PEERS ALLEY MEDIA

Peers Alley Media
1126 59 Ave East, V5X 1Y9, Vancouver BC, Canada
WhatsApp No: +1 (506) 909-0537

JOINT EVENT

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

SEPTEMBER 25-26, 2025

BERLIN, GERMANY

ADV. IMMUNOLOGY 2025 & FUTURE VIROLOGY 2025

SCIENTIFIC PROGRAM

DAY 01

THURSDAY

SEPTEMBER 25, 2025

08:00-08:30	**Registrations**
08:30-08:40	**Inaugural Ceremony**
Moderator:	**Sahar Jahanikia,** *Aspiring Scholars Directed Research Program (ASDRP), Human and Life Sciences Fremont, USA*

Sessions: Immunology | Medical Virology | Cancer Immunology | Immune System | Antiviral Research | Inflammation | Immune Mechanisms | Cytokines and Chemokines | Hypersensitivity, Asthma, and Allergy | Innate Immune Responses and Host Defense | Vaccines and Immunotherapy | Viral Vectors | Clinical and Diagnostic Virology | Emerging and Re-Emerging Viral Diseases

Distinguished Speaker Talks

Session Chair:	**Thomas Böldicke,** Helmholtz Centre for Infection Research, Germany
Session Chair:	**Patrizia Russo,** San Raffaele University and IRCCS San Raffaele Roma, Italy
08:40-09:00	Title: Cortical Grey Matter Volume Depletion Links with Neurological Sequelae in post COVID-19 “Long Haulers” **Ted L. Rothstein MD,** *George Washington University, USA*
09:00-09:20	Title: Exercise Training Improves Symptoms and Immune Cell Phenotype in Long COVID Patients **Asghar Abbasi,** *The Lundquist Institute for Biomedical Innovation at Harbor-UCLA Medical Center, USA*
09:20-09:40	Title: The Promise of Machine Learning: Using Seismic’s IMPACT Platform to Design IgE Cleaving Enzymes for Chronic Treatment of Allergy **Alex Pellerin,** *Seismic Therapeutic, USA*
09:40-10:00	Title: Laboratory Diagnosis of Bovine Coronavirus Infections **Binu T. Velayudhan,** *University of Georgia, USA*
10:00-10:20	Title: Microglia: The Drunken Gardeners of Early Adversity **Arie Kaffman,** *Yale University School of Medicine, USA*

GROUP PHOTO 10:20-10:30

REFRESHMENT BREAK 10:30-10:50

10:50-11:10	Title: A Global Network Analysis of COVID-19 Vaccine Distribution to Predict Breakthrough Cases among the Vaccinated Population **Sahar Jahanikia,** *Aspiring Scholars Directed Research Program (ASDRP), Human and Life Sciences Fremont, USA*
11:10-11:30	Title: IL-18 Primes $CD44^{high}CD8^{+}$ T and NK Cells for Expansion and Differentiation Dependently on Notch Signalling and the Action is Regulated by PGE2 **Haruki Okamura,** *Osaka Medical and Pharmaceutical University & Hyogo College of Medicine, Japan*
11:30-11:50	Title: Mobilization of Endogenous $CD34^{+}/CD133^{+}$ Endothelial Progenitor Cells by Enhanced External Counter Pulsation for Treatment of Refractory Angina **Joseph John Tartaglia,** *New York Medical College, USA*
11:50-12:10	Title: Riboflavin Kinase Binds and Activates Inducible Nitric Oxide Synthase to Reprogram Macrophage Polarization **Qiujing Yu,** *Sichuan Provincial People's Hospital, University of Electronic Science and Technology of China, China*
12:10-12:30	Title: Predicting Immune Dysfunction and Clinical Outcomes through TTV-Virome Analysis in COPD Patients **Patrizia Russo,** *San Raffaele University and IRCCS San Raffaele Roma, Italy*
12:30-12:50	Title: *Immunotherapy with Recombinant T-Cells and Recombinant* Antibodies against Solid Tumors **Thomas Böldicke,** *Helmholtz Centre for Infection Research, Germany*
GROUP PHOTO 12:50-13:00	
LUNCH BREAK 13:00-13:40	
13:40-14:00	Title: The Link Between Metabolic Syndrome and Cellulite - Our Clinical Experience from Athens Medical Center Lifestyle Medicine Program **Sotirios Adamidis,** *Athens Medical Center, Greece*
14:00-14:20	Title: The Role of Nutrients and Microbial Imbalance in Leaky Gut Syndrome **Maria Emilia Soares Martins dos Santos,** *Federal University of Triângulo Mineiro, Brazil*
14:20-14:40	Title: Samuel Darling Returns from the Tomb - Advanced AIDS still Challenges Medicine: A Case Report **Gabriel Moreira Accetta,** *Universidade Estadual Paulista "Júlio de Mesquita Filho – UNESP, Brazil*

14:40-15:00	Title: The Evolving Landscape of Alcoholic Liver Disease: Molecular Insights and Treatment Innovations **Palash Mandal,** *Charotar University of Science and Technology, India*
15:00-15:20	Title: Prevalence of Chronic Rhino Sinusitis in Patients with Concha Bullosa **Rishabh R. Reddy,** *Vydehi Institute of Medical Sciences and Research Centre, India*
15:20-15:40	Title: The Impact of Walnut Butter Consumption and Nutrition Education on Weight Management, Body Composition and Endothelial Health in Adolescents with Overweight and Obesity **Lisa Aschenberg,** *Texas Woman's University, USA*
15:40-16:00	Title: *In silico* Study of Fungal Immunomodulatory and Antiviral Proteins of Edible Mushrooms **Aveek Samanta,** *Prabhat Kumar College, India*
REFRESHMENT BREAK 16:00-16:20	
16:20-16:40	Title: Molecular Analyses of MEFV Gene Mutation Variants in Turkish Population **Darya Farhoomand Aksoy,** *Ankara University, Turkey*
16:40-17:00	Title: Alternative Root Canal İrragation Solutions which is Non Cytotoxic and High Antibacterial Effectivenes // in the Case of *in vitro* Study which is held in Laboratory **Tahir Ataözden,** *Biruni University, Turkey*
17:00-17:20	Title: Infection of Neonates with *Staphylococcus aureus* and Methicillin-Resistant *Staphylococcus aureus* at Dormaa Presbyterian Hospital, Ghana **Jerome Adinkrah Obeng,** *Kwame Nkrumah University of Science and Technology, Ghana*
17:20-17:40	Title: Safety and Efficacy of Oral Edible Bird's Nest Supplementation: Anti-Inflammatory and Immunomodulatory Effects in Arabian Horses **Khalid Obaid Al-Khaldi,** *Veterinary Services Administration, Mounted Police Headquarter, Sultanate of Oman*
17:40-18:00	Title: HIV-1 Budding Control by Inducible Inhibition of ESCRT-III **Cécile Boscheron,** *University Grenoble Alpes, France*
NETWORKING	
END OF DAY 1	

SCIENTIFIC PROGRAM DAY 02

FRIDAY

SEPTEMBER 26, 2025

08:30-08:40	**Introduction**
Moderator	**Mohamed I. Husseiny Elsayed,** City of Hope National Medical Center, USA

Sessions: Immunology | Medical Virology | Cancer Immunology | Immune System | Antiviral Research | Inflammation | Immune Mechanisms | Cytokines and Chemokines | Hypersensitivity, Asthma, and Allergy | Innate Immune Responses and Host Defense | Vaccines and Immunotherapy | Viral Vectors | Clinical and Diagnostic Virology | Emerging and Re-Emerging Viral Diseases

Distinguished Speaker Talks

Session Chair:	**Giorgio Mangino,** Sapienza University of Rome, Italy
08:40-09:00	Title: MIPC, the Universal Antiviral Cells **Fawzy Abdelatty,** *Heidelberger Center for Cellular Therapy, Germany*
09:00-09:20	Title: Flipons and the Genetics of Innate Immunity **Alan Herbert,** *InsideOutBio, USA*
09:20-09:40	Title: Extracellular Vesicles as Mediators of Inflammation in an *in vitro* Model of Psoriasis **Giorgio Mangino,** *Sapienza University of Rome, Italy*
09:40-10:00	Title: Oral Vaccine for Type 1 Diabetes **Mohamed I. Husseiny Elsayed,** *City of Hope National Medical Center, USA*
10:00-10:20	Title: Etiopathogenesis of Nasal Polyps Elucidated from Histopathological Examinations of Nasal Polyps of Different Size **Per Leganger Larsen,** *Copenhagen University and Capital Region Hospitals, Denmark*
GROUP PHOTO 10:20-10:30	
REFRESHMENT BREAK 10:30-10:50	
10:50-11:00 (Poster)	Title: Precision Mapping of Influenza A Virus RNA Dynamics and Antiviral Responses using an Advanced CRISPR- Cas12 Platform **Tran Anh Tu,** *Chang Gung University, Taiwan*

11:00-11:10 (Poster)	Title: Macrophage-Specific PHGDH Protects against MAFLD by Suppressing TAK1 **Zhe Wang,** *University of Electronic Science and Technology of China, China*
11:10-11:30	Title: Distinct Tumor Immune Responses to Nanosecond Pulsed Electric Fields (nsPEFs) Determine Immunity **Stephen J Beebe,** *Frank Reidy Research Center for Bioelectrics; Old Dominion University Norfolk Va, USA*
11:30-11:50	Title: The CTCF Anatomy of Human Chromosomes: Implications for Enhancer-Promoter Communication **Colin Logie,** *Radboud Institute for Molecular Life Science (M850), The Netherlands*
11:50-12:10	Title: Epigenetic Regulation and Nuclear Actin Dynamics in Early and Differentiated T Helper Cells **Moran Titelbaum,** *Bar-Ilan University, Israel*
12:10-12:30	Title: Impacts During Viral Infections on Immune Mechanisms and Effects of Nutraceuticals and Pharmacological **Anju Kaushal,** *New Zealand Organization for Quality, New Zealand*
GROUP PHOTO 12:30-12:40	
LUNCH BREAK 12:40-13:20	
13:20-13:40 (Video Presentation)	Title: The Potential Roles of IL-1β, IL-6, and RIPK3 in the Pathogenesis of Stevens Johnson Syndrome/Toxic Epidermal Necrolysis **Omer Iqbal,** *Loyola University Stritch School of Medicine, USA*
13:40-14:00	Title: Ultrasonic-Assisted Coprecipitation of Magnetite Nanoparticles (Fe_3O_4) and Functionalization of Fe_3O_4@Sio_2-NH_2 for Potential Magnetic Resonance Imaging Contrast Enhancement **Jahaziel Amaya,** *Universidad Antonio Nariño, Colombia*
14:00-14:20	Title: Exploring Hepatitis B Virus Replicative Space **Pooja Bhatia,** *Shiv Nadar Institution of Eminence, India*
14:20-14:40	Title: Lymphocytosis and Heteropenia in Rainbow Agama Lizards: A Haematological Study from Zaria, Nigeria **Olufisoye Olusegun OJO,** *University of Ibadan, Nigeria*

14:40-15:00	Title: Ergosterol & Quercetagetin as Dual-Action Anti-Cancer Agents: Decoding Steroid Metabolism *via* AR/ESR1 Crosstalk
	Yujiao Chen, *Zhejiang University School of Medicine, China*
15:00-15:20	Title: A Rare Case of Sepsis Caused by *Klebsiella oxytoca* and *Aeromonas hydrophila*
	Lixin Hua, *Nantong University, Wuxi Huishan District People's Hospital, China*
15:20-15:40	Title: Influenza Virus Regulation of Smad Signaling in Macrophages: Theoretical and Experimental Approach
	Kareem Awad, *University of Turku, InFLAMES, Finland*
15:40-16:00 (Video Presentation)	Title: Development of *ex vivo* Analysis for Examining Cell Composition, Immunological Landscape, Tumor and Immune Related Markers in Non-Small-Cell Lung Cancer
	Vadim V. Kozlov, *Novosibirsk State Medical University, Novosibirsk Regional Clinical Oncology Dispensary, Russian Federation*

PANEL DISCUSSION

END OF DAY 2

ADV. IMMUNOLOGY 2025
FUTURE VIROLOGY 2025

DAY 01

JOINT EVENT

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

BERLIN, GERMANY

SEPTEMBER 25-26, 2025

SPEAKER TALKS

Cortical Grey Matter Volume Depletion Links with Neurological Sequelae in post COVID-19 "Long Haulers"

Ted L. Rothstein MD

Professor of Neurology, George Washington University, USA

COVID-19 infection has been associated with neurologic sequelae even among those patients with mild respiratory illness. Patients experiencing cognitive symptoms such as mental confusion or "brain fog," as well as other neurological symptoms after 8 or more weeks define "long COVID" or "post COVID condition (PCC)". Information is limited regarding the underlying neuroanatomic changes responsible for their cognitive deficits.

Advanced automated volumetric segmentation is an atlas-based Magnetic Resonance (MR) technique which can analyze, segment, and quantify volumes of key brain structures important for memory that are not identified with conventional Brain MR imaging. Results are highly consistent and reproducible allowing for the development of an age and sex adjusted range, which can be applied to long COVID-19 infected patients, who are then compared to a normative database drawn from healthy controls.

A retrospective study from George Washington University analyzed 52 consecutive long term COVID-19 infected patients evaluated at a median of 85 days following laboratory confirmation of COVID-19 infection. Criteria for inclusion included age 60 years or less, having mild respiratory symptoms not requiring oxygen supplementation, hospitalization or ventilatory support, and were experiencing long-term neurologic symptoms. Volumetric analysis was obtained for whole brain and forebrain parenchyma, cortical grey matter (CGM), hippocampus and thalamus. The results documented a statistically significant volume depletion limited to CGM which is the likely source for their persistent neurologic sequelae.

There have been more than 400,000 scientific peer-reviewed papers published regarding various aspects of COVID-19 infection, but few have documented their neuroanatomic effects. This study contributes to an understanding of the neuroanatomic basis for prolonged

effects of COVID-19 infection on patients' neurocognitive and neurological function, with the potential for producing serious long-term personal effects on their quality of life, productivity, and presents ongoing challenges to our public health systems.

Biography

Ted L. Rothstein MD is Professor of Neurology at the Department of Neurology, George Washington University in Washington, DC, USA. He received his Medical Degree at University College of Medicine, Richmond Virginia, served his Medical Internship at the Queens Hospital, Honolulu Hawaii, and Neurology Residency at University of Washington, Seattle Washington. He is Professor of Neurology at George Washington University, specializing in Cognitive Disorders and Demyelinating Diseases. His current research is focused on the use of advanced imaging techniques in a variety of Neurologic Disorders.

Exercise Training Improves Symptoms and Immune Cell Phenotype in Long COVID Patients

Asghar Abbasi, Chiara Gattoni and **William Stringer**

The Lundquist Institute for Biomedical Innovation at Harbor-UCLA Medical Center, USA

Objectives: Fatigue is a prominent feature of long COVID (LC) and may be related to several pathophysiologic mechanisms, including immune hyperstimulation. Aerobic endurance exercise training may be a useful therapy, with appropriate attention to preventing post-exertional malaise.

Methods: Cardiopulmonary exercise testing, physical activity steps/day, 6MWD, quality of life (QOL) and symptom questionnaires were performed pre- and post-training (10 weeks, 20 1-hour sessions per week). Immune cell phenotypes were assessed at rest and peak exercise, pre- and post-training.

Results: 14 participants completed training: age 56±11y, 52% females, low normal pulmonary function (FEV1 93%±18), BMI 31.9±8kg/m^2. Training compliance was 96%. Post-training exercise tolerance, QOL, fatigue, mental acuity and depression symptoms improved, but not steps/day or 6MWD. The circulating number of CD3+, CD4+, CD19+, CD14++CD16-, and CD16++CD14+ monocytes, along with CD56+ cells (measured *via* flow cytometry), increased during acute exercise (from rest to peak) and were not adversely affected or augmented by exercise training. Plasma concentrations of TNF-α, IL-6, IL-8, IL-10, INF-γ, and INF-λ were within normal limits at baseline and remained unaffected by exercise or training.

Conclusion: Aerobic endurance exercise training in individuals with LC delivered beneficial effects on cardiorespiratory fitness, quality of life, anxiety, depression, and fatigue without detrimental effects on immunologic function.

Biography

Dr. Abbasi is an Assistant Professor of Medicine at the David Geffen School of Medicine at UCLA and a Principal Investigator at the Lundquist Institute for Biomedical Innovation at Harbor-UCLA Medical Center. He received a PhD in “Exercise Immunology” from University Hospital Tübingen and completed postdoctoral trainings in Neuroimmunology (The Hertie Institute for Clinical Brain Research at University Hospital Tübingen, Germany), Neurobiology of Aging (MIND institute at UC Irvine), and Respiratory Medicine and Physiology (The Lundquist Institute for Biomedical Innovation at Harbor-UCLA Medical Center). His research centers on the immunological mechanisms by which exercise improves chronic lung disease such as COPD and long COVID.

