and their possible association to immune failure

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The aim of the study was to determine the presence of Candida species in regard to immune status of thirty HIV+/AIDS pediatric patients undergoing Highly Active Antiretroviral Therapy (HAART). The CD4 lymphocyte cell count/mL, viral load and the immune and virological statuses were obtained from medical records. Thirty HIV+/AIDS children from the north border of Mexico 3–13 years-old (7.7 ± 3.6), perinatally infected under antiretroviral treatment were included. Samples from total oral mucosa were taken and inoculated in HardyChrom culture medium for the isolation of C. tropicalis, krusei and albicans. Candida albicans isolates were present in 17 of the 30 (56.66%) HIV+/AIDS samples. Candida tropicalis isolates were present in 8 samples (26.66) and two different species of Candida were isolated in 9 samples. Both species of Candida (C. albicans and C. tropicalis) were detected in 7 patients (23.33%) with undetectable viral load, 2 patients (6.66%) with viral load of 50-10,000 copies and none with viral load of 10,000-100,000. In regard to immune status, Candida isolates were present in 13 patients (43.33%) without immunosuppression, 3 patients (10%) with moderate immunosuppression and only one patient (3.33%) with severe immunosuppression. These findings indicate that HIV+/AIDS pediatric patients show high frequency of oral Candida in spite of the adherence to antiretroviral therapy. The oral presence of Candida spp was not associated with CD4 cell count or viral load in this study and was not useful as a marker of the immune status in HIV+/AIDS children.

Biography

Dr. Jorge Arturo Alvelais Palacios is Professor of Immunology at the Center for Health Sciences of the Autonomous University of Baja California. He got his degree of Physician at the Autonomous University of Puebla and has a degree as a Specialist Physician in Clinical Microbiology from the Catholic University of Argentina.

Dr. Alvelais has been director of CAPASITS (Outpatient Center for Prevention and Treatment of AIDS and STDs) and is currently director of the emergency services at the General Hospital in Tecate, BC, Mexico.

Dr. Alvelais has several recent publications about HIV-infections in drug injection users at the US-Mexico border.

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Three-dimensional modeling of DCIR and identification of new drugs blocking HIV-1 attachment and propagation

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Introduction: The HIV-1 pandemic continues to expand while no effective vaccine is yet available. Finding new therapeutic targets and drugs is therefore crucial. We have previously shown that the dendritic cell immunoreceptor (DCIR), a C-type lectin receptor expressed in dendritic cells (DCs), acts as an attachment factor for HIV-1 to DCs and contributes to HIV-1 transmission to CD4+ T lymphocytes (CD4TL). Directly involved in HIV-1 infection, DCIR is expressed in apoptotic or infected CD4TL and promotes trans-infection to bystander cells. The aim of the present study is to characterize the extracellular domain of DCIR and to test chemical inhibitors of HIV-1 attachment thereto.

Results: We present the first three-dimensional model of DCIR structure. Based on this structure, several inhibitors were selected to target viral interaction with the carbohydrate recognition domain and the EPS motif. Preliminary screening using Raji-CD4-DCIR cells identified two inhibitors that decreased HIV-1 attachment and propagation. These inhibitors did not affect the proliferation of peripheral blood mononuclear cells.

Conclusions: The results of this study thus suggest structures for novel molecules capable of blocking HIV-1 transmission by DCs and CD4TL.

Biography

Dr. Arezki Azzi has completed his Ph.D in 1988 from Laval University, Canada and postdoctoral studies from Florida State University in Molecular Biophysics. He is an associate professor at the Al Imam Ibn Saud Islamic University department of Phamacology. His research interests are in antivirals design.

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Crystallization and X-ray diffraction of virus-like particles of a grouper betanodavirus