The Promise of Machine Learning: Using Seismic's IMPACT Platform to Design IgE Cleaving Enzymes for Chronic Treatment of Allergy

Alex Pellerin

Seismic Therapeutic, USA

The dysregulation of humoral immune mechanisms results in pathogenic IgE production that contributes to a range of allergic and atopic diseases such as food allergies, acute anaphylaxis, allergic asthma, allergic rhinitis and chronic spontaneous urticaria. We present a novel Fc-fused bacterially-derived IgE protease that was engineered using a proprietary machine learning enabled platform to reduce immunogenicity and improve manufacturability while maintaining potency. The protease selectively cleaves IgE, eliminating it from circulation and the cell-surface which provides a novel therapeutic opportunity to treat IgE-mediated inflammation.

The engineered Fc-fused IgE protease was identified using our proprietary IMPACT platform and was characterized *in vitro* to determine its ability to cleave IgE using MSD and flow cytometry-based cleavage assays. Immunogenicity was determined using T cell proliferation assays. Pharmacokinetics, pharmacodynamics and in vivo efficacy were tested using relevant preclinical models.

The engineered Fc-protease selectively cleaves IgE while maintaining stability and demonstrating low immunogenicity. The protease shows extended pharmacokinetics and efficacy in preclinical models of local and systemic acute anaphylaxis. Additionally, we show a clear PK/PD relationship and reduction of endogenous IgE in non human primates.

Given its ability to simultaneously address multiple aspects of IgE pathogenesis and its efficacy in preclinical models of anaphylaxis, the engineered Fc-protease offers a new approach to targeted therapy for allergic and atopic diseases where IgE is a key driver.

Biography

Alex Pellerin trained in immunology and autoimmunity and received his PhD from Boston University. He has 15 years experience in the pharma industry working on both large and small molecules. During his time at Biogen he helped lead the Litifilimab program which is now in Phase III clinical trials for Lupus. Currently he is the director of pre clinical translation at Seismic Therapeutic.

Laboratory Diagnosis of Bovine Coronavirus Infections

Binu T. Velayudhan and **Shaun van den Hurk**

Athens Veterinary Diagnostic Laboratory, University of Georgia, USA

Bovine coronavirus (BCoV) causes respiratory and gastroenteric infections in cattle. The viruses causing both forms of clinical presentations belong to *Betacoronavirus* genus. Laboratory diagnosis of BCoV involves detection of viruses or viral antigens and immune response. Real-time reverse-transcription polymerase chain reaction (qRT-PCR). electron microscopy, virus isolation, and direct or indirect immuno-assays are commonly used for the laboratory diagnosis BCoV infections. Virus isolation using cell cultures is very challenging as the virus grows poorly on cells and requires multiple blind passages. Our laboratory primarily uses TaqMan-based qRT-PCR for the detection of BCoV. Serum neutralization test (SNT) using Madin-Darby bovine kidney cells is used for the detection of BCoV antibodies. Based on data from the past five years on clinical submissions to our laboratory, the prevalence rate of respiratory BCoV detections varied from year to year with an average of 6.7%, which peaked in 2020 to 17.9%. Other pathogens associated with respiratory diseases included *Histophilus somni* (*50.4%*), *Pasteurella multocida* (*32%*), *Mycoplasma* spp. (*30.1%*), *Mannheimia haemolytic*a (*16.7%*), bovine respiratory syncytial virus (14.9%), bovine viral diarrhea virus (7.1%), bovine herpes virus-1 (1.8%), and parainfluenza vurus-3 (0.7%). From clinical cases of diarrhea including calf and adult diarrhea, enteric BCoV prevalence averaged around 1.5%. Other pathogens detected from gastroenteric cases included rotavirus, BVDV, cryptosporidium, and salmonella. Data from the last five years showed a seroprevalence of BCoV antibodies around 45%. The respiratory and enteric variants of BCoV are genetically and antigenically very similar and it's difficult to differentiate between the two, especially using serological assays. Recent approaches on the development of point-of-care testing for BCoV include reverse-transcription loop-mediated isothermal amplification and surface enhanced Ramen spectroscopy with deep learning algorithm. Early and rapid detection,

and molecular characterization of BCoV from clinical cases are critical in developing effective control and intervention strategies to tackle this economically important pathogen.

Biography

Dr. Binu Velayudhan is the Director of the Athens Veterinary Diagnostic Laboratory and Associate Professor of infectious diseases at the University of Georgia, Athens, Georgia, USA. He is a veterinarian and a virologist by training. He is board-certified in virology by the American College of Veterinary Microbiologists. Dr. Velayudhan has long years of experience in the field of infectious disease diagnostics. In the past, he worked as the Assistant Director of North Carolina Veterinary Diagnostic Laboratory System in Raleigh, NC and as the Section Head of Virology & Molecular Diagnostics at the Texas A&M Veterinary Medical Diagnostic Laboratory in Amarillo, TX. He serves as an Associate Editor of *Frontiers in Veterinary Science* and as a Guest Editor of *Pathogens*. He also serves on the NIH-ZRG1 AIDC grant review panel. Dr. Velayudhan is actively involved in the development and validation of novel multiplex assays for the rapid detection of animal pathogens.

Joint Event
ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY
&
FUTURE VIROLOGY
September 25-26, 2025 | Berlin, Germany

Microglia: The Drunken Gardeners of Early Adversity

Arie Kaffman[1], Sahabuddin Ahmed[1], Baruh Polis[1], Sumit Jamwal[1], Basavaraju G. Sanganahalli[2], Christian Bowers[1], Zoe MacDowell Kaswan[1], Lauryn Giuliano[1] and **Fahmeed Hyder[2,3]**

[1]Department of Psychiatry, Yale University School of Medicine, USA
[2]Department of Radiology & Biomedical Imaging and Magnetic Resonance Research Center Yale University, USA
[3]Department of Biomedical Engineering, Yale University, USA

Synaptic pruning is an activity-dependent process in which non-functional and redundant synapses are marked for elimination by glial cells, such as microglia and astrocytes. This process peaks in the rodent hippocampus during the second and third weeks of life and is essential for normal synaptic maturation, connectivity, and cognitive function later in life. Using limited bedding (LB) as a mouse model of early life adversity (ELA), we recently found significant deficits in microglial-mediated synaptic pruning in the hippocampus of male and female pups on postnatal day 17 (P17). These deficits were not present in adolescent P33 mice, when levels of synaptic pruning are substantially lower. Abnormal microglial-mediated synaptic pruning was associated with reduced synaptic connectivity and impaired hippocampal function, which were more pronounced in adolescent LB males compared to their LB female littermates. Transient ablation of microglia during the second and third weeks of life resulted in similar sex-specific deficits in synaptic connectivity and hippocampal function. Additionally, chemogenetic activation of microglia during this period rescued the synaptic and cognitive deficits observed in adolescent LB males. Female LB mice, but not their male littermates, were able to upregulate synaptic pruning in astrocytes, providing a potential mechanism to explain their ability to compensate for the microglial-mediated phagocytic deficits. Together, these findings suggest a novel role for glial cells in mediating sex-specific hippocampal deficits in a mouse model of ELA. The objectives of this

presentation are: 1) to review these published data alongside additional unpublished studies from our group, and 2) to discuss these findings in the context of human clinical studies and their potential implications for developing novel diagnostic and treatment strategies.

Biography

Dr. Kaffman is a physician-scientist and an associate professor in the Department of Psychiatry at Yale University. He provides psychiatric treatment for veterans with complex trauma and manages a basic neuroscience laboratory at Yale. His research focuses on the impact of early life adversity (ELA) on microglial function in mice and examines how disruptions in this function during development can lead to alterations in neural connectivity and increased vulnerability to subsequent challenges, such as traumatic brain injury and additional stress in adulthood. As the Principal Investigator on several NIH-funded grants, he integrates molecular, cellular, genomic, pharmacological, and behavioral approaches with imaging techniques, including resting-state functional magnetic resonance imaging and high-resolution diffusion magnetic resonance imaging in rodent models. This translational approach facilitates a direct comparison between findings in rodents and humans, with the goal of leveraging this knowledge to develop innovative diagnostic and treatment strategies.

A Global Network Analysis of COVID-19 Vaccine Distribution to Predict Breakthrough Cases among the Vaccinated Population

Sahar Jahanikia, Pragyaa Bodapati, Eddie Zhang, Sathya Padmanabhan, Anisha Das and **Medha Bhattacharya**

Aspiring Scholars Directed Research Program (ASDRP), Department of Biological, Human and Life Sciences Fremont, USA

As the COVID-19 pandemic rapidly spread in late 2019 and early 2020, numerous vaccines were developed to combat the virus. Despite these advancements, the emergence of new variants such as Omicron and Delta continues to pose challenges, leaving some countries and populations more vulnerable to future outbreaks. This research aims to analyze global susceptibility to COVID-19 outbreaks, visualize the spread of the virus, and predict which countries face heightened risk from new variants based on key factors. We developed interactive maps to track the pandemic's spread and identify high-risk countries by analyzing vaccination rates. Using machine learning models, including binary classification, K-nearest neighbors (KNN), and neural networks, we predicted each country's risk level. Our neural network model demonstrated the highest accuracy, achieving a 94% success rate in classifying countries as high-risk or low-risk based on vaccine coverage and government response stringency. Additionally, we constructed network graphs inspired by the Albert-László Barabási model to visualize connections between countries based on vaccination percentages. These graphs illustrate correlations between vaccination rates and the likelihood of future COVID-19 outbreaks, offering a unique perspective on global vulnerabilities. Our findings highlight the importance of equitable vaccine distribution and the role of stringent policies in mitigating the risk of new variants. This research not only provides a comprehensive analysis of current risks but also establishes a framework for predicting and preventing future outbreaks, contributing to a more proactive global pandemic response.

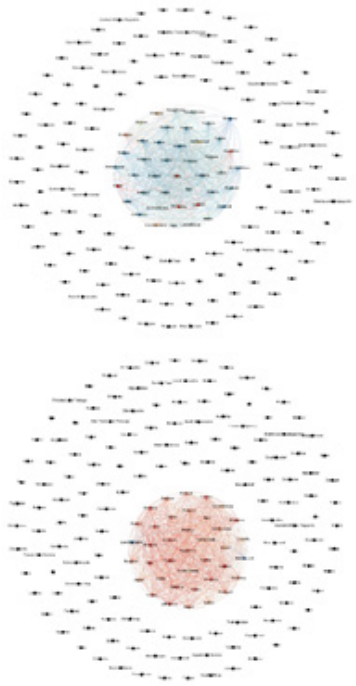


Fig: Pfizer (top) and Moderna (bottom) networks. The networks were developed using the Albert Figure 5. Pfizer (top) and Moderna (bottom) networks. The networks developed using Albert Barabasi's model demonstrate the similarities of each country's vaccination percentages.

Vaccine	Countries at Risk
Pfizer (countries with this vaccine are at a higher risk because most of their population has the vaccine—around 75%.)	Japan, Austria, Finland, South Africa, Bulgaria, Ireland, Estonia, Norway, Czechia, Denmark
Moderna (countries with this vaccine and at "risk" are not at a very high level of risk, because the highest vaccination rate is around 30%; in other words, not very many people are vaccinated with this vaccine. As long as other vaccine candidates are used, these countries should not be at high risk.)	Latvia, Netherlands, Spain, Canada, US, Switzerland, Italy, Luxembourg, Lichtenstein
Johnson & Johnson (countries with this vaccine and at "risk" are not at a very high level of risk, because the highest vaccination rate is around 10%; in other words, not very many people are vaccinated with this vaccine.)	Romania, Bulgaria, Latvia, South Africa
Sinovac (countries with this vaccine are at a somewhat high risk because almost half of their population is vaccinated—around 45%.)	Chile, Ecuador
Sinopharm (countries with this vaccine are at a higher risk because most of their population has the vaccine—around 50%.)	Argentina
Sputnik (countries with this vaccine are at a somewhat high risk because almost half of their population is vaccinated—around 45%.)	Argentina
Oxford (countries with this vaccine and at "risk" are not at a very high level of risk, because the highest vaccination rate is around 30%; in other words, not many people are vaccinated with that vaccine.)	Argentina, Nepal, South Korea, Malta
Covaxin (countries with this vaccine and at "risk" are not at a very high level of risk, because the highest vaccination rate is around 26%; in other words, not many people are vaccinated with this vaccine.)	India

Table: Summary of risks for each vaccine candidate. This table summarizes the risk for each vaccine candidate and the countries most affected by each candidate.

Biography

Sahar Jahanikia is a research neuroscientist and serial entrepreneur dedicated to advancing healthcare through innovation. She is the Co-founder of AlzoHealth, Founder of CognoTrain, and a pioneer in Alzheimer's healthcare technology. As Founder & Director of Jahanikia NeuroLab and Research Scientist Advisor at ASDRP, she leads cutting-edge projects addressing critical challenges in neuroscience. With extensive experience at UCSF and Stanford School of Medicine, Sahar focuses on impactful research, including using machine learning to predict future pandemics and studying long-term COVID-19, brain fog, and their effects on daily life. Her interdisciplinary approach bridges neuroscience and technology, driving solutions that transform healthcare and improve quality of life.

IL-18 Primes CD44^{high}CD8^{+} T and NK Cells for Expansion and Differentiation Dependently on Notch Signalling and the Action is Regulated by PGE2

Haruki Okamura[1,3], Wen L[1,3], Shinji Takai[1], Denan Jin[1], KyousukeYamanishi[3] and **Yoshimasa Tanaka[2]**

[1]Department of Innovative Medicine, Graduate School of medicine, Osaka Medical and Pharmaceutical University, Japan
[2]Center for Medical Innovation, Nagasaki University, Japan
[3]Hyogo College of Medicine, Japan

CD44 is regarded as one of the markers of memory T cells who experienced exposure to antigens. On the other side, CD44 has been regarded as a marker of stem cells. Significant frequency of CD8^{+} T cells in animals, including germ free mice, who have not experienced foreign antigens, are known to highly express CD44, like NK cells. In the present study, we observed that mouse splenic CD44^{high}CD8^{+} T cells as well as NK cells, but not CD^{44}lowCD8^{+}T cells, expressed functional IL-18 receptors, α and β chains, together with IL-2 receptors CD122/CD132, CD62L, CCR7, and chemokine receptor CXCR3. In response to IL-18 stimulation, phosphorylated NF-κB, S6/p70 protein, eIF-1, BCl-XL, and ATP levels, were up-regulated together with augmented expression of CD25 (IL-2 receptorα chain), Notch-related c-Kit (CD117), and PD-1 in these cells, which progressed into G1/S phase in the cell cycle. IL-18-primed cells, expressing three IL-2 receptor chains, robustly expanded in response to IL-2, and underwent differentiation into effector cells with strong cytotoxicity and ability to produce cytokines. While IL-18-primed cells retained high levels of Tcf-1, the expanding cells induced by IL-2 diminished them, suggesting the decrease of stemness in them. Stimulation of CD44^{high}CD^{8+} T cells with IL-18 upregulated Notch-1 receptor and c-Myc, and inclusion of γ-secretase inhibitors attenuated the effect of IL-18 on them. Notably, the effects of IL-18 and IL-2 on NK cells and CD44^{high} CD8^{+} T cells were inhibited by prostaglandin E2 (PGE2) agonists. In tumor models, administration of IL-18 increased the accumulation of CD8^{+} T cells in both the lymph nodes and tumors, and administration of IL-18 and EP4 antagonists significantly inhibited tumor growth.

These results suggested that IL-18 promotes self-renewal and functional differentiation of $CD44^{high}CD8^{+}$T cells and NK cells in a Notch signalling-dependent manner. The action of IL-18 was suggested to be influenced by PGE2.

Biography

- 1946 Born in Japan
- 1976 Assistant prof. Dept of Microbiol. Hyogo College of Medicine
- 1997 Prof. Laboratory of Host Defense, Hyogo College of Medicine
- 2011 Prof. Dept. of Cancer Immunol.
- 2018 Retired

Mobilization of Endogenous CD34⁺/CD133⁺ Endothelial Progenitor Cells by Enhanced External Counter Pulsation for Treatment of Refractory Angina

Joseph J Tartaglia[1], Carol A Eisenberg[1] and **Joseph D DeMarco[2]**

[1]Department of Medicine, New York Medical College, USA
[2]Summit Health, Florham Park, USA

Adult stem cell therapy *via* intramyocardial injection of autologous CD34⁺ stem cells has been shown to improve exercise capacity and reduce angina frequency and mortality in patients with refractory angina (RA). However, the cost of such therapy is a limitation to its adoption in clinical practice. Our goal was to determine whether the less costly, less invasive, and widely accessible, FDA-approved alternative treatment for RA patients, known as enhanced external counter pulsation (EECP), mobilizes endogenous CD34⁺ stem cells and whether such mobilization is associated with the clinical benefits seen with intramyocardial injections. We monitored changes in circulating levels of CD34⁺/CD133⁺ and CD34⁺/KDR⁺⁻ cells in RA patients undergoing EECP therapy and in a comparator cohort of RA patients undergoing an exercise regimen known as cardiac rehabilitation. Changes in exercise capacity in both cohorts were monitored by measuring treadmill times (TT), double product (DP) scores, and Canadian Cardiovascular Society (CCS) angina scores between pre- and post-treatment treadmill stress tests. Circulating levels of CD34⁺/CD133⁺ cells increased in patients undergoing EECP and were significant ($\beta = -2.38$, $p = 0.012$) predictors of improved exercise capacity in these patients. CD34⁺/CD133⁺ cells isolated from RA patients could differentiate into endothelial cells, and their numbers increased during EECP therapy. Our results support the hypothesis that mobilized CD34⁺/CD133⁺ cells repair vascular damage and increase collateral circulation in RA patients. They further support clinical interventions that can mobilize adult CD34⁺ stem cells as therapy for patients with RA and other vascular diseases.

Biography

Joseph Tartaglia completed his MD from the University of Rome, La Sapienza in 1984, his residency in Internal Medicine at Wakefield, Montefiore Einstein in 1988, and his Fellowship in Cardiovascular Disease at North Shore University Hospital, Weill Cornell Medicine in 1990. Boarded in Internal Medicine, Cardiovascular Disease, and Geriatric Medicine, he is an Assistant Prof. of Medicine at New York Medical College and an adjunct Prof. at Weill Cornell. The author of numerous papers in his field, he has been a pioneer of Enhanced External Counter Pulsation since 1995 when he served on the steering committee of the first international registry for Enhanced External Counter Pulsation. He has teaching positions at Westchester County Medical Center, Weill Cornell Medical College, Greenwich Hospital, a Yale, New Haven Health division.

Riboflavin Kinase Binds and Activates Inducible Nitric Oxide Synthase to Reprogram Macrophage Polarization

Qiujing Yu, Xiao Shan and **Zemin Ji**

Department of Health Management Center & Institute of Health Management, Translational Clinical Immunology Key Laboratory of Sichuan Province, Sichuan Provincial People's Hospital, University of Electronic Science and Technology of China, China

Riboflavin kinase (RFK) is essential in riboflavin metabolism, converting riboflavin to flavin mononucleotide (FMN), which is further processed to flavin adenine dinucleotide (FAD). While RFK enhances macrophage phagocytosis of Listeria monocytogenes, its role in macrophage polarization is not well understood. Our study reveals that RFK deficiency impairs M(IFN-γ) and promotes M(IL-4) polarization, both *in vitro* and *in vivo*. Mechanistically, RFK interacts with inducible nitric oxide (NO) synthase (iNOS), which requires FMN and FAD as cofactors for activation, leading to increased NO production that alters energy metabolism by inhibiting the tricarboxylic acid cycle and mitochondrial electron transport chain. Exogenous FAD reverses the metabolic and polarization changes caused by RFK deficiency. Furthermore, bone marrow adoptive transfer from high-riboflavin-fed mice into wild-type tumor-bearing mice reprograms tumor-associated macrophage polarization and inhibits tumor growth. These results suggest that targeting RFK-iNOS or modulating riboflavin metabolism could be potential therapies for macrophage-related immune diseases.

Biography

Qiujing Yu is a professor in the Department of Health Management Center & Institute of Health Management, Sichuan Provincial People's Hospital. Prof. Yu developed a keen interest in the field of the regulation of serine-glycine-one-carbon (SGOC) metabolism in macrophages during the pathological processes of inflammation-related diseases, achieving a series of innovative results: uncovering the key mechanisms by which SGOC metabolism regulates the production of type I interferons and pro-inflammatory cytokines mediated by PRR signaling pathways; discovering that SGOC metabolism inhibits the IFNγ-STAT1 pathway and promotes the IL4-STAT6 pathway, thereby regulating macrophage polarization to promote tumor growth. In the past five years, Prof. Yu has published 9 papers as corresponding or co-corresponding author, including in *Cell Metab, Cell Mol Immunol, Cell Rep, Nat Commun*, and *Mol Cell*, providing new targets for the treatment of viral infections, septic shock, and tumors.

Joint Event
ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY
&
FUTURE VIROLOGY
September 25-26, 2025 | Berlin, Germany

Predicting Immune Dysfunction and Clinical Outcomes through TTV-Virome Analysis in COPD Patients

Patrizia Russo[1], Laura Vitiello[1], Francesca Milani[1], Dolores Limongi[1] Fabrizio Maggi[2], Guido Antonelli[3] and **Stefano Bonassi[1]**

[1]Department of Human Sciences and Quality, San Raffaele University and IRCCS San Raffaele Roma, Italy
[2]Laboratory of Virology, National Institute for Infectious Diseases, Lazzaro Spallanzani IRCCS, Italy
[3]Department of Public Health and Infectious Diseases, Sapienza University of Rome, Italy

Introduction: The non-pathogenic Torquetenovirus (TTV) is the main representative of the Anelloviridae family. Studies have shown that immunocompromised patients exhibit higher TTV viral loads in the blood compared to healthy controls (≥ 4 log10 copies/mL), which is associated with increased clinical frailty and poor prognosis (patients with COPD). The primary aim of this study is to determine whether TTV viral load (≥ 4 log10 copies/mL) in the blood can serve as a reliable marker of immune system functionality and, consequently, as an indicator of a more favorable or unfavorable prognosis in a cohort of COPD patients.

Methods: Serum samples from 102 COPD patients were collected and analyzed using real-time polymerase chain reaction (RT-PCR) to quantify TTV viral load. PBMCs, stored in liquid nitrogen, were used for immunophenotypic analysis of circulating T lymphocytes (CD45, CD3, CD4, CD8, CD45RA, CD197, CD183, CD184, CD186, CD127, CD25) thus allowing the evaluation of cytotoxic T lymphocytes, regulatory T cells, and Th1, Th2, and Th17 subsets.

Statistical analysis was performed using SPSS software, version 23. The heterogeneity of contingency tables was assessed using Fisher's exact test. Differences in data distribution were calculated using the non-parametric Mann-Whitney test. Correlations between non-normally distributed continuous variables were evaluated using Spearman's rho coefficient.

Results: Among the patients, 62.75% had TTV viremia levels > 4 log10 copies/mL. Higher TTV viremia correlated with a reduction in CD8 lymphocytes and a significantly lower 5-year survival probability.

Conclusion: Patients with TTV levels ≥ 4 log10 copies/mL exhibited the lowest survival probability, likely due to reduced immunological activity. This study raises key scientific questions regarding the role of TTV in COPD. Specifically, it explores whether TTV may serve as a potential marker of poor prognosis in COPD and whether rehabilitation strategies for these patients could be tailored based on immunological status and/or viremia levels.

Biography

Patrizia Russo works at San Raffaele University in Rome as an Associate Professor of Pathology and History of Medicine.

She is UO (Operational Unit) responsible for the grant: TTV-virome prediction of dysregulated immunity and clinical differential diagnosis n. B53D2300376006/G53D23000700001 AWARDED by MUR (Ministry of University and Research) in Rome, Italy, from 2023 to 2025.

Her h-index is 39 according to Scopus, and she has 187 published works. Her Scopus Author ID is 57192333601, her ORCID ID is 0000-0003-1745-7827, and her RESEARCH ID is J-8767-2016.

Immunotherapy with Recombinant T-Cells and Recombinant Antibodies against Solid Tumors

Thomas Böldicke[1] and **Ana Maria Waaga-Gasser**[2]

[1]Department Structure and Functions of Proteins, Helmholtz Centre for Infection Research, Germany
[2]Renal Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, USA

Two immunotherapeutic approaches have come to the forefront of clinical interest for targeting intracellular neoantigens in solid tumors: recombinant T-cell and recombinant antibody based therapies. Most promising T cell-based therapies targeting sell surface proteins or intracellular neoantigens use T cells expressing a chimeric antigen receptor (CAR) or a recombinant complete TCR (TCR-T cell), respectively. Seven CAR T-cell therapies and one TCR-T cell therapy have been FDA-approved. The most promising recombinant T-cells targeting neoantigens in solid tumors are T-cells expressing a recombinant complete TCR (TCR-T cell) or T-cells expressing the synthetic T cell receptor and antigen receptor (STAR) or heterodimeric TCR-like CAR (T-CAR). STAR and T-CAR comprises a double-chain TCRαβ-based receptor with variable regions of immunoglobulin heavy and light chains (VH and VL) or scFv fragments, respectively. STAR and T-CAR mediates strong and sensitive TCR-like signaling, and STAR-T and T-CAR cells exhibit less susceptibility to dysfunction and better proliferation than traditional CAR-T cells comprising intracellularly the CD3ζ and costimulatory TCR domains fused to a hinge region, a transmembrane domain and a scFv recognizing a cell surface protein. There are major limitations to recombinant T cell therapy that still must be addressed including limited persistence, poor trafficking and tumor infiltration and the immunosuppressive microenvironment which is assembled by immunosuppressing cells infiltrating solid tumors. Recombinant antibodies targeting MHC/neopeptide complexes include TCR-like antibodies and bi-specific T-cell engagers (BITEs). 100 BITEs are in clinical investigation and 7 FDA approved. The effectivity of BITEs and CARs are similar but CARs demonstrated more toxicities. In addition intrabodies against neoantigens seems to be a very promising tool to inhibit cancer growth too, because of easy selection and high speci-

ficity compared to TCR-like antibodies and soluble TCRs. Recently intrabodies against TLR2 and TLR9 were developed inhibiting human pancreatic tumor celllines.

Biography

Associate Professor Dr. Thomas Böldicke received his PhD 1982 at the Max-Planck-Institut of Molecular Genetics, Berlin. He started his carrier as post doc at the German Research Centre for Biotechnology (GBF, Braunschweig, Germany) in the Department of Genetics and Cell Biology by John Collins. Now he is senior scientist at the Helmholtz Centre for Infection Research (HZI, former GBF) and project leader intrabodies. He developed recombinant antibodies against tumor antigens, particularly against tumour angiogenesis, rhabdyomyosarcoma and recently against TLR2 and TLR9 in pancreatic cancer. He edited two books: “Protein Targeting Compounds” with Springer (2016) and “Antibody Engineering” with IntechOpen (2017). He has published 51 manuscripts. Over 10 years he gave lectures at the Technical University in Braunschweig about immunology, cancer development and immunotherapies. He is in the editorial board of the journal Antibodies as academic editor and Frontiers in Immunology.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

The Link Between Metabolic Syndrome and Cellulite -Our Clinical Experience from Athens Medical Center Lifestyle Medicine Program

Sotirios Adamidis

Athens Medical Lifestyle Medicine Department, Athens Medical Center, Greece

Objectives: To explore the potential association between Metabolic Syndrome (MetS) and cellulite, focusing on shared pathophysiological mechanisms.

Scope: MetS significantly increases the risk of cardiovascular disease and Type 2 diabetes mellitus. Subcutaneous adipose tissue (SAT) distribution, in certain locations, is also a risk factor for cardiometabolic disease. Cellulite is a common condition affecting mostly womens' skin texture. Despite being considered primarily a cosmetic issue, cellulite shares multiple common features with MetS.

Methods: Shared clinical and pathogenetic features between MetS and cellulite are discussed.

Results: Common pathophysiological processes between MetS and cellulite are outlined in Table 1.

Pathophysiological process	MetS	Cellulite
Inflammation	Systemic inflammation from adipose tissue dysfunction	Local inflammation from adipose hypertrophy and fibrosis
Hormonal imbalance	Estrogen affects fat distribution	Estrogen affects fat distribution and connective tissue structure
Adipose tissue dysfunction	Visceral adiposity contributing to insulin resistance and inflammation	Subcutaneous fat hypertrophy and altered extracellular matrix, leading to typical skin changes
Microcirculatory changes	Microcirculation impairment, affecting blood flow and lymphatic drainage, contributing to tissue edema and fibrosis.	

Table 1. Shared pathophysiological processes between MetS and Cellulite

We have observed tirzepatide's beneficial effects in women with polycystic ovarian syndrome and increased androgens; apart from improving insulin resistance and reducing inflammation, tirzepatide treatment also led to improved skin appearance with less cellulite, skin tags and discolored patches, all of which are also signs of insulin resistance.

Conclusions: MetS and cellulite share several pathophysiological processes. Our hypothesis is that the association between SAT and cardiometabolic risk may also apply to SAT in areas where cellulite appears, independent of location. Further research is essential to elucidate the pathophysiology of these conditions and their common processes, identify clinically relevant biomarkers and develop targeted therapies.

Biography

Sotiris Adamidis is the Director of Internal Medicine in Athens Medical Center, President of the European Society of Diabetes, Metabolic Syndrome and Obesity (EsoDiMeSO), and Director of newly established Lifestyle Medicine Department in Athens Medical Center.

He received his MD from Athens University School of Medicine. He is board certified in Internal Medicine with special interest in metabolism, diabetes, obesity and related conditions. He is member of American Diabetes Association and Southern Medical Association.

He is well published in medical journals, lay press, and serves on the editorial board of numerous journals. He is invited as medical expert in television, often commenting on health and other matters, commenting on conditions such as hypercholesterolemia, cardiometabolic diseases and COVID-19 related conditions.

He has authored two books "Metabolic Syndrome, Obestiy and Diabetes" and "From Metabolic Syndrome to Cellulite: The Medical Solution", as well as 2 poetry books.

Joint Event
ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY
&
FUTURE VIROLOGY
September 25-26, 2025 | Berlin, Germany

The Role of Nutrients and Microbial Imbalance in Leaky Gut Syndrome

Maria Emilia Soares Martins dos Santos[1], Ana Beatriz Marcari[1], Aline Dias Paiva[2] and **Claudio Roberto Simon[3]**

[1]Department of Biochemistry, Pharmacology and Physiology, Federal University of Triângulo Mineiro, Brazil
[2]Department of Microbiology, Immunology and Parasitology, Federal University of Triângulo Mineiro, Brazil
[3]Department of Structural Biology, Federal University of Triângulo Mineiro, Brazil

Introduction: The gut microbiota plays a crucial role in maintaining health and preventing disease. Chronic stress, alcohol consumption, and antibiotic use can cause dysbiosis, a microbial imbalance that significantly affects the host's health. In addition to these factors, diet is one of the most significant element affecting the composition and function of the gut microbiota.

Objectives: To provide a comprehensive examination of the literature on the influence of diet on dysbiosis and increased intestinal permeability.

Methods: A non-systematic narrative review was conducted by searching scientific articles published on databases: MEDLINE [*via* PubMed], Portal BVS Saúde, Scielo, Web of Science, Scopus, and Embase over the past five years. Filters were applied for terms such as “diet and dysbiosis”, “diet and Leaky Gut Syndrome”, “dysbiosis and Leaky Gut Syndrome”. A manual search was also conducted to compile a list of relevant articles in the field.

Results: High intake of simple sugars, saturated fats, and processed foods has been strongly associated with activation of immune responses, dysbiosis and the development of increased intestinal permeability, or leaky gut syndrome. In contrast, diets rich in plant-based foods like vegetables, fruits, nuts, fish, and poultry, with moderate consumption of red meat, promote a more diverse and beneficial gut microbiota. Additionally, supplementation with prebiotics, probiotics, omega-3 fatty acids, polyunsaturated fats, and key vitamins

has been shown to positively influence gut health by modifying microbial activity, improving gut barrier integrity, and enhancing microbial diversity.

Conclusion: The work shows the importance of personalized dietary strategies in managing gut microbiota to enhance gut diversity and quality and ensure individuals' comprehensive health.

Biography

B.Sc. in Biomedicine/Medical Laboratory Technology from the University of Uberaba (2003); PhD in Biochemistry (2008) from the Department of Biochemistry and Immunology at the Faculty of Medicine of Ribeirão Preto, University of São Paulo (FMRP/USP), and Case Western Reserve University, Cleveland, OH, USA. Postdoctoral fellowship (2014) in Metabolism field (Pathophysiology and Molecular Pharmacology) at the Joslin Diabetes Center - School of Medicine, Harvard University. Associate Professor at the Federal University of Triângulo Mineiro, in the field of Clinical Biochemistry. Research focuses on Control and Regulation of Metabolism in several physiological or pathological situations: Action and therapeutic potential of bioactive compounds.