Yu-Chun Luo and Chan-Shing Lin
National Sun Yat-sen University, Taiwan

The grouper is a high-value fish in seafood market, whereas grouper nervous necrosis virus (GNNV) causes near 100% mortality in larvae and juveniles. We expressed GNNV virus-like particles in Escherichia coli and crystallized it by sitting-drop vapor diffusion method. The crystals grew to the size of 0.22-0.27 mm in one week and diffracted by X-rays to 7.5 Å resolution. The data were indexed in a primitive rhombohedral crystal system. Preliminary processing of the DGNNV VLPs diffracting data suggests that the crystal belong to space group R32 with a = b = 353.00 Å, c = 800.40 Å, α = β = 90°,γ = 120°. A total of 299 images were collected from a single VLP crystal by 60° diffracting range, 0.2 oscillation angle, and 10s exposure. 60,035 observed reflections were reduced to 23,268 unique reflections with an overall Rmerge of 18.2% and a completeness of 93.2%. Self-rotation function maps were calculated using the program POLARRFN from the CCP4 suite; spherical angles of κ = 72°, 120°, and 180° were for fivefold, threefold and twofold symmetry axes. The 3D atomic model of the asymmetric subunit were predicted. This is the first attempt to solve betanodavirus structure in addition to electron cryomicroscopy.

Biography

Yu-Chun Luo is Ph.D student and Dr. Chan-Shing Lin is the Professor from Department of Marine Biotechnology and Resources, National Sun Yat-sen University, Kaohsiung 80424, Taiwan. Their primary field of interest is analysis of DGNNV VLPs protein expression and utilization of DGNNV VLPs as tools in various fields of biology including Virology, Biochemistry, Pharmacology and Crystallography.

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GenOMICS of a Zoonosis: Full adaptation of Hepatitis E virus quasispecies during interspecies transmission

Jerome Bouquet
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Hepatitis E virus (HEV) is responsible for epidemics of entero-transmissible hepatitis in tropical and subtropical countries, but also of sporadic infections of uncertain origin in industrialized countries. However, HEV has been found to infect animals other than primates. Particularly, strong identities between partial HEV sequences isolated from human and swine were found. Evaluation of the genomic modifications of HEV – at the level of its full-length consensus sequence and its quasispecies – was conducted during an interspecies transmission in order to assess the extent of the species barrier between human and swine.

High-throughput sequencing of bile and feces from two pigs experimentally infected with human HEV of genotype 3 revealed the same full-length consensus sequence as in the human sample. In addition, 29% of polymorphic sites found in HEV from the human sample were conserved throughout the infection of the heterologous host. The inter-species transmission of HEV quasispecies is the result of a genomic negative selection pressure on random mutations which are mostly deleterious to the viral population. HEV intrahost nucleotide diversity was found to be in the lower range of other human RNA viruses but correlated with values found for zoonotic viruses, suggesting.

In conclusion, these results suggest that HEV transmission is modulated by ecological drivers rather than host-specific mutations and that its full genomic adaptation to both human and swine represents a zoonotic risk.

Biography

Jerome Bouquet is a PhD student working at the French Agency for Food, Environmental and Occupational Health and Safety. His research focuses on the genetics, evolution and epidemiology of hepatitis E virus in human and animals and the risk for zoonotic transmission.

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Transcription level of ifnar1 and socs1 in patients infected with Hepatitis C Virus responders and non-responders to the IFN-alpha treatment

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A great ratio of patients infected with hepatitis C viruses (HCV) do not respond to antiviral therapy of pegilated interferon-alpha plus ribavirin. Antiviral response is established by the equilibrium between expression of interferon stimulated genes (ISG) and their negative regulators (NR), as well as the efficient interaction of IFN with the functional subunits 1 and 2 from receptor (IFNAR). ISG, NR and IFNAR1 isoform transcription level was determined and analyzed their association with capacity of antiviral response.

Methods: Patients were grouped as responders to treatment (R), no-responders (NR) untreated (NT) and individuals not infected (NI). Ifnar1 and socs1 transcription levels were analyze by real time RT-PCR in peripheral blood mononuclear cells (PBMC) of 59 patients infected with hepatitis C virus (HCV) and 17 non-infected individuals.