Samuel Darling Returns from the Tomb - Advanced AIDS still Challenges Medicine: A Case Report

Gabriel Moreira Accetta, Douglas Otomo Duarte, Giovanna Beatrice de Sousa, Mariana Menezes Lourenço, Alexandre Naime Barbosa and **Maria Aparecida Marchesan Rodrigues Kobayasi**

Universidade Estadual Paulista "Júlio de Mesquita Filho" – UNESP, Brazil

Samuel Darling was an American pathologist, born in 1872, who during his work in Panama in 1905, described an invasive cell pathogen causing an unlicensed endemic illness, which was pursuing with whitish granulomatous lesions in lungs, liver, spleen, and bone marrow. At the time, Darling noted that this microorganism, along with the lesions it caused, had histological characteristics similar to those found in cells affected by the AMASTIGOTA form of protozoa of the genus Leishmania spp., And thus presented them as a new pathogenic species than described as a microorganism oval shaped, wrapped in a colorless capsule, like the protozoan Leishmania, and named *Histoplasma capsulatum*.

Male patient, 63 years old, from Piraju -SP, has been referred in emergency room at the Botucatu Hospital das Clínicas complaining of inappetence, weakness and abdominal pain associated with diarrhea, as well as weight loss of about 10 kg in about 3 months.

Patient also reveals that 1 month ago he had started with cough associated with night sweats. Regarding the clinical picture, research and diagnosis is carried out with HIV sero-positive testing. In determining its symptomatology with external adjacent exams such as high digestive endoscopy an oroesophageal moniliasis and colonoscopy examination with colon biopsy histopathological diagnosis (HSTP) of intestinal leishmaniosis.

Opted to hospitalize the patient, for treatment: moniliasis oroesophagian and leishmaniasis and investigation of other possible opportunistic infections.

He carried out an active investigation with identification of: Cryptococcus neoformans, tuberculosis and CMV.

In instituting research of the pulmonary for OI with bronchoscopy procedure was identified in pulmonary biopsy, alveoli filled with vacuotalized macrophages, “foamy”, showing in the cytoplasm numerous small gem spores, characteristic of pulmonary histoplasmosis. Given this fact, the possibilities of diagnosis error in the external examination of the colon HTP were served, due to similarities known in the histopathological aspect of intestinal infection by histoplasma and leishmania in their form of amastigote. Being requested from the Pathology Service of the Hospital das Clínicas de Botucatu, the revision of the external laboratory blades. Concluding that the first sample performed was also histoplasmosis in the intestinal site.

This clinical case brings up a reflection on that even after 10 decades since the discovery of the etiological agent *Histoplasma capsulatum* it is also possible to find diagnostic failures that may affect the patient's erroneous treatments. Especially in patients living with AIDS. Therefore, so far, the year 2025, there is also the reflection of risky arbitrics by health professionals about the realization of inaccurate diagnoses.

Biography

Pasqualotto AC, Lana DD, Godoy CSM, et al. Single High Dose of Liposomal Amphotericin B in Human Immunodeficiency Virus/AIDS-Related Disseminated Histoplasmosis: A Randomized Trial. *Clin Infect Dis*. 2023;77(8):1126-1132. doi:10.1093/cid/ciad313.

Duarte, Eitel. "Histoplasmose." Memórias do Instituto Oswaldo Cruz 43 (1945): 457- 494.

The Evolving Landscape of Alcoholic Liver Disease: Molecular Insights and Treatment Innovations

Palash Mandal

P D Patel Institute of Applied Sciences, Charotar University of Science and Technology, India

Alcohol-related liver disease (ALD) remains a significant global health concern, accounting for approximately 0.9% of worldwide mortality. Among ALD-related deaths, liver cancer constitutes nearly 19%. Steatosis, characterized by excessive fat accumulation in the liver, is one of the earliest manifestations of ALD and affects up to 90% of chronic heavy alcohol consumers. Individuals with blood alcohol concentrations of 10 mmol/L are at particularly high risk for alcohol-induced steatosis, and nearly half of these cases may progress to alcoholic steatohepatitis (ASH). While early stages of ALD—such as steatosis and ASH—are potentially reversible with timely intervention, advanced stages, including fibrosis, cirrhosis, and hepatocellular carcinoma (HCC), result in irreversible liver damage.

The pathogenesis of ALD predominantly targets hepatocytes and is exacerbated by mechanisms such as chronic gut dysbiosis and increased intestinal permeability. These changes facilitate the translocation of lipopolysaccharides (LPS) into the liver, triggering Kupffer cell activation and promoting hepatic inflammation. Despite numerous investigational treatments—including herbal remedies, probiotics, prebiotics, antibiotics, anti-TNFα agents, and fecal microbiota transplantation—no FDA-approved pharmacological therapy currently exists for ALD. In early stages, complete alcohol abstinence may reverse liver damage; however, advanced cases often require liver transplantation, with sustained sobriety essential post-operatively.

Emerging microbiota-focused strategies offer promising therapeutic potential. Prebiotics like aged garlic extract support the growth of beneficial gut bacteria, thereby enhancing gut barrier function. This synergistic relationship between prebiotics and probiotics contributes to the restoration of gut eubiosis, improved intestinal permeability, and attenuation of liver inflammation. Such approaches may represent a novel and effective direction in the management and treatment of ALD.

Biography

Prof. Palash Mandal, PhD, is Professor in the Department of Biological Sciences at P. D. Patel Institute of Applied Sciences of CHARUSAT University. In 1995, Dr. Mandal completed his PhD degree at Bose Institute and Jadavpur University. He obtained post-doctoral and research associate training respectively from the departments of Immunology and Pathobiology of Cleveland Clinic, USA (2003-2010).

His research interests include: TLR4 signalling, oxidative stress and inflammation in both alcoholic and non-alcoholic liver diseases and to explore novel therapeutic targets for the treatment of this disorder, role of gut microbiota in liver disease, innate immunity and cytokines in liver diseases, mechanisms and biomarkers of apoptosis in liver disease.

Additionally, he serves as an Associate Editor (Frontiers in Molecular Biosciences, Frontiers in Gastroenterology), Academic Editor (PLOS ONE, Mediators of Inflammation, Gastroenterology research & practice, Discovery Applied Science, etc) & Review Editor for numerous reputable journals.

Joint Event
ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY
&
FUTURE VIROLOGY
September 25-26, 2025 | Berlin, Germany

Prevalence of Chronic Rhino Sinusitis in Patients with Concha Bullosa

Rishabh R. Reddy[1], M Nabeel[2] and **Sriram Rebala[1]**

[1]Vydehi Institute of Medical Sciences and Research Centre, India
[2]Department of ENT, Vydehi Institute of Medical Sciences and Research Centre, India

Background and Purpose: This study aims to investigate the prevalence of chronic rhino sinusitis in patients with concha bullosa, a common anatomical variation characterised by pneumatization of the middle turbinates. Chronic rhino sinusitis is an inflammatory disorder of the paranasal sinuses and the linings of the nasal passages that lasts for 12 weeks or longer. The incidence of chronic rhino sinusitis was assessed among patients with diagnosed concha bullosa, focusing on the presence of symptoms, and severity. There seems to be a strong relationship between concha bullosa and the development of chronic rhino sinusitis.

AIM:

1. To screen the patients of concha bullosa for chronic rhino sinusitis.
2. To find out of the percentage of chronic rhino sinusitis in patients with concha bullosa.
3. To determine the sinuses affected and their respective sides.

Materials and Methods Study Design: This is a retrospective study to investigate the incidence of chronic rhino sinusitis in patients diagnosed with concha bullosa. The study was conducted at Vydehi institute of medical science and research centre, Whitefield, Bangalore and the data from 01/02/23 to 01/09/23 were analyzed. Medical records of patients with diagnosed concha bullosa were identified through electronic medical records and imaging databases (CT scan paranasal sinuses). Inclusion criteria consisted of patients with a confirmed diagnosis of concha bullosa based on radiological imaging (CT scans) and clinical assessment. Exclusion criteria: patients with previous sinus surgeries and malignancy.

Observation and result: A total of 73 patients who were diagnosed with concha bullosa were screened for the presence of chronic rhinosinusitis and associated deviated nasal septum. Out of the 73 patients, 34 were males and 39 were females The maximum number of patients were seen in the age group of 20-39 followed by 40-59, and least in the age of group 0-9 years. All of the concha are located in the middle turbinate: 30.1% on right, 24.7% on left and 45.2% had a bilateral presentation. DNS was associated with concha bullosa in 59 patients (80.8%). Which shows us a strong correlation between the two. Out of the 73 patients who presented with concha bullosa 57 patients had chronic rhinosinusitis out of which 75.4%(43) showed mucosal thickening of the epithelial lining and 24.6%(14) of the patients showed a polypoidal mass in their paranasal sinuses. 46.6% of the patient scan revealed CRS ipsilaterally while 53.4 had a contralateral association with the concha bullosa. In our study, the prevalence of sinusitis among the patient population was observed, revealing distinct patterns of sinus involvement. Maxillary sinusitis was the most common, affecting 44.8% of the individuals. Frontal sinusitis was observed in 13.9% of cases, ethmoid sinusitis in 26.4%, and sphenoid sinusitis in 14.9%.

Conclusion: The study's methods focused on retrospectively assessing the incidence of chronic rhino sinusitis in patients diagnosed with concha bullosa. In the above study it is concluded that concha bullosa plays major role in development of chronic rhino sinusitis. Thus after confirming the presence of concha bullosa practitioner should keep in mind the possibility of development of chronic rhino sinusitis.

Biography

Rishabh R. Reddy is a final-year MBBS student at Vydehi Institute of Medical Sciences and Research Centre, Bangalore Karnataka, currently awaiting his final exams before commencing his internship in three months. With a strong passion for clinical medicine and research, he looks forward to gaining hands-on experience and contributing to the medical field during his internship.

Rishabh has published a research paper titled "Prevalence of Chronic Rhinosinusitis in Patients with Concha Bullosa" and is actively working on several ongoing research projects in the fields of ENT, Cardiothoracic and Vascular Surgery (CTVS), and Orthopedics. His academic interests are fueled by a deep commitment to advancing medical knowledge and improving patient care.

In addition to his academic pursuits, Rishabh has demonstrated leadership and social responsibility as the Chartered Joint Secretary of the Rotaract Club of Medikardia, Bangalore, and as the State Director of GAIMS. In these roles, he successfully organized numerous hypertension and diabetes awareness camps and blood donation drives, contributing to the health and well-being of the community.

Rishabh is particularly passionate about research, continuously seeking opportunities to collaborate with fellow researchers, engage with innovative scientific findings, and expand his knowledge base. He aspires to pursue a career in surgery, with a specific focus on orthopaedics, and is dedicated to improving his skills in this specialized field. Furthermore, Rishabh is eager to explore global healthcare practices, comparing diagnostic approaches, treatment strategies, and patient care models in different countries.

The Impact of Walnut Butter Consumption and Nutrition Education on Weight Management, Body Composition and Endothelial Health in Adolescents with Overweight and Obesity

Lisa Aschenberg, Kasey Lewis, Randah Shrouro, Keith Crabtree, Wanyi Wang, Kathleen Davis and **Shanil Juma**

Texas Woman's University, USA

The purpose of this study was to examine if combination of regular consumption of a walnut-based spread and nutrition education in comparison to nutrition education alone would improve fat to muscle mass ratio and have beneficial effects on cardiovascular health in adolescents with overweight or obesity. A total of 80 adolescents between the ages of 13 and 17 years consented to participate in this study. The data presented is a subset of the participant data, as the study has recently concluded. The reported findings are preliminary and will be updated once all participant data has been analyzed.

Participants were randomized into either control (n=31) or treatment (n=24) groups and received nutrition education at baseline and midpoint visits. The treatment group consumed 45 grams of walnut spread product for 120 days. Dual energy x-ray absorptiometry (DEXA) was performed on all participants at baseline and final visit to evaluate body composition. Endothelial vasodilator function was assessed using EndoPAT at baseline and final visits, and endothelial health biomarkers vascular cellular adhesion molecule 1 (VCAM-1), intercellular adhesion molecule 1 (ICAM-1), and P-selectin were evaluated at baseline, midpoint, and final visits for all participants.

After four-week intervention the treatment group saw an increase in lean mass, although not statistically significant. Reactive Hyperemia Index (RHI) results from EndoPAT testing indicated the control group had improved endothelial function compared to treatment group. The treatment group saw favorable changes in each individual biomarker from baseline to final visit, although this difference was not statistically significant.

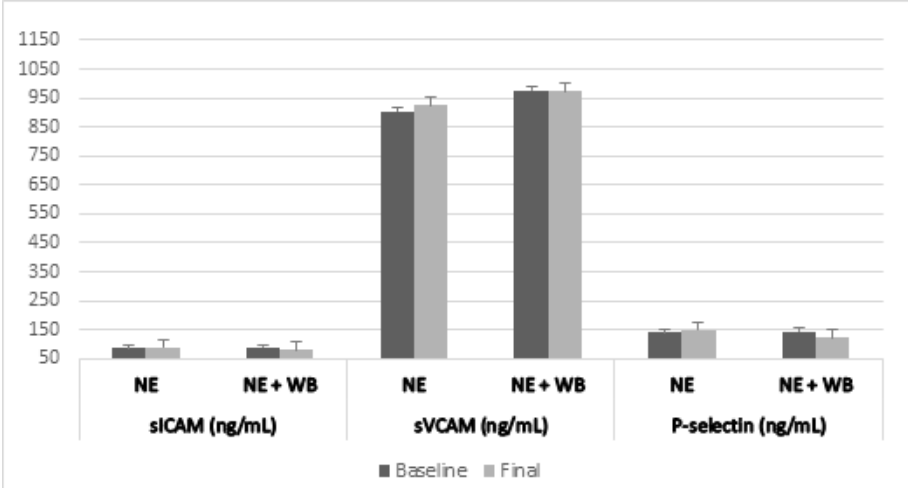


Figure 1. Effects of NE+WB on CAMs

Note. *Analysis of biomarkers via Magpix. Mean ± SEM. $p < 0.05$. NE n = 19; NE+WB treatment n = 17.*

	NE		NE+WB	
Body Composition	Baseline	Final	Baseline	Final
BMI kg/m²	32.4 ± 1.1	32.7 ± 1.3	31.8 ± 1.4	32.5 ± 1.6
Lean Mass (kg)	51.5 ± 1.8	49.7 ± 1.8	49.5 ± 2.6	53.3 ± 2.8
VAT Mass (g)	600 ± 45.7	581 ± 44.3	600 ± 50.3	606 ± 52.5
VAT Volume (cm³)	648 ± 49.4	628 ± 47.9	650 ± 54.4	656 ± 56.8

Table 1. Effects of NE+WB on Body Composition

Note. Body composition. Values represent Mean ± SEM. NE *n* = 31; NE+WB *n* = 24.

Due to small sample size, it was harder to detect significant differences in the statistical results. Data from all participants, currently being analyzed, may show more beneficial impact of the walnut spread in the diet.

Biography

Lisa Aschenberg is a doctorate student at Texas Woman's University. Her focus in nutrition education, prevention and treatment of disease encompasses interests in education and treatment strategies, overweight and obesity and related diseases, inflammation, and management of age-related conditions. Her current research project focuses on the nutrient profile and bioactive compounds in walnuts and their ability to favorably impact feelings of satiety, weight management, lipid biomarkers, and cardiovascular function. Recent publications include the chapter contribution "Satiety Sensation and Its Associated Food Compositions and Flavors" in the newly published book "Flavor-Associated Applications in Health and Wellness Food Products."

In silico Study of Fungal Immunomodulatory and Antiviral Proteins of Edible Mushrooms

Aveek Samanta[1] and **Ayyagari Ramlal**[2]

[1]Department of Botany, Prabhat Kumar College, India
[2]School of Biological Sciences, Universiti Sains Malaysia (USM), Malaysia

The mushrooms are highly nutritious and have numerous physiological properties. They are a well-known source of many bioactive and nutritional compounds with immense applicability in both the pharmaceutical and food industries. They are widely used to cure various kinds of ailments in traditional medicines. They have a low amount of fats and cholesterol and possess a high number of proteins. Immunomodulators have the ability which can improve immunity and act as defensive agents against pathogens. One such class of immunomodulators is fungal immunomodulatory proteins (FIPs). FIPs have potential roles in the treatment of cancer, and immunostimulatory effects and show anti-tumor activities. FIPs from edible mushrooms have been compared and analyzed on their conserved motifs. Phylogenetic analysis was also carried out using the selected FIPs. The conserved motif analysis revealed that some of the motifs are identified as FIPs while some are novel sequences. Phylogenetic analysis was also carried out using the FIPs. The molecular basis for selective affinity for the major histocompatibility (MHC) class 1 protein (1CE6) with some bioactive molecules obtained from P. *sajor-caju* also have been studied by using *in silico* molecular docking approaches. The results show the selected bioactive molecules have the potential ability to bind with MHC class 1 protein with different affinity and could be novel antiviral agents. From the present observations, it can be also be concluded that the identification and functional characterization of the proposed novel motifs and the potential roles of FIPs for developing newer drugs.

Biography

Dr. Aveek Samanta is an assistant professor of the Department of Botany, Prabhat Kumar College, Contai, West Bengal. He has qualified NET and GATE examination in 2010 and completed PhD in 2015. He has awarded as Young Scientist Award in Indian Plant Science Congress, Chennai in 2019. He is a life member of Indian Science Congress. He has been teaching Botany from past 10years and published 05 books and 04 book chapters and 24 international publications. Currently he has 2 registered PhD scholar working under him. He has two projects from IISC, Bangalore and IIT Madras. He has awarded for prototype development from WBSCST, Dept of Science and Technology in 2023. He has registered three patents and one trademark and one copyright. He works as visiting professor at Kobe University, Kobe, Japan from 1st October to 31st October, 2024.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

Molecular Analyses of MEFV Gene Mutation Variants in Turkish Population

Darya Farhoomand Aksoy[1], Rahime Aksoy[2] and **Ebru Us[3]**

[1]Faculty of Science, Department of Biology, Ankara University, Turkey
[2]Faculty of Medicine, Department of Hematology, Ankara University, Turkey
[3]Faculty of Medicine, Department of Medical Microbiology, Ankara University, Turkey

Background: Familial Mediterranean fever (FMF) is an autosomal recessive autoinflammatory disease primarily affecting individuals of Turkish, Armenian, Arab, and non-Ashkenazi Jewish descent, caused by mutations in the MEFV gene. The aim of this study was to review the common genotype distributions of MEFV variants and mutations in the Turkish population and evaluate rare mutations.

Methods and Results: The study included 2984 patients who applied to Ankara University Ibni Sina Hospital Immunology Laboratory with clinical suspicion of FMF between 2004 and 2014. The data of patients from different regions of the country who were followed up in the immunology-rheumatology clinic with clinical suspicion and presumptive diagnosis of FMF were evaluated retrospectively. Patients were tested for all mutations in Exon 2 and Exon 10, including M694V, M680I, M694I, V726A, E148Q and R202Q. There were 2504 patients with FMF variant. According to genotyping, R202Q (*n*=1567, 39.2%) was the most common mutation. The most common co-variant was the R202Q/M694V genotype (*n*=507,16.98%). Allele frequencies for MEFV mutations were as follows: R202Q (*n*=1567, 39.2%), M694V (*n*=1004, 25.1%),E148Q (*n*=463, 11.5%), M680I (*n*=354, 8.8%), V726A (*n*=319, 7.9%), A744S (*n*=51, 1.2%), R761H (*N*=41, 1.0%),P706P (*N*=25, 0.6%), E167D (*N*=23, 0.5%), M694I (*N*=23, 0.5%), and K695R (*N*=20, 0.5%).

Conclusion: This research revealed the prevalence of both common and rare MEFV gene mutations in Turkish FMF patients in various age groups. R202Q was the most prevalent mutation.

Biography

Darya Farhoomand Aksoy is a Ph.D. candidate in Biology (Biotechnology) at Ankara University, Turkey. Her academic journey began with a Bachelor's degree in Cell and Molecular Biology (Microbiology) from Islamic Azad University of Zanjan, followed by a Master's in Internal Medicine and Diagnostic Immunology at Ankara University. Her research interests span molecular biology, biotechnology, immunology, bioinformatics, and plant science. She has actively participated in several international and national conferences, presenting her work on gene polymorphisms, genome-wide analyses, and molecular mechanisms related to health. Her publications reflect on commitment to advancing knowledge in these fields. Currently, her doctoral research focuses on the regenerative effects of ATRA on healthy keratinocyte skin cells and its regulation of key ERAD-associated proteins. She is dedicated to translating molecular research into actionable solutions to tackle challenges in health, with a focus on driving innovation and advancing interdisciplinary collaboration.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

Alternative Root Canal İrragation Solutions which is Non Cytotoxic and High Antibacterial Effectivenes // in the Case of *in vitro* Study which is held in Laboratory

Tahir Ataözden[1], Semanur Özüdoğru[2], Muhammet Yayla[3] and **Mustafa Çoşkun[3]**

[1]Biruni University, Turkey
[2]Istanbul Gelişim University, Turkey
[3]Kafkas University, Turkey

Aim: Root Canal İrragation solutions and medicine in endodontic treatment is available for to use alternative materials (N acetyl cysteine, boric acid, (chitosan) different concentrations mouse fibroblast cell L929 for to Check the Cytotoxicity and Q. aureus

Biofilms for to check antibacterial effectiveness of *in vitro* aspect evaluation was aimed.

Equipment Method: Cell culture test for experiment groups; Chitosan 2048ug/ml- 4ug/ml 10 in different concentration, N Acetyl cysteine (NAC) 50 mg/ml- 0.39 mg/ml between 8 in different concentration, Boric Acid (NA) 64 mg/ml- 0.125 mg/ml between 10 Sodium in different concentration Hypochlorite (NaOCl) 10.5%-5.25 %-2.625% rates 3 different prepared in concentration was created. Antimicrobial test for article concentrations Chitosan 1- 0.002mg/ml, NAC 25- 0.195 mg/ml, Boric acid 32- 0.0625mg/ml aspect was carried out. Prepared microplate at 37ºC 18 hour incubation was released. Study Results group intra- and groups inter- data by comparison analysis was done.

Findings: Positive control group the one which... To NaOCl according to all experiment groups more is cytotoxic. Chitosan 128 microgram/ml also first acute toxic the effect of has shown. Q. Aureus on MIC value whereas 0.031 mg/ml is. Antimicrobial dose on the border toxic has been found. N Acetyl Cysteine (NAC) MIC value 1,563 mg/ml while first 24 per hour 25-50 mg/ml in doses toxic It has been found. That is antimicrobial dose on the border toxic It is not has been observed. Boric Acid MIC value 4 While mg/ml. This at the rate first 24 per hour cytotoxic not while toxic effect dose and to time connected aspect is increasing.

NaOCl all in their concentrations and time in the intervals -most good antimicrobial agent found however -most cytotoxic aspect has been observed.

Conclusion: Experiment in groups used NAC And Boric Acid antimicrobial dose borderline cytotoxicity in terms of other from groups more Good has been found.

Biography

- Tahir Ataözden/PHD/Oral Surgeon
- Tahir Ataözden was born in Edirne in 1957 and graduated from the English Dentistry Department of Marmara University in 1980.
- In 1983, he completed his doctoral program in oral surgery at Atatürk University Faculty of Dentistry, Department of Oral, Jaw, and Facial Surgery.
- In 1985, he opened his private clinic in Edirne. In 1987, he began working in Saudi Arabia, Riyadh, where he practiced until 1995.
- In 1995, he returned to Turkey and settled in Istanbul, continuing his clinical practice. He opened his private oral and dental health clinic in Istanbul in 2019.
- In the same year, he was appointed as an Associate Professor at Kafkas University, Faculty of Dentistry.
- He continues his academic and clinical studies in the fields of oral surgery and clinical practice. He speaks English and Arabic fluently.

Education Information:

- 1980 FACULTY OF DENTISTRY / DEPARTMENT OF CLINICAL SCIENCES / DEPARTMENT OF ORAL AND MAXILLOFACIAL SURGERY
- Thesis Title: Prioritization of the field width measurement method in the differential diagnosis of periapical radiolucent lesions from a clinical, radiological, and histopathological perspective (1983)
- PhD: ATATURK UNIVERSITY, July 7, 19831974 FACULTY OF DENTISTRY (Graduated: 1979)
- Bachelor's Degree: MARMARA UNIVERSITY, August 9, 1979
- Academic Position
- ASSISTANT PROFESSOR – KAFKAS UNIVERSITY / FACULTY OF DENTISTRY (2020)
- RESEARCH ASSISTANT – ATATURK UNIVERSITY / FACULTY OF DENTISTRY (1980–1985)

Projects:

- Evaluation of the Biocompatibility and Antibacterial Effectiveness of Alternative Materials Used in Endodontics and Regenerative Treatments in Dentistry – A Scientific Research Project Supported by Higher Education Institutions
- Principal Investigators: TAHIR ATAOZDEN, SEMANUR OZUDOGRU Date: 13/12/2021 (Ongoing) (NATIONAL) Administrative Duties
- 2020 – Vice Dean, KAFKAS UNIVERSITY / FACULTY OF DENTISTRY
- Memberships in Scientific Organizations
- Member, INTERNATIONAL CONGRESS OF ORAL IMPLANTOLOGISTS (1994–2011)

Infection of Neonates with *Staphylococcus aureus* and Methicillin-Resistant *Staphylococcus aureus* at Dormaa Presbyterian Hospital, Ghana

Jerome Adinkrah Obeng, William Gariba Akanwariwiak and **Augustina Angelina Sylverken**

Department of Theoretical and Applied Biology, Kwame Nkrumah University of Science and Technology, Ghana

Staphylococcus aureus is the second most common pathogen found in all cases of nosocomial infections globally. Methicillin-resistant *Staphylococcus aureus* (MRSA) is a common cause of infection among preterm and critically ill newborns in neonatal intensive care units (NICUs). Newborns are predominantly susceptible to *S. aureus* and MRSA colonization and infection due to their weaker immune systems. Treating infections in newborns associated with *S. aureus* and MRSA has proven challenging due to decreasing susceptibility to first-line antibiotics leaving clinicians with few treatment options. This cross-sectional study aimed to determine the prevalence of hospital-acquired MRSA among neonates at the Dormaa Presbyterian Hospital (DPH) in Ghana, the susceptibility profile to selected antibiotics and the associated risk factors.

Venous blood samples were taken from each of the neonatal participants and inoculated into Brain Heart Infusion broth. Standard biochemical tests and were performed and 36% (9/25) of the *S. aureus* isolates were identified as MRSA. The MRSA isolates were more susceptible to Ciprofloxacin, Levofloxacin, Gentamicin, Co-trimoxazole, Tetracycline and Cephalexin but resistant to Cloxacillin, Ampicillin, Roxithromycin and Lincomycin. The study found that gestational period (χ^2=3.865, p=0.049) and longer length of hospital stay (χ^2=10.911, p=0.012) were statistically significant for *S. aureus* and MRSA infection. Surveillance systems should be put in place by health authorities at the hospital targeting decolonization of MRSA strains at the hospital as well as monitoring antibiotic resistance that occurs through inappropriate access and use of antibiotics.

Biography

Jerome Adinkrah Obeng is a skilled researcher with a history of accomplishments in academia and clinical laboratory practice. He holds MPhil. Microbiology from Kwame Nkrumah University of Science and Technology, Kumasi, Post Graduate Diploma in Science Education, University of Cape Coast and BSc. Applied Biology from University for Development Studies (U.D.S) all of Ghana.

Jerome as he is lovingly known, has more than a decade working experience in clinical laboratory practice with interest in bacteriology and focus on antibiotic resistance and has worked at Laboratory Department of Dormaa Presbyterian Hospital since 2011. His passion for science education led him to classroom, serving as Teaching Assistant (T.A) at University of Energy and Natural Resource, Sunyani, Ghana in the year 2013/2014 academic year. He currently tutors high school biology students and gives part-time lectures in biology for the Centre for Distance Education (CoDE), University of Cape Coast, Ghana, alongside his clinical practice.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

Safety and Efficacy of Oral Edible Bird's Nest Supplementation: Anti-Inflammatory and Immunomodulatory Effects in Arabian Horses

Khalid Obaid AL-Khaldi[1], Khalid Hamed Al-Ruzaiqi[1], Mohammed Babatunde Sadiq[4], Abdul Salam Babji[2,3], Seng Joe Lim[2,3] and **Nurhusien Yimer[5,6]**

[1]Veterinary Services Administration, Mounted Police Headquarter, Sultanate of Oman
[2]Faculty of Science and Technology, Department of Food Sciences, Universiti Kebangsaan Malaysia, Malaysia
[3]Faculty of Science and Technology, Innovation Centre for Confectionery Technology (MANIS), Universiti Kebangsaan Malaysia, Malaysia
[4]Faculty of Veterinary Medicine, Department of Farm and Exotic Animal Medicine and Surgery, Universiti Putra Malaysia, Malaysia
[5]School of Medicine, Department of Veterinary Sciences, IMU University, Malaysia
[6]Faculty of Veterinary Medicine, Veterinary Reproduction Division, Airlangga University, Indonesia

Exercise-induced oxidative stress and inflammation can negatively impact the performance and recovery of athletic horses. This study aimed to evaluate the safety and potential anti-inflammatory and immunomodulatory effects of edible bird's nest (EBN) supplementation in Arabian race stallions subjected to a structured exercise regimen. Two experiments were conducted using 18 horses. In Experiment 1, six healthy stallions were assigned to control (n=2) and EBN-supplemented groups (n=4) to assess safety over 12 days of daily oral EBN administration (10 g/day), with monitoring of vital signs, hematological profiles, and hepatic and renal function markers. In Experiment 2, twelve stallions were divided into EBN (n=3), Premier E® (n=3), and control (n=6) groups and subjected to a 30-day exercise program comprising walking and cantering sessions. Blood samples were collected before and after exercise to analyze hematological indices and sialic acid (SA) levels as markers of inflammation. EBN supplementation was well-tolerated, with no adverse effects noted and stable clinical and laboratory values. Notably, creatine kinase, total bilirubin, and AST levels were significantly reduced in the EBN group, indicating a protective effect against hepatic and muscular stress. Additionally, post-exercise SA levels were significantly higher in the

EBN group compared to the Premier E® and control groups, reflecting a stronger anti-inflammatory response. Hematological parameters such as MCV, RDW, and platelet counts also improved, suggesting enhanced immunomodulatory activity.

These results demonstrate that EBN is a safe dietary supplement with beneficial effects on inflammation and immune modulation in performance horses. Further studies are recommended to elucidate its long-term and mechanistic impacts.

Biography

Dr. Khalid bin Obaid Al-Khaldi is the Assistant Director of the Veterinary Services Administration at the Mounted Police Headquarter of the Royal Oman Police and a senior equine veterinary specialist with over 20 years of experience in equine clinical practice. He holds a Ph.D. in Theriogenology from Universiti Putra Malaysia. Dr. Al-Khaldi has a strong research interest in integrative and evidence-based veterinary medicine, with pioneering work in natural supplementation, equine reproduction, and therapeutic innovation. He has authored multiple peer-reviewed scientific publications and holds several registered patents in veterinary applications. A member of both the national and international equestrian federations, Dr. Al-Khaldi actively contributes to the global veterinary community through scientific collaborations, conference participation, and ongoing research in Arabian horse health and performance optimization.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

HIV-1 Budding Control by Inducible Inhibition of ESCRT-III

Cécile Boscheron[1], Haiyan Wang[1], Benoit Gallet[1], Christine Moriscot[2], Mylène Pezet[3], Christine Chatellard[1], Jean-Philippe Kleman[1], Heinrich Göttlinger[4] and **Winfried Weissenhorn[1]**

[1]Institut de Biologie Structurale (IBS), University Grenoble Alpes, CEA, CNRS, France
[2]University Grenoble Alpes, CEA, CNRS, ISBG, France
[3]University Grenoble Alpes, INSERM, IAB, France
[4]University of Massachusetts Chan Medical School, USA

HIV-1 budding, like many other cellular processes, relies on the Endosomal Sorting Complex Required for Transport (ESCRT) machinery, which is essential for virus release via membrane fission. The core membrane remodeling complex, composed of ESCRT-III and VPS4, is highly conserved. However, understanding the native architecture of ESCRT-III at HIV-1 budding sites is limited due to spatial resolution constraints and its transient recruitment. To overcome this challenge, we developed a drug-inducible tool to transiently inhibit HIV-1 budding by extending the lifetime of ESCRT-III at budding sites. We engineered autocleavable CHMP2A, CHMP3, and CHMP4B fusion proteins with the hepatitis C virus NS3 protease, designed to be converted into VPS4-deficient variants upon NS3 inhibitor treatment. We characterized these CHMP-NS3 fusion proteins through immunoblotting, fluorescence-based localization studies, transmission electron microscopy, and live-cell imaging, both in the presence and absence of the protease inhibitor Glecaprevir. Our results demonstrate that CHMP-NS3 fusion proteins accumulate rapidly and remain stable upon drug administration. CHMP2A-NS3 and CHMP4B-NS3 significantly inhibited virus-like particle (VLP) release, while CHMP3-NS3 alone had no effect but synergized with CHMP2A-NS3. Localization analyses revealed the redistribution of CHMP-NS3 fusion proteins to the plasma membrane, endosomes, and HIV-1 Gag VLP budding sites. Electron and video microscopy further revealed a drug-dependent accumulation of CHMP2A-NS3 and CHMP4B-NS3, leading to delayed HIV-1 Gag-VLP release.