Results: Ifnar1 transcription was increased significantly in HCV-infected patients respect non-infected individuals (1 ± 0.34; P =0.005), while socs1 transcription level was greater in responder than others groups. Ifnar1 and socs1 transcription was greater in patients infected with genotype 1a than those infected with genotypes 1a and 1a1b. Furthermore socs1 transcript was absent in three patients infected with HCV genotype 1b. A negative and weak correlation between ifnar1 and socs1 transcription was found, associated with viral genotype. Conclusion: Our results suggest that HCV infection may up-regulate ifnar1 transcription. HCV genotype viral seems to be an important factor in the ifnar1 and socs1 transcription as well as in the ability to evade the antiviral response

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Effect of site-directed mutagenesis within the 5'UTR and VP1 regions on temperature sensitivity of Enterovirus 71 in vitro

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Objective: In the poliomyelitis eradication era Enterovirus 71 (EV71) is a newly emerged human pathogen which has been associated with numerous cases of neurological complications observed mainly in young children. Understanding the pathogen biology is a keystone in the development of both an effective vaccine and specific antiviral treatment. The aim of this research was to identify molecular determinants of temperature sensitivity within the VP1 protein of EV71 and study their effect on viral growth, when combined with a Sabin 3 attenuating determinant within the 5'UTR.

Methods: Site directed mutagenesis was employed to introduce mutations within the 5'UTR and VP1 regions of an infectious cDNA clone of EV71. Mutant viruses were rescued in cell culture from the in vitro RNA transcripts. Growth phenotype of mutant viruses was studied in cell culture.

Results: Twenty nine EV71 mutants within the VP1 protein were constructed. Fourteen of them were infectious upon transfection of Vero cells with in vitro transcribed RNA. Substitutions with alanine at amino acid positions 162, 164 and 213 resulted in restricted viral replication in cell culture at an elevated temperature. Growth characteristics of the mutant viruses were similar to those of the mutant EV71 carrying the main attenuating determinant of Sabin 3 within the 5'UTR. The temperature sensitivity of EV71 was further enhanced when the 5'UTR and VP1 mutations were combined.

Conclusions: Mutations within the VP1 protein of EV71 can lead to temperature sensitivity of the virus. Combination of VP1 and 5'UTR mutations has a cooperative effect on viral phenotype.

Biography

Natallia Lazouskaya completed her MSc from the Belarussian State University in 1995. She was a junior scientist at the Research Institute for Epidemiology and Microbiology, Clinical Virology Department (Minsk, Belarus) for ten years. During that time she collaborated in scientific projects on HIV and Hepatitis B and C with the Academic Medical Center, University of Amsterdam (Netherlands) and the Laboratorie National de Sante, Department of Immunology (Luxembourg). Currently, she is a PhD student at Swinburne University (Melbourne, Australia) and undertaking a research project on Enterovirus 71. She is a recipient of a Swinburne University Centenary Postgraduate Research Award (SUCPRA) and an Endeavour International Postgraduate Research Scholarship (EIPRS). Her research interests are human viruses, the evolution of viruses, and vaccine development.

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Expression of Hepatitis B virus "E" antigen gene region in yeast

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The "e" antigen (HBeAg) gene region of hepatitis B virus (HBV) was transformed and expressed into an eukaryotic expression vector by recombinant DNA technology in order to obtain protein used in anti-HBe tests which is being one of the most important marker for serodiagnosis of HBV infections.

For this purpose, HBV-DNA positive patient sera were used as the source of viral nucleic acids, and primers coding HBeAg gene region have been designed. After the amplification of HBeAg gene region by polymerase chain reaction (PCR), the amplicons were purified and cloned to expression vector (pYES2.1 plasmid) and this vector was transformed to competent bacteria (TOPO 10F' Escherichia coli) by CaCl2 method. After competent bacteria were grown on selective media, the plasmid "pYES2.1 + HBeAg" were isolated and transformed to Saccharomyces cerevisiae.

Finally, whether HBeAg was expressed by the yeast was checked through an automatized microparticule chemiluminescence system.

HBeAg producing yeast cells obtained may render it possible to purely isolate proteins in future studies. Since the diagnostic kits used in our country for hepatitis B serology are usually imported products, this creates a great economical burden. Thus, the experience and knowledge that builds up following such studies will help to produce our own diagnostic products using our equity.

Biography

Dr. Berksan SIMSEK has graduated from faculty of medicine in 2005 and still a senior resident in medical microbiology department in Gulhane Military Medical Academy since 2008.

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Hepatitis B virus in HIV infected patients in northern South Africa: Prevalence, exposure, protection, response to HAART and genetic characterization

Pascal Bessong, Julius Ayuk and Jeffrey Mphahlele
University of Venda, South Africa

Hepatitis B virus (HBV) and HIV are endemic infections in many African countries. The objectives of this study was to determine the levels of exposure and protection to HBV, prevalence of HIV/HBV co-infection, the response of HBV to HAART, and HBV genotypes in a cross-section of HIV infected patients in northeastern South Africa where data is very limited. Three hundred and eighty patients were screened by ELISA for HBsAg, anti-HBc and anti-HBs. Samples non-reactive for HBsAg but reactive for anti-HBc were examined for occult infection. Response to HAART was assessed by measuring HBV viral loads, seroconversion from HBeAg to anti-HBe, and levels of alanine transaminoferase before and after 12 months of therapy. HBV genotypes were determined phylogenetically. Sixty percent (95% CI, 54.8-64.9) of the patients were exposed to HBV based on HBsAg, anti-HBs or anti-HBc; 20% (95% CI, 16.1-24.4) had active HBV infection based on HBsAg serology, and 30% (95% CI, 25.2-35.2) were protected. Sixteen percent (95% CI, 12.5-20.1) had occult HBV infection. The differences in prevalence were not statistically significant when gender, marital status, and CD4+ cell counts were considered. At least 80% of the patients showed adequate response to the first line HAART regimen (stavudine/lamivudine/efavirenz or nevirapine) after 12 months of use. Ninety three percent had HBV genotype A, and the others were of genotype D. High levels of exposure and active HBV infection, and a moderate level of protection were observed. A stavudine/lamivudine/efavirenz or nevirapine regimen may be beneficial in controlling HBV in HIV patients. [Word count = 248]

Biography

Pascal Bessong, PhD, is a professor and head of Microbiology at the University of Venda, South Africa. He did his postdoctoral studies at the Myles H. Thaler Center for AIDS and Human Retrovirus Research, University of Virginia, USA. His research interests include HIV genetic diversity and drug resistance; and co-infections in HIV/AIDS.

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Characterizing the non-coding regions of 1918 'Spanish Flu' pandemic H1N1 influenza virus

Ruixue Wang and Jeffery K. Taubenberger
National Institutes of Health, USA

Influenza A virus can cause moderate and severe respiratory disease in humans and belong to the family of Orthomyxoviridae. It is a negative sense, single-stranded, segmented RNA virus. Influenza A viruses are classified into different subtypes based on the antigenic properties of the hemagglutinin (HA) and neuraminidase (NA) glycoproteins expressed on the surface of virus particles. The 1918 'Spanish flu' pandemic virus is an H1N1 subtype and caused a pandemic in 1918-1918 that killed approximately 2% of the world's population. To understand the origins and pathogenesis of 1918 influenza virus, decoding the full-length genomic sequences of the virus is critical. In this study, we successfully cloned and sequenced the non-coding regions of 1918 influenza A virus genome using RNA isolated from the frozen lung tissue of a 1918 influenza victim exhumed from a mass grave in Brevig Mission on the Seward Peninsula of Alaska in 1997. Infectious 1918 viruses were produced by reverse genetics. The growth kinetics of 1918 influenza viruses with the determined non-coding regions were compared with 1918 viruses containing the non-coding regions of two other viruses, A/WSN/33 (H1N1) and A/New York/312/2001 (H1N1).

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Analysis of chicken anemia virus genome: Evidence of intersubtype recombination

Yassir M Eltahir
University of Nyala, Sudan

Recombination is one of the evolutionary processes that shape the architecture of viral genomes. Ignoring the occurrence of recombination may influence the analysis of genetic data and the conclusions derived from it. Recombination analysis of the genomes of Chicken anemia virus (CAV), the causative agent of chicken infectious anemia is very limited. CAV putative intergenotypic recombinants which reported to occur in the VP1 region have suggested the speed up of CAV evolution. This fact is based on the previous classification of CAV sequences into three (I, II.III) genotypes. However, to date, there is no evidence of inter- or intrasubtype recombination between the recently reported four (A, B, C, D) CAV genotypes and the five (A1, A2, A3, D1, D2) subtypes of genome sequences. Phylogenetic analysis using the Mega software, together with a variety of computational recombination detection algorithms implemented in the RDP program were used to investigate CAV approximately full genomes. Statistically significant evidence of intersubtype recombination was detected in the parent-like and two putative CAV recombinant sequences. This event was shown to occur between CAV subgroup A1 and A2 sequences in the phylogenetic trees .This resulted in the sequence from subgroup A2 being located within subgroup A1. The recombination breakpoints detected in CAV subgroups sequences here were also found in the VP1 protein. The cumulative evidence of recombination breakpoints in the VP1 of the CAV genome may be indicative of favourable selection for recombination in this variable region as has been shown in HIV-1 isolates. Here, we report the first evidence of intersubtype recombination between CAV genome sequences which played a role in generating genetic diversity within the natural population of CAV.