These findings provide novel insights into the functional consequences of ESCRT-III inhibition during HIV-1 budding. Our approach enables precise temporal control over CHMP2A-NS3, CHMP3-NS3, and CHMP4B-NS3 expression, facilitating targeted inhibition of distinct HIV-1 budding stages. Initially developed for HEK293 and HeLa cells, this assay can be adapted to study a wide range of ESCRT-III-driven cellular processes across different cell types.

Biography

Dr. Cécile Boscheron is a researcher at the French Alternative Energies and Atomic Energy Commission (CEA) within the Institute of Structural Biology (IBS) in Grenoble, France, where she has been part of the Enveloped Virus Entry and Budding Group since 2018. She began her career as an *élève* at École Normale Supérieure de Lyon, earning a PhD in Molecular and Cellular Biology from ENSL – Université Claude Bernard Lyon in 1998, followed by her Habilitation to Direct Research (HDR) in 2008. Dr. Boscheron has held research positions at INSERM, CEA, and Université Grenoble Alpes, developing expertise in yeast genetics, live-cell imaging, super-resolution and confocal microscopy, CRISPR/Cas9 gene editing, and viral infections in cell culture. She has led multiple research projects, securing funding from ANRS, FRM, and ANR. Her research focuses on virology, cellular, and structural biology, particularly the structural dynamics of HIV-1 cellular complexes and ESCRT-III polymer remodeling during viral budding.

ADV. IMMUNOLOGY 2025 & FUTURE VIROLOGY 2025

ADV. IMMUNOLOGY 2025
FUTURE VIROLOGY 2025

DAY 02

JOINT EVENT

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & VIROLOGY

BERLIN, GERMANY

SEPTEMBER 25-26, 2025

SPEAKER TALKS

MIPC, the Universal Antiviral Cells

Fawzy Abdelatty

Heidelberger Center for Cellular Therapy, Germany

MIPC are therapeutic Methylation Induced Pluripotent Cells which are prepared from peripheral blood samples. For patients, these samples can be obtained from autologous, heterologous or xenographic sources. These cells are very potent in infection prevention and control. They have the potential to eliminate infections (viral or bacterial) and antibiotics resistant hospital infections within hours. The preparation and handling of these cells is very simple and has no negative side effects. The cells do not only remove the infection but can also regenerate the organs damaged due to this infection effectively. MIPC will make it possible to start treating a pandemic infection, even before characterizing the microbe. They can even be used as a prophylactic measure in such cases. Examples for treating different pathogens are presented.

Biography

- 1980 B.Sc. Biology Department, Faculty of Science, Cairo University
- 1987 M.Sc. Hormone Research Department, Weizmann Institute of Science, Rehovot, Israel
- 1987 Ph.D. Fellowship, EMBL, Heidelberg
- 1992 Dr.Sc.hum., Institute of Human Genetics, Faculty of Medicine, University of Heidelberg, Germany
- Postdocs: Max Plank Institute for Medical Research, Surgery Clinic of Heidelberg University, Immunology and Applied Virology Departments of the German Cancer Research Center in Heidelberg
- 1998 Registering a patent for Gene Detection
- 1999 Establishing R&D Laboratory to Produce Molecularly Designed Pharmaceuticals for the Pharma Holding Company, Egypt
- 2007 Establishing R&D Laboratory for Vaccine Development and Stem Cells at the German University in Cairo
- 2019 Registering a Patent for Novel Reprogrammed, Therapeutic Pluripotent Cells
- Currently, CSO of the Heidelberger Centre for Cellular Therapy, Heidelberg, Germany

Flipons and the Genetics of Innate Immunity

Alan Herbert

InsideOutBio, USA

An evolutionary challenge to metazoans arises from the spread of endogenous retroelements (ERE) throughout their genomes. In humans, EREs contribute to over 50% of the genome. EREs that insert into genes can cause splicing errors, resulting in reading frame changes and premature stop codons. ERE can also be inserted close to each other in the opposite orientation. When transcribed, these inverted repeats form double-stranded RNAs to activate immune sensors like MDA5. Both these outcomes can be countered by the Adenosine Deaminase activated by RNA (ADAR) protein, which recognizes non-canonical nucleic acid structures formed by the EREs through Zα and Zβ domains. The sequences that flip their conformation are called flipons. Flipons that form the left-handed Z-DNA and Z-RNA (collectively called ZNAs) are bound by Zα, and those that fold into G-quadruplexes (GQ) bind Zα and Zβ domains. Zα loss-of-function ADAR variants in humans and mice produce Aicardi-Goutières syndrome type 6, an interferonopathy. Conversely, tumors use ADAR to silence immune responses. The recognition of GQ by Zβ in the nucleus can lead to the recoding of RNA to produce variant proteins. ADAR also regulates the induction of programmed cell death when cells become dysfunctional or are infected by viruses. The talk will focus on recent experimental data that reveals the mechanisms involved and how ERE sequences have been exapted to protect the host against such threats.

Biography

Alan Herbert trained in New Zealand. His work at MIT, Boston University, Merck, and InsideOuBio has contributed to our understanding of the genetic variants that cause human disease and of the various biological roles played by flipons.

Joint Event
ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY
&
FUTURE VIROLOGY
September 25-26, 2025 | Berlin, Germany

Extracellular Vesicles as Mediators of Inflammation in an *in vitro* Model of Psoriasis

Giorgio Mangino[1], Marco Iuliano[1], Lorena Capriotti[1], Roberto Lande[2], Loredana Frasca[2], Nicoletta Bernardini[1,3], Paolo Rosa[1], Elio Pellegrini[1], Nevena Skroza[1,3], Concetta Potenza[1,3] and **Giovanna Romeo[1]**

[1]Department of Medico-Surgical Sciences and Biotechnologies, Sapienza University of Rome, Italy
[2]Pharmacological Research and Experimental Therapy Section, National Center for Drug Research and Evaluation, Istituto Superiore di Sanità, Italy
[3]Dermatology Unit "Daniele Innocenzi", Fiorini Hospital, Italy

Background: Psoriasis is a chronic inflammatory skin disease caused by the excessive secretion of inflammatory cytokines. Deregulation of the interleukin-17/-23 axis allows the activation of Th17 lymphocytes and the reprogramming of keratinocytes proliferative response, thereby inducing the secretion of cyto-/chemokines and antimicrobial peptides.

Psoriasis-associated inflammation also affect systemic functions associated with extracutaneous manifestations. Beside cell-to-cell contacts and release of cytokines, hormones and second messengers, cells communicate through the release of extracellular vesicles (EVs) containing DNA, RNA, microRNAs and proteins. It has been reported the alteration of EVs trafficking in several diseases, but there is already scarce evidence of the involvement of EVs in the pathogenesis of psoriasis.

Objective: To characterize the release, the cargo content and the capacity to transfer bioactive molecules of EVs produced by keratinocytes following the treatment of human keratinocytes with psoriasis-associated cytokines (*i.e.* IL-17A, IFN-γ, TNF-α, IL-22 or IL-23).

Methods: A combined approach of standard ultracentrifugation, RNA isolation and Real Time RT-PCR techniques was used to characterize EVs cargo. Nanoparticle Track Analysis was used to enumerate EVs whereas Confocal Microscopy was used to evaluate cell-to-cell extracellular vesicles transfer and Netosis.

Results: We report that the treatment of human keratinocytes with IL-17A significantly modify EVs release and cargo content. Vesicles from IL-17A-treated cells display a specific pattern of mRNA which is abrogated by anti-IL-17A neutralization. This pattern is also induced by other psoriasis related cytokines as TNFα and IL-23. EVs are taken up by acceptor cells irrespective of their content but only those derived from cells treated with inflammatory cytokines enable receiving cells to express psoriasis-associated mRNA. Finally, EVs collected by psoriasis-associated cytokines trigger Netosis *in vitro*.

Conclusion: The obtained results imply a role of extracellular vesicles in amplifying the pro-inflammatory cascade induced in keratinocyte by psoriasis-associated cytokines.

Biography

Giorgio Mangino obtained his degree in Biological Sciences in 1995 and his PhD in Immunological Sciences in 2006. Early in his career, his main interests were to elucidate type I and type II Interferon signal transduction pathways and then, the role of CD28 costimulatory molecule in T lymphocyte activation. Later on, he characterized the role of HIV-1 Nef protein in the induction of inflammatory response in macrophages through the interaction with TRAFs adapters and activation of NF-κB signalling. Since 2015 his main interest is focused on the role of extracellular vesicles in shaping the microenvironment in HPV-induced tumorigenesis and in inflammatory diseases as psoriasis through the characterization of the vescicles cargo content and the use of vesicles-associated mediators as putative diagnostic markers.

Oral Vaccine for Type 1 Diabetes

Mohamed I. Husseiny Elsayed, Jacob Cobb, Jeffrey Rawson, Nelson Gonzalez and **Fouad Kandeel**

Department of Translational Research & Cellular Therapeutics, Arthur Riggs Diabetes & Metabolism Research Institute, Beckman Research Institute, City of Hope National Medical Center, USA

Type 1 diabetes (T1D) is a complex disease characterized by loss of immune tolerance to self-autoantigens and destruction of insulin-producing beta-cells. No single therapy confronts the several contributors to the disease. We developed an oral Salmonella-based vaccine that provides autoantigen proinsulin (PI) in combination with TGFβ, IL10, and anti-CD3. The vaccine prevented and reversed autoimmune diabetes in non-obese diabetic (NOD) mice.

The vaccine-mediated beneficial effects were associated with increased numbers of antigen- specific Foxp3+ Tregs, Tr1-cells, and tolerogenic dendritic-cells (tol-DCs) in the spleens and lymphatic organs of treated mice. Despite this, the immune response to Salmonella infection was not altered. Furthermore, the vaccine and GAST-17 (gastrin analogue) combination were administered to autoimmune diabetic mice and outcomes compared with animals that received the vaccine or GAST-17 alone. Administration of the vaccine with GAST-17 reversed disease in 80% of diabetic mice compared to 63% of vaccine alone and 5% of GAST-17 alone treated animals. This was associated with increased antigen-specific regulatory T-cells, decreased islet-infiltrating lymphocytes, and increased beta-cell mass.

Increased serum levels of the tolerogenic cytokines (IL10, IL2, and IL13) and chemokine ligand 2 (CCL2) and decreased levels of inflammatory cytokines (IFNγ, GM-CSF, IL6, IL12, and TNFα) and chemokines (CXCL1, CXCL2, and CXCL5). Overall, the data suggest that the Salmonella-based vaccine modulates the immune response, reduces inflammation, and promotes tolerance specifically to an antigen involved in autoimmune diabetes. A novel combination treatment of an oral diabetes vaccine and GAST-17 was superior in the reversal

of established T1D in mice. This combination strategy could be worthwhile examining in individuals with autoimmune diabetes.

Biography

Dr. Mohamed I. Husseiny Elsayed completed his Bachelor of Pharmacy in 1992 and Master's in Medical Microbiology 1998 from Zagazig University, Egypt. Following that, he completed his Ph.D. in 2004 from Institute for Clinical Microbiology, Immunology and Hygiene, Friedrich-Alexander University, Erlangen-Nürnberg, Germany. During his Ph.D., he established a novel system for gene therapy. He developed an oral vaccine against *Listeriosis* using genetically engineered *Salmonella*. He employed the same system to develop a vaccine for cancer during his postdoctoral fellowship at the Children's Hospital of Los Angeles.

Currently, He is an Associate Research Professor at the Department of Translational Research and Cellular Therapeutics, AR-DMRI, BRI, City of Hope National Medical Center. He developed an oral *Salmonella*-based vaccine to treat animal models of autoimmune T1D. He worked with this vaccination strategy for over 24 years. Throughout his career, his goal remains to employ vaccine strategies to ameliorate and prevent disease especially autoimmune disease such as T1D.

Etiopathogenesis of Nasal Polyps Elucidated from Histopathological Examinations of Nasal Polyps of Different Size

Per Leganger Larsen

Copenhagen University and Capital Region Hospitals, Denmark

Epithelium, glands, stroma, stromal cells and possible cellular interactions in relation to polyp formation has been described from histopathological studies of large ”fully developed” polyps. Although immunohistochemical techniques have advanced and improved and our understanding of cellular interactions have been further elucidated, reaching a definite conclusion regarding the etiopathogenesis of nasal polyps remains challenging. In this presentation we aim to synthesize findings from our earlier studies concerning suprastructure, epithelial types, goblet cells, glands, sites of origination and prevalence with the hope of further elucidating the pathogenesis of nasal polyps. Epithelium and goblet cells were studied in cross sections and Whole Mount preparations from both anterior and posterior polyps, as well as from superior- and inferior regions of fully developed polyps. Variability in goblet cell density and epithelial type (pseudostratified, cylindric, transitional, squamous transitional, stratified squamous) was observed within the same polyp. Both gradual and abrupt transitions between epithelial types were noted. These findings are thought to result from dynamic changes influenced by air current, contact with adjacent polyps, infection, polyp growth, aging, and other unidentified factors. Characteristic long tubulous mucous glands were observed in Whole Mount preparations from fully developed nasal polyps. These glands displayed structural and architectural differences compared to the glands of normal nasal mucosa (Fig.1a)

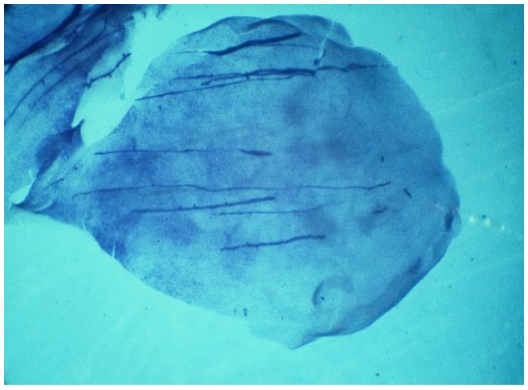
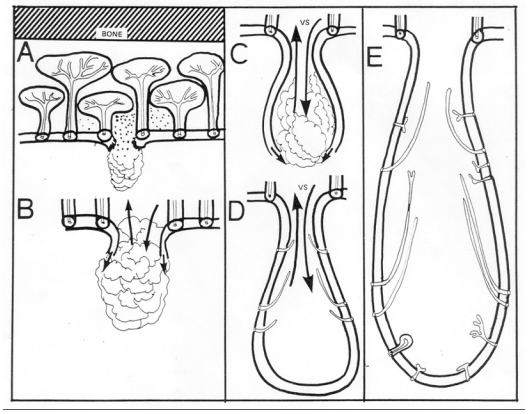


Fig. 1a) Long tubulous mucous glands in a Pas-Alcian Blue Whole Mount specimen.



1b) Theories of Epithelial Rupture- and Glandular New Formation.

Glandular density was significantly lower in polyps (0,1-0,5 / mm^2), compared to normal nasal mucosa (7/ mm^2). Moreover, the glands lacked myoepithelial basket cells, in contrast to the seromucous glands of the nasal mucosa. This suggests that polyps represent de novo formations in the nasal mucosa.

Origination and frequency:

Three studies were conducted to examine the origin and prevalence of nasal polyps:

Study 1: Anterior rhinoscopy using a nasal speculum was performed on 300 autopsies. If polyps were detected, the entire naso-ethmoidal block was excised and examined. Polyps primarily originated in relation to the sinus ostia, and the frequency was consistent with the literature. The origin was mainly the sinus outlets and the frequency as found in the literature (2 %, 6 out of 300).

Study 2: The naso-ethmoidal block was removed from 19 autopsies without prior rhinoscopic examination. Again, polyps were mainly found in the sinus outlet region, and the prevalence was unexpectedly high (26%).

Studies 3 and 4: Systematic endoscopic examination and sinus surgery were performed in 150 autopsies, revealing a prevalence of 32%. Most polyps were < 5 mm. in size and originated from the mucosa in the sinus outlet.