Biography

Yassir M Eltahir has completed his PhD at the age of 35 years from Aristotle University of Thessaloniki, Greece and postdoctoral studies from Yangzhou University, China. He is the head department of preventive medicine and veterinary public health, faculty of veterinary science, University of Nyala, Sudan. He has published more than 10 papers in peer reviewed journals.

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Cancer is a side effect of evolution of viruses and bacteria

Gampa Vijayakumar[1], Srinivasareddy Bhavanam[1] and Rajakrupakar Nagavalli[2]
[1]Vikas institute of pharmaceutical sciences, India
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Any human organism is home to viruses and bacteria. However, viruses (as well as other intracellular parasites) are interested in continuous division of the host cells. Unlimited division of the host cells means unlimited expansion of the living space and possibility for unlimited multiplication of viral particles. For this, human Cell Cycle Regulation System has to be affected in such a way to induce unlimited division of host cells. The present work describes oncogenome`s model of cancer development, according to which, in order to stimulate cell division, viruses affect a gene (or several genes) during transduction of a signal for cell division from growth factor to cyclin/CDK system. At the same time, controlling gene (p53, RB etc.) function and switch to apoptosis are suppressed. Cell switch to cell division implies inability of the cell to carry out the functions of a differentiated cell. Spreading of viruses (bacteria) to the adjacent cells and forcing them into division, with simultaneous loss of differentiated cell functions, will become evident as the formation of malignant tumor or cancer.

Biography

Dr. G.Vijaykumar working as professor and principal in Department of Biotechnology at Vikas institute of pharmaceutical sciences, Rajahmundry has experience of research in microbiology and biotechnology, and has attended various conferences and seminars like FIP-CHINA, TURKEY, LISBON, INDIA.AASP-PHILIPPINES, FAPA-THAIWAN, INDONESIA. IPC-INDIA to share his research experience

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Effect of HEV ORF3 protein in liver injury

Peng Xiao
China Agricultural University, China

This study is to observe, analyze the mechanism about the immune defense function of liver during infecting hepatitis E virus (HEV) and the location of HEV ORF3 protein in the liver cells (ultrastructural level location); reveal more about the mechanism of the mast cell(MC) in HE, and the interaction between MC and HEV ORF3 protein. (Methods) Mongolian gerbils were used in the experiment, HE models were established by intraperitoneal injection of swine HEV. Acquiring blood samples and livers, respectively on 7days, 14days, 21days, 28days after infection, serum tests were checked the liver function, the level of HEV mRNA expression was evaluated with RT-polymerase chain reaction (PCR) ,a portion of the liver carried out the pathology testing (hematoxylin-eosin staining, transmission electron microscopy). Toluidine blue staining method was used to study the change of mast cells number in liver and Periodic acid Schiff reagent(PAS) was used to determine the contents of liver glycogen. And the expressions of Caspase-3 and NF-κB were detected in the liver by the immunohistochemical method. (Result)The result of RT-PCR showed the animal model was successfully established. Other results showed that, the swine HEV caused 1: challenged gerbils of clinical symptoms: by 7 days post-challenge, symptoms of nervous hyperaction; after 21 days, symptoms of piloerections, abnormal excitement, strong aggressivity. 2: The ratios of liver to body weight increased obviously in challenged gerbils, which indicated HEV can cause the hepatomegaly. 3:Pathological section with HE staining and TEM showed that HEV caused hepatic cell injury, autolysis, organelles disappear, glycogen increased. 4: PAS chroma analysis confirmed glycogen increased in challenged gerbils. 5: The result of immunohistochemistry methods confirmed that G-6-P positive signals which related with endoplasmic reticulum(ER) stress mainly distributed in liver cell cytoplasm and nucleus, image quantitative analysis found that, by 21d post-challenge, G-6-P expression level was the highest. And it showed that HEV can cause the endoplasmic reticulum damage. 6: The result of IHC also confirmed that the expression of HEV ORF3 protein mainly positioned in liver cell, the staining signals was located in liver cell cytoplasm and nuclear membrane. And the result of quantitative analysis showed that the expression of HEV ORF3 protein ballooned with the growth of time and on 28 days at the highest, and decreased thereafter. 7:At the same time, also detected the expressions of Caspase-3 related with the liver apoptosis proteins and the nuclear factor(NF)-κB by IHC. The positive signals of Caspase-3 mainly presented in the liver cell cytoplasm. And its expression in challenged- groups had a significantly increase compared with control group on 7 days, but thereafter, it had a significant drop; The positive signals of NF-κB mainly presented in liver cell cytoplasm and nucleus, and the expression in nucleus reached the highest on 21 days. An analysis suggested that HEV can induce cell apoptosis by NF-κB. 8: mast cells were determined by toluidine blue staining: the number of mast cells in portal area increased compared with the control group and significantly by 21 days, the results showed that the mast cells involved in inflammation immunologic reaction by HEV. (Conclusion) the above results suggested that: 1,the expression of HEV ORF3 protein increased after infection, showed that HEV ORF3 protein has played an important role in the pathological courses of lesion of liver tissue. 2,mast cell plays an important role in inflammation immunologic reaction. 3,it is confirmed HEV induce liver cell apoptosis via NF-κB signaling pathway, by measuring the transcription factors and apoptosis-related factors. 4,this suggests that endoplasmic reticulum stress might be one of the mechanisms of liver injury during infected HEV.

Biography

Peng Xiao is currently a Ph.D. student at China Agricultural University in the lab of Dr. Ruiping She and her field of interest is the relationship between HEV and mucosal immunity.

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STING regulates DNA-mediated innate immune signaling

Hiroyasu Konno, Guoxin Ni, Keiko Konno and Glen N. Barber
University of Miami Miller School of Medicine, USA

The innate immune system is critical for the early detection of invading pathogens and for initiating innate immune signaling, which induces the production of type I interferon (IFN). The innate immune system starts with recognition of pathogen associated molecule such as nucleic acid or bacterial cell walls. In case of RNA viruses, RIG-like helicases such as RIG-I or MDA5 recognizes RNA form RNA viruses and initiates innate immune system. However, how intracellular DNA is sensed and induces the production of IFN remains unknown. We showed before that STING (stimulator of interferon genes) is critical for the induction of IFN by intracellular DNA species produced by various DNA pathogens. Fibroblasts as well as antigen presenting cells such as macrophages and dendritic cells exposed to intracellular DNA, herpes simplex virus-1 (HSV-1) or bacteria Listeria monocytogenes, were found to require STING to initiate IFN production. STING activates IFN regulatory factor 3 (IRF3) through TANK-binding kinase 1 (TBK1) as well as NFkB. In resting cells STING localizes endoplasmic reticulum (ER) but move from ER to peri-nuclear region with TBK1 in response to intracellular DNA or HSV-1 infection. Interestingly, Brefeldin A, which inhibits transport of protein from ER to Golgi, blocks not only the STING trafficking but also IFN production. STING deficient mice are susceptible to HSV-1 infection. In addition, cytotoxic T-cell responses induced by plasmid DNA vaccination were reduced in STING deficient mice. Collectively, STING is essential for innate immunes system to sense intracellular DNA as well as DNA virus infection.

Biography

Hiroyasu Konno became Ph. D. at University of Tokyo, Japan in 2008 and joined to Dr. Glen Barber's laboratory for postdoc to study innate immune system, especially focusing on DNA mediated innate immune signaling.

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Accelerating Scientific Discovery

International Conference and Exhibition on
Orthopedics
August 13-15, 2012
Hilton Chicago/Northbrook, USA

International Conference and Exhibition on
Rheumatology & Therapeutics
August 14-15, 2012
Hilton Chicago/Northbrook, USA

2nd International Conference on
Proteomics & Bioinformatics
July 2-4, 2012
Embassy Suites Las Vegas, USA

3rd International Conference on
Biomarkers & Clinical Research
July 2-4, 2012
Embassy Suites Las Vegas, USA

International Conference and Exhibition on
Neurology & Therapeutics
May 14-16, 2012
Embassy Suites Las Vegas, USA