The small polyps and corresponding mucosa were fixed in 4% formaldehyde, embedded in paraffin, and cross sections of 5my were made for further analysis. Epithelia and eosinophil presence were primarily assessed using Hematoxylin-Eosin and Pas-Alcian Blue staining. Goblet cells were studied using Pas-Alcian Blue in Whole Mount preparations. Glands with a Pas-Alcian Blue in Whole Mounts. Glands were also assessed using immunohistochemical staining for myoepithelial basket cells and growth factors. Mast cells were stained with Naphtol-AS-D-Chloracetate. Biofilm with DAPI - 4´,6-diamidino-2-phenylindole (DNA-staining).

Over the past 160 years, several pathogenetic theories have been proposed for nasal polyp development.

The theories are based on oedema (by Billroth), increased tubulo-alveolar gland proliferation, presence of cystic glands and mucous filled cysts, and mucous glands proliferation.

We have earlier ventilated that nasal polyps could be new formations in the existing nasal mucosa and described an “Epithelial Rupture Theory” and a “Glandular New Formation Theory” for the pathogenesis of nasal polyps. Starting with an epithelial defect (Fig.1b).

We have shown that oolyp formation starting with epithelial rupture and subsequent epithelialization can take place in the infected rat middle ear.

Although epithelial defects were not directly observed, the density of eosinophils was significantly higher at the origin site compared to the polyp stroma. Mast cells showed a similar distribution.

We hypothesize that an etiologic trigger may be located at the site of origin. We have previously demonstrated eosinophil recruitment to nasal polyps *via* vascular cell adhesion molecules potentially in response to superantigens. Notably, local IgE synthesis against staphylococcus aureus enterotoxins has been identified in polyp tissue, suggesting a superantigen-driven process. A bacterial biofilm was also identified at the site of origin, primarily consisting of Staphylococcus species.

Although eosinophils were present in most nasal polyps and it has been ventilated that Major Basic Proteins from eosinophils can lead to damage of the epithelium in the human nasal mucosa and in nasal polyps, no epithelial defects were identified in our specimens.

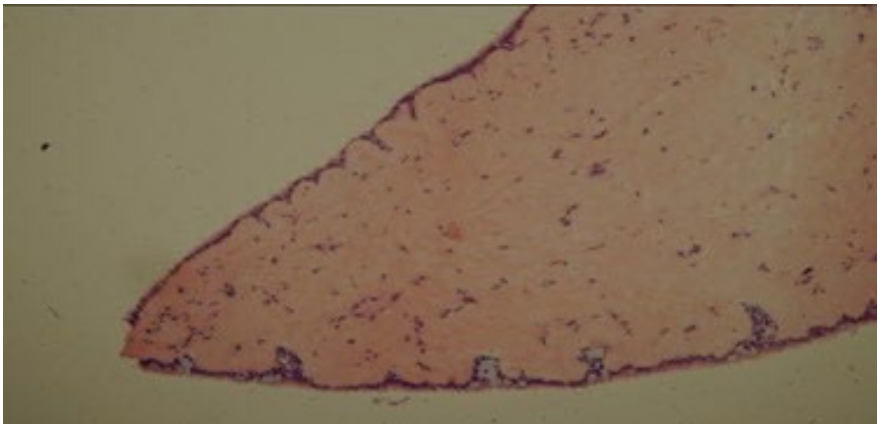
We have found that the ciliary epithelium in the human nose can develop and regenerate very fast but we have also found that if the polyp epithelium was well preserved, scanning- and transmission electron microscopy and light microscopy showed no epithelial defects, when using a cutting forceps instead of a snare when removing a polyp and a very gentle method of fixation and dehydration by adding oxygenated fluorocarbon to a glutaralde-

hyde fixative, so that enough oxygen was made accessible for cellular respiration as well as for the oxygen-consuming chemical reaction of glutaraldehyde within the polyp tissue.

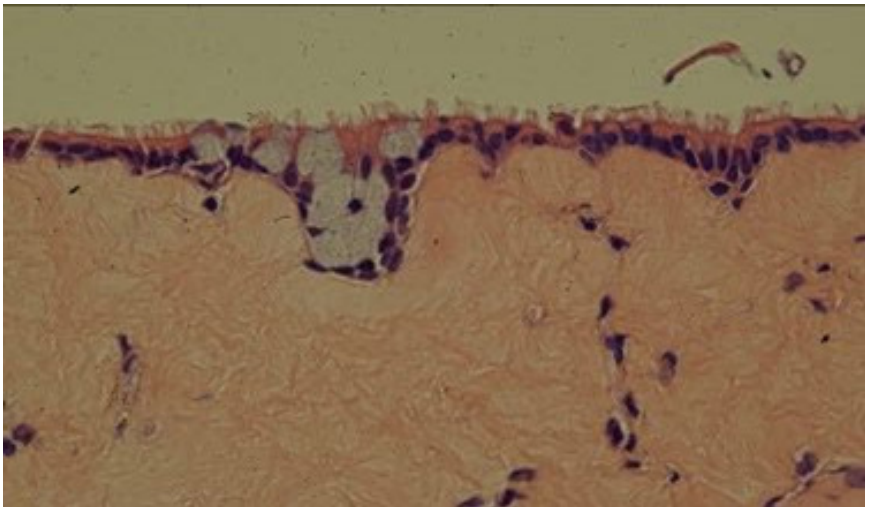
It seems like we now, after several years behind the microscope studying the structure of fully developed nasal polyps and finally small polyps (< 5 mm.), identified by endoscopy in autopsies, are able to further elucidate the initial stages of polyp formation;

The small polyps were covered by a very low epithelium (Fig. 2a), and different epithelial were observed on opposing sides of the same polyp as a sign of epithelialization following a defect (Fig. 2a). Initial stages of gland formation and early gland formation was evident as small epithelial “buds” and secretory cells within the surface epithelium of the distal polyp region (Fig. 2b).

Fig. 2a; Polyp covered by low epithelium. Epithelial types differ on each side.



2b; Small epithelial “Bud” and small initial gland with secretory cells.



Ethiopathogenesis of nasal polyps will be discussed from these results which seems to show that:

Nasal Polyps are new formations in the existing nasal mucosa starting with a primary epithelial defect, epithelialization and glandular formation which supports the “Epithelial Rupture and Glandular new formation Theory”.

The prevalence of nasal polyps is higher than traditionally assumed (up to 32% in postmortem examinations)

The sinus ostia represents the most common site of origin.

Inflammatory cells and a bacterial biofilm could be shown at the site of origination.

Biography

- Sequelae after secretory otitis media in humans.
- Sequelae after experimental tuba occlusion in rats and cats.
- Development of Ciliary Cells in the Human Nose.
- Origin of nasal Polyps.
- Mucous Elements / Goblet Cells in *upper and lower airways of* Nude (Athymic) and normal Rats.
- Changes of Nasal Mucosa after implantation into the middle Ear.
- Humoral Immunity in the Nasopharynx of Children.
- Introduced Endoscopic Sinus Surgery in Denmark 1989.

Precision Mapping of Influenza A Virus RNA Dynamics and Antiviral Responses using an Advanced CRISPR- Cas12 Platform

Tran Anh Tu[1,2] and **Jim-Tong Horng**[1,2,3,4]

[1]Research Center for Emerging Viral Infections, College of Medicine, Chang Gung University, Taiwan
[2]Department of Biochemistry and Molecular Biology and Graduate Institute of Biomedical Sciences, College of Medicine, Chang Gung University, Taiwan
[3]Research Center for Food and Cosmetic Safety, College of Human Ecology, Chang Gung University of Science and Technology, Taiwan
[4]Molecular Infectious Disease Research Center, Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Taiwan

Influenza A virus (IAV) remains a significant global health threat, necessitating advanced tools to decode viral behavior and inform therapeutic strategies. Here, we introduce a groundbreaking CRISPR-Cas12-based platform that precisely distinguishes and quantifies viral RNA species—vRNA, cRNA, and mRNA—across the infection cycle. This platform achieves unparalleled sensitivity, detecting as few as 100 RNA copies, and delivers a tenfold improvement in speed and accuracy compared to traditional methods. Focusing on viral segments 5 (NP) and 6 (NA), our study revealed novel early infection dynamics, including an unexpected vRNA decline within 0–40 minutes post-infection, followed by recovery, challenging existing replication models. We validated the platform across MDCK and A549 cells, a mouse model, and clinical nasopharyngeal samples, demonstrating its robustness and translational relevance. Its broad detection range (10^2–10^{10} copies) enables comprehensive monitoring of RNA kinetics from early to late infection stages. In addition to RNA dynamics, we evaluated the effects of antiviral drugs—Baloxavir, Favipiravir, Molnupiravir, and Ribavirin— highlighting their distinct impacts on RNA synthesis at various stages of infection. These findings offer critical insights into drug mechanisms and their influence on viral replication. Looking ahead, we aim to refine and validate this platform through high-resolution time-course analyses of viral RNA dynamics, focusing on the early infection stage where we observed the novel vRNA pattern. These studies will integrate live-cell imaging

to visualize real-time RNA changes and RNA stability assays to elucidate the mechanisms behind these dynamics. Furthermore, we will investigate ribonucleoprotein (RNP) complex dynamics and their role in RNA stability and function. Building on these findings, we plan to optimize the platform for zoonotic and avian influenza strains, enabling strain-specific RNA dynamics detection and contributing to understanding virulence and transmission. Expanding to other clinically significant RNA viruses, this adaptable system holds the potential to transform viral diagnostics, deepen our understanding of viral biology, and accelerate antiviral drug development.

Biography

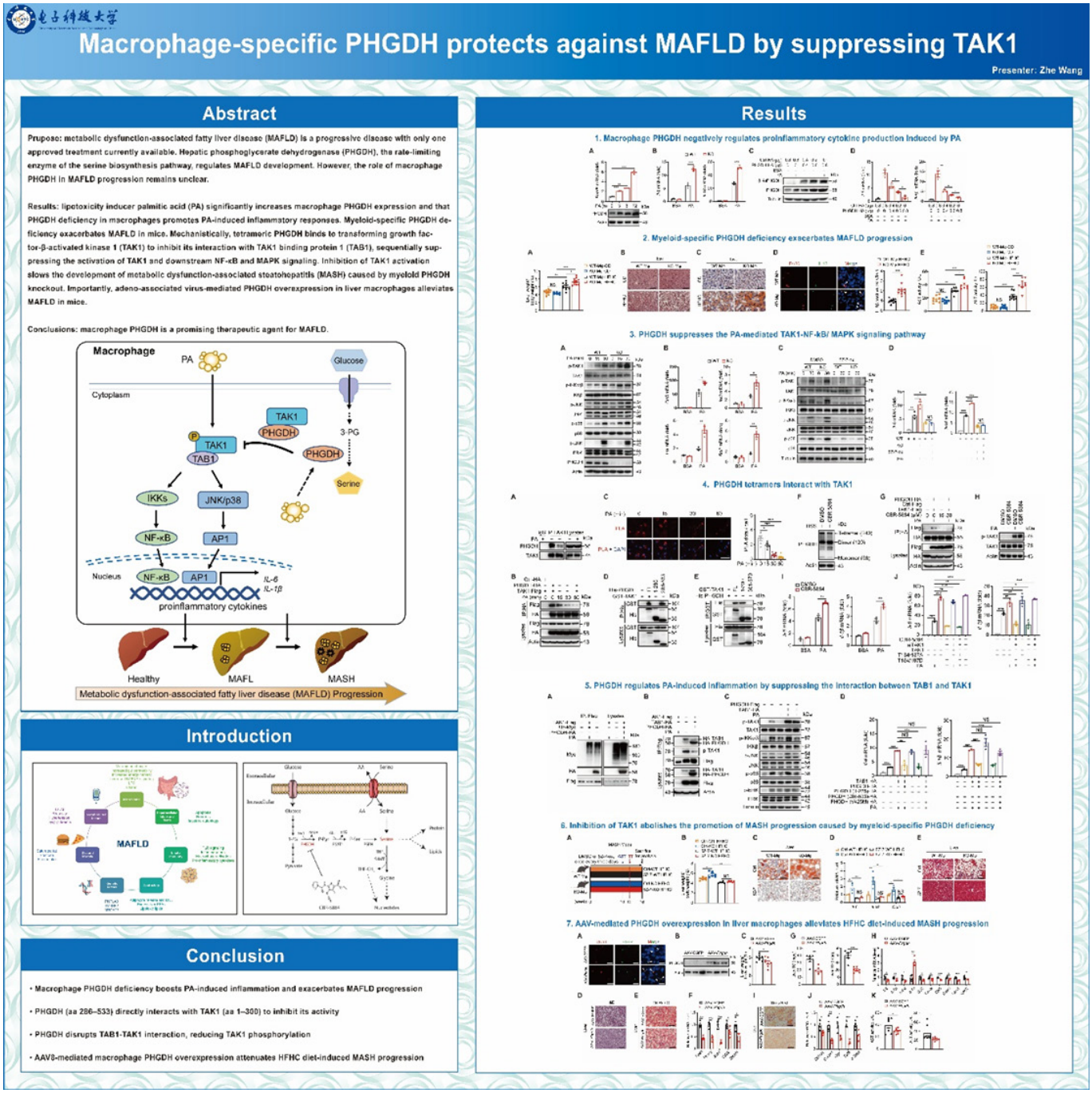
Anh-Tu Tran is a PhD candidate in Biochemistry and Molecular Biology at Chang Gung University, Taiwan. His research focuses on influenza virus RNA dynamics, antiviral drug mechanisms, and CRISPR-based diagnostics. He has developed CRISPR/Cas12 platforms for rapid viral RNA detection and studied the effects of polymerase inhibitors on RNA synthesis. In master degree, he conducted research on enzyme kinetics and genetic variations in autoimmune diseases. He has presented at international conferences and has published in peer-reviewed journals. Anh-Tu is a member of the Taiwan Society of Virology and Vaccinology.

Macrophage-Specific PHGDH Protects against MAFLD by Suppressing TAK1

Zhe Wang[1], Penghui Hu[2] and **Xiao Shan[1]**

[1]Department of Health Management Center and Institute of Health Management, Sichuan Provincial People's Hospital, University of Electronic Science and Technology of China, China
[2]Department of Critical Care Medicine, Tianjin Medical University General Hospital, China

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a progressive disease with only one approved treatment currently available. Hepatic phosphoglycerate dehydrogenase (PHGDH), the rate-limiting enzyme of the serine biosynthesis pathway, regulates MAFLD development. However, the role of macrophage PHGDH in MAFLD progression remains unclear. Here, we demonstrate that the lipotoxicity inducer palmitic acid (PA) significantly increases macrophage PHGDH expression and that PHGDH deficiency in macrophages promotes PA-induced inflammatory responses. Myeloid-specific PHGDH deficiency exacerbates MAFLD in mice. Mechanistically, tetrameric PHGDH binds to transforming growth factor-β-activated kinase 1 (TAK1) to inhibit its interaction with TAK1 binding protein 1 (TAB1), sequentially suppressing the activation of TAK1 and downstream NF-κB and MAPK signaling. Inhibition of TAK1 activation slows the development of metabolic dysfunction-associated steatohepatitis (MASH) caused by myeloid PHGDH knockout. Importantly, adeno-associated virus-mediated PHGDH overexpression in liver macrophages alleviates MAFLD in mice. Collectively, these results identify macrophage PHGDH as a promising therapeutic agent for MAFLD.



Biography

Zhe Wang received the B.S. degree in clinical laboratory technology from Jiangsu University, Zhenjiang, China, in 2017 and the M.S. degree in biochemistry and molecular biology from Tianjin Medical University, Tianjin, China, in 2020. She is currently working toward the Ph.D. degree in biomedical engineering with the department of medicine, University of Electronic Science and Technology of China, Chengdu, China. Her research interests include macrophage, innate immunity and inflammation.

Distinct Tumor Immune Responses to Nanosecond Pulsed Electric Fields (nsPEFs) Determine Immunity

Stephen J Beebe, Anthony Nanajian, Brittney Ruedlinger and **Siqi Guo**

Frank Reidy Research Center for Bioelectrics; Old Dominion University Norfolk Va, USA

Nanosecond pulsed electric field fields (nsPEFs) is a pulsed power technology that stores and releases high-powered, non-thermal electric pulses in nanosecond durations that induce *in situ* vaccination (ISV) after ablation of orthotopic rat N1-S1 liver (75-80%) and mouse 4T1-luc breast tumors (80-95%); however, not in the mouse melanoma (10-20%) models These studies are designed to determine immune mechanisms for nsPEFs in cancer models that do or do not readily induce immunity with ISV.