International Conference and Exhibition on
Biosensors & Bioelectronics
May 14-16, 2012
Embassy Suites Las Vegas, USA

3rd World Congress on
Bioavailability & Bioequivalence:
Pharmaceutical R & D Summit
March 26-28, 2012 Hyderabad Marriott Hotel, India

World Congress on
Gastroenterology & Urology
March 12-14, 2012
Omaha Marriott, Omaha, USA

International Conference and Exhibition on
Nanotechnology & Nanomedicine
March 12-14, 2012
Omaha Marriott, Omaha, USA

2nd International Conference on
Clinical & Experimental Opthalmology
March 5-7, 2012 Omaha Marriott, Omaha, USA

2nd International Conference on
Clinical & Experimental Dermatology
March 5-7, 2012 Omaha Marriott, Omaha, USA

2nd International Conference on
Clinical & Experimental Cardiology
March 5-7, 2012 Omaha Marriott, Omaha, USA

International Conference and Exhibition on
Biometrics & Biostatistics
March 5-7, 2012 Omaha Marriott, Omaha, USA

International Conference on
Metabolomics & Systems Biology
February 20-22, 2012
San Francisco Airport Marriott Waterfront, USA

2nd World Congress on
Pharmaceutics & Novel Drug Delivery Systems
February 20-22, 2012
San Francisco Airport Marriott Waterfront, USA

2nd International Conference on
Analytical & Bioanalytical Techniques
December 16-17, 2011 San Francisco, USA

2nd World Congress on
Diabetes & Metabolism
December 6-8, 2011 Philadelphia, USA

International Conference on
Pediatrics & Gynecology
December 6-8, 2011 Philadelphia, USA

International Conference and Exhibition on
Cell Science & Stem Cell Research
Nov29-Dec1, 2011 Philadelphia, USA

2nd World Congress on
Biotechnology
Nov29-Dec1, 2011 Philadelphia, USA

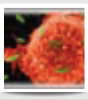
International Conference and Exhibition on
Vaccines & Vaccination
November 22-24, 2011 Philadelphia, USA

International Conference-cum-exhibition on
Agribusiness and Food Processing
November 20-22, 2011 Hyderabad, India

2nd World Congress on
Biomarkers & Clinical Research
September 12-14, 2011 Baltimore, USA

International Conference and Exhibition on
Pharmaceutical Regulatory Affairs
September 6-7, 2011 Baltimore, USA

International Conference and Exhibition on
Virology
September 5-7, 2011 Baltimore, USA



International Conference and Exhibition on
Cancer Science & Therapy
August 15-17, 2011 Las Vegas, USA

International Conference and Exhibition on
Clinical Research: Dermatology, Ophthalmology and Cardiology
July 5-6, 2011 San Francisco, USA

International Conference & Exhibition on
Proteomics & Bioinformatics
June 6-8, 2011 HICC, Hyderabad, India

International Conference & Exhibition on
Pharmaceutical Biotechnology
June 6-8, 2011 HICC, Hyderabad, India

2nd World Congress on
Bioavailability & Bioequivalence: Pharmaceutical R & D Summit
June 6-8, 2011 Las Vegas, USA

International Conference on
Pharmaceutics & Novel Drug Delivery Systems
June 7-8, Las Vegas,USA

World Congress on
Biotechnology
March 21-23, 2011 Hyderabad, India

International Conference on
Diabetes and Metabolism
December 13-14, 2010 Santa Clara, USA

International Conference and Exhibition on
Biomarkers & Clinical Research
November 22-23, 2010 Santa Clara, USA

International Conference and Exhibition on
Analytical and Bioanalytical Techniques:
Pharmaceutical R & D Summit
November 1-3, 2010 Hyderabad, India

International Conference and Exhibition on
Bioequivalence & Bioavailability 2010,
Pharmaceutical R & D Summit
March 1-3, 2010 Hyderabad, India

Integrating Glycomics with other Omics in Cancer Detection and Diagnosis Cancer Detection and Diagnosis
January 19-20, 2010
Stanford University School of Medicine

3rd World Congress of
Gene-2009
December 1-7, 2009

7th Annual World Congress of
International Drug Discovery Science & Technology
October 22-25

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July 18-20, 2009

1st CCSB-2009
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