In 4T1-luc breast cancer, nsPEFs selectively targeted apoptosis-induction in activated T-regulatory cells (Tregs) and tumor-associated macrophages (TAMs) and eliminating myeloid-derived suppressor cells (MDSC), showing relief of immunosuppression in local and systemic environments. There was a reduction in functional Treg suppression capacity, likely explained by decreases in activation markers (4-1BB and TGFβ) and a shift of Treg phenotype from predominantly activated ($CD44^+CD62L^-$) to naïve ($CD44^-CD62L^+$). This stronger nsPEF apoptotic bias for activated Tregs spared effector $CD4^+$ and $CD8^+$ T cells leading to a concomitant rise in effector $CD4^+$ T cells and a 2.7-fold increase in the ratio of resident memory $CD8^+$ $CD103^+$ T-cells to $CD4^+$ Tregs. These findings show nsPEFs effectively switch the TME and secondary lymphatics from immunosuppressive to immunoactive allowing cytotoxic T cell function and immune memory formation to eliminate cancer cells and account for nsPEF-induced ISV.

Studies with nsPEF conditions that induce ISV in rat liver and mouse breast cancer models do not readily induce ISV in mouse melanoma. In the TME, there were increases in DCs expressing costimulatory molecules indicating the first step in an immune response, but memory T-cell numbers were low and likely anergic. Unlike the 4T1-luc tumors, Tregs, MDSC, and TAMs were not significantly increase on post treatment days 3 and 7. Overall,

these responses allude to a narrow potential for immunity in the mouse melanoma model due primarily to immunosuppression.

Biography

Prof. Beebe received a BS in Zoology, Ohio Univ., Athens (1970); toured the Northern United States on his motorcycle, before becoming a Peace Corps Volunteer (British West Indies, 1973-1975). He received a Ph.D. in Medical Sciences (Pharmacology), Medical College of Ohio (1982), now Toledo University School of Medicine. He was Post-Doctoral Fellow / Associate at Howard Hugh Medical Institute, Dept Molecular Physiology and Biophysics, Vanderbilt (1982-1986); a Fulbright and Marshal Scholar, Oslo (1986-1988); Assistant Professor (Gynecology), and Pediatrics / Physiological Sciences), Eastern Virginia Medical School (1989-2007). He is now a Professor in the Frank Reidy Research Center for Bioelectrics, Old Dominion University (ODU), Norfolk, VA. He served as IACUC chair for 13 years (2010-2023). According to a Stanford study, he was included in the top 2% of most cited scientists in his field worldwide over the last 4 years. He is a member of Sigma Xi.

The CTCF Anatomy of Human Chromosomes: Implications for Enhancer-Promoter Communication

Colin Logie

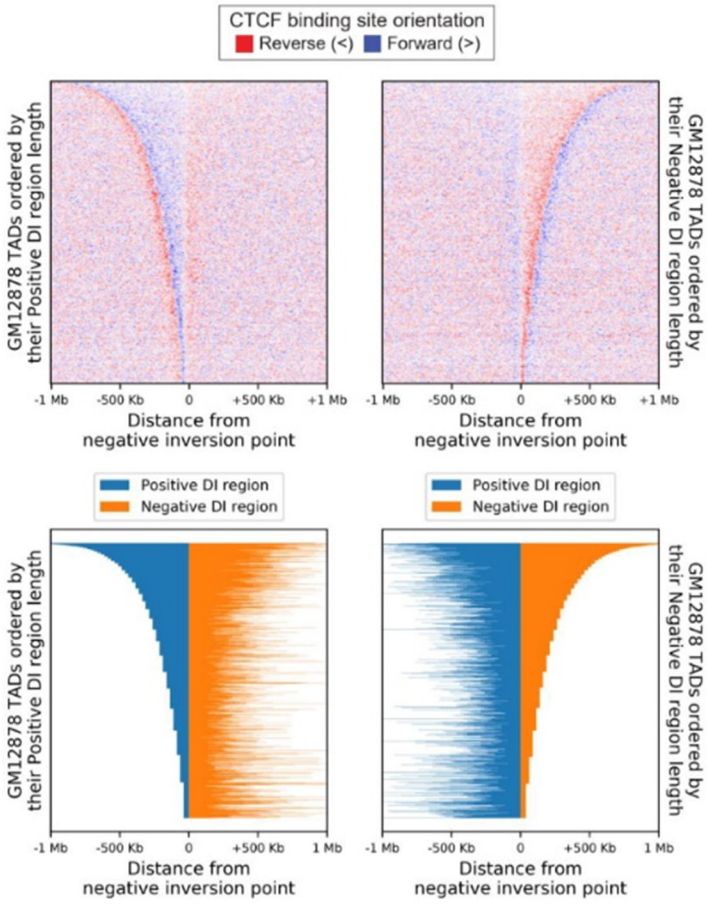
Radboud Institute for Molecular Life Science (M850), The Netherlands

We found that the human genome is divided in alternating domains showing interaction biases to the right or to the left [1].

These paired domains form topologically associated chromosome domains. They are hard-coded by genomic CTCF sites that are asymmetric 19 bp sequences (see figure). CTCF sites can form stable chromatin loop anchors via a process of chromatin loop extrusion driven by SMC chromatin motors.

Gene regulation by enhancers is largely constrained by chromatin loop dimensions. We will discuss the implications of this chromosome folding pathway for enhancer-promoter communication in the context of glucocorticoid signaling during innate immune cell development.

[1] Nanni et al. Spatial patterns of CTCF sites define the anatomy of TADs and their boundaries (2020) Genome biology 21, 1-25.



Biography

Colin Logie was born and raised in Brussels, Belgium. After a genetics and molecular biology degree at Glasgow University, he obtained a PhD for developing the first prototype of a tamoxifen inducible site specific recombinase at EMBL in Heidelberg, opening the door to many sophisticated reverse genetics approaches in mice. After a postdoctoral stint at the molecular medicine institute of UMASS he joined the Faculty of Science Radboud University in Nijmegen the Netherlands where he still researches chromatin with a focus on glucocorticoid signaling and myeloid cell development.

Epigenetic Regulation and Nuclear Actin Dynamics in Early and Differentiated T Helper Cells

Moran Titelbaum, Boris Brant, Yiftah Barsheshet and **Orly Avni**

The Azrieli Faculty of Medicine, Bar-Ilan University, Israel

Naïve CD4⁺ T-helper (Th) cells differentiate into specialized subsets that orchestrate immune responses through precise transcriptional programs. While the Polycomb group (PcG) protein Ezh2 is classically associated with gene repression, our previous studies revealed an unexpected role for Ezh2 as a transcriptional activator in differentiated Th cells. Moreover, Ezh2 is implicated in cytoskeletal remodelling, suggesting possible crosstalk between epigenetic regulators and the actin machinery.

In early differentiating Th cells, we found that the methyltransferase-dependent activity of Ezh2 regulates the formation of nuclear actin filaments. These filaments colocalize with known actin regulators, align along the T cell receptor (TCR) axis, and interweave with chromatin, forming a dynamic nuclear scaffold. This network is essential for chromatin spreading and nuclear expansion, linking cytoskeletal architecture to gene regulation during the early phases of Th cell differentiation.

Building upon these findings, we further explored the potential involvement of additional nuclear cytoskeletal components in gene regulation. Our imaging-based approaches uncovered evidence for the presence of additional filamentous networks in the nucleus of differentiated Th cells, which exhibit spatial and functional coordination with nuclear actin structures. These observations raise the possibility that the nuclear cytoskeleton not only supports nuclear integrity but also facilitates transcriptional activation, possibly by mediating spatial organization and transport within the nucleus.

I will present new data highlighting the emerging role of the nuclear cytoskeleton as an active participant in gene regulatory processes, particularly in the context of T cell activation. These findings open new avenues for understanding how structural elements within the nucleus contribute to the orchestration of immune responses.

Biography

Moran Titelbaum is the Molecular Immunology Lab Manager and a Lecturer for advanced degree students at the Faculty of Medicine in the Galilee, Bar-Ilan University. Her academic journey began with a deep fascination for how immune cells communicate and regulate gene expression. During her doctoral studies, she explored the intersection between epigenetic regulation and cytoskeletal dynamics in T-helper cell activation, uncovering novel aspects of nuclear organization.

In recent years, she has also initiated collaborations with researchers and medical doctors to investigate how immune and molecular mechanisms contribute to reproductive system function and heart diseases. Today, she continues to pursue these questions while mentoring students and teaching courses in immunology and microscopy. She is passionate about making complex cellular processes accessible and engaging for the next generation of scientists. Sharing science—whether in the lab, the classroom, or at conferences—is one of the most rewarding aspects of her work.

Impacts During Viral Infections on Immune Mechanisms and Effects of Nutraceuticals and Pharmacological

Anju Kaushal

New Zealand Organization for Quality, New Zealand

The pathological conditions and COVID-19 severity eventually prolonged the hospital stay for seriously ill people escalated the mortalities. A large population has suffered unprecedently from mental stress and isolation during COVID-19 pandemic. The overwhelming number of cases in hospitals have shown people suffered from “PACS syndrome” linked to brain fog and cognitive impairments was the most concerning feature. Prolonged stay in hospitals, COVID-19 fatigue syndrome and microbial dysbiosis are mainly speculated to worsen the situation. Microbiome research has already made its place delivering the beneficial effects of bioactive compounds like, probiotics, prebiotics, and postbiotics harboring the natural capacity to moderate the metabolic and immune functions to sustain the homeostasis. The exacerbated viral infections always risk for longer dysbiosis than usual that involve with disproportionate propagation of *Coprobacillus, Clostridium, Firmicutes, Bacteroides, Protobacteria* and *Actinobacteria* etc. Which allow the pathogens to settle down and flourish in our body releasing toxins, transforming the immune cells to hyperactive state tend to secrete TNF-α, IL-1β, MCP-1, IL-6 and chemokines could not only interfere to affect the vital organs, but also interfere with psychological wellbeing. Although probiotic interventions are not a standardized therapy yet, however, early use of bioactive compounds could confer the health benefits for patients. Vitamins A, E, C, B and D also potentiate the antiviral effects and contribute to metabolic and immune regulations. Moreover, the pharmacological like IFNs, sera and corticosteroids modulate the immune stimulatory response, whereas the antibiotics may remain harmful in case of their excessive usage, if not used appropriately. Vaccines and adjuvants are the better prophylactics to protect frontliners and normal population. Overall, the optimal use of nutraceuticals and pharmacological agents

not only shorten people's stay in hospitals, but also protect them contracting the secondary and opportunistic infections in any given susceptible environment, hence, reduce the healthcare burden.

Results:

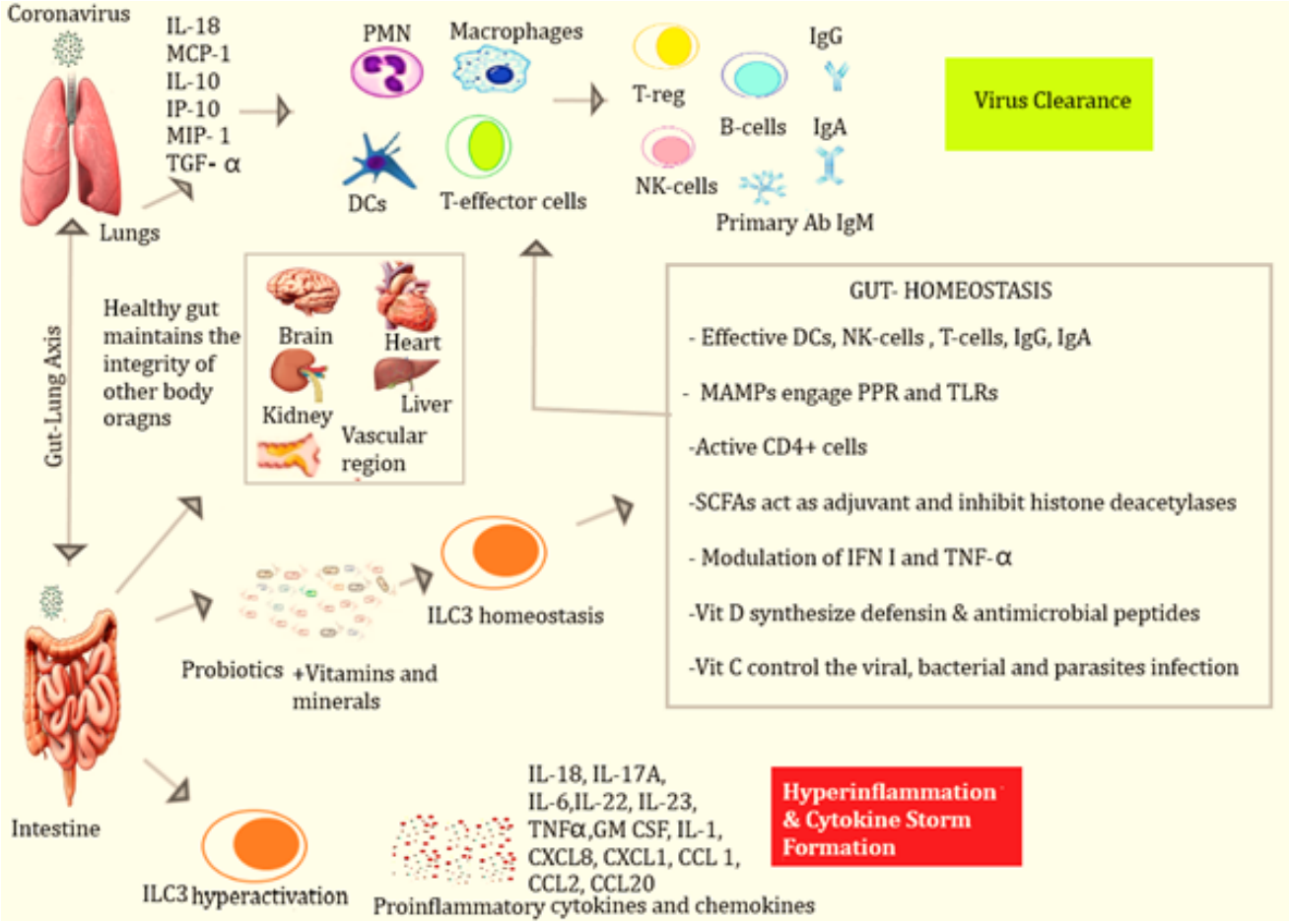


Figure 1:

Biography

Dr. Anju Kaushal is PhD Science (Microbiology -2003) from Panjab University, Chandigarh, India. She holds certificates in Quality Assurance and internal auditing from New Zealand Organization for Quality, NZ. Recently, she has also attained a certificate on Leading Strategic Projects - Portfolio and Program Management from University of Auckland, NZ. Currently, she is acting as Research Topic Co-ordinator for Journal – Frontiers in Antibiotics and also serving as a Guest Editor for Cureus Journal of Medical Science. She worked in various scientific & medical institutes and companies in India and New Zealand. Her expertise is in R&Ds, Productions and QA/QC in the field of biologicals, diagnostics and academia. She worked on Rabies, Aspergillus, Candida, HIV, Enzymes, fermentation technologies and others. Her area of interest includes vaccines, sera & diagnostics and novel therapeutics. Besides, she attained the skills in small business management, marketing and communications. She has >18 publications in peer reviewed journals and more than 40 publications on LinkedIn and attended many international conferences.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

The Potential Roles of IL-1β, IL-6, and RIPK3 in the Pathogenesis of Stevens Johnson Syndrome/Toxic Epidermal Necrolysis

Omer Iqbal[1], Chandana Sooranahalli[1] and **Charles Bouchard[2]**

[1]Loyola University Stritch School of Medicine, USA
[2]Loyola University Medical Center, USA

Stevens Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN) are a spectrum of disorders classified as Severe Cutaneous Adverse Reactions (SCARs), which are most often drug-induced and sometimes induced by bacterial infection due to Mycoplasma pneumoniae. These are T-cell mediated reactions. Specifically, cytotoxic $CD8^+$ T cells and natural killer (NK) cells play a key role. These T cells can induce apoptosis in keratinocytes through different pathways such as Fas-Fas ligand (FasL) pathway or the perforin/granzyme pathway. SJS/TEN is clinically characterized by a rash that spreads across the body and can include blistering and epidermal detachment and mucosal involvement of eyes, mouth and genitalia. Given that the pathogenesis of this systemic condition is not completely understood, the treatment is symptomatic and supportive involving multidisciplinary approaches. We studied the expression levels of inflammatory mediators such as IL-1β, IL-6 and receptor-interacting protein kinase 3 (RIPK3) in skin biopsies slides from patients with biopsy-confirmed SJS/TEN, using lichen planus as a positive control and normal skin as a baseline control. Immunohistochemistry was employed for this analysis. Additionally, the impact of SJS/TEN patient plasma on mitochondrial function was assessed in platelets and human corneal epithelial cells. Using a fluorescent plate reader, mitochondrial activity and superoxide ion levels were measured, comparing plasma from SJS.TEN patients to normal human plasma. Skin biopsies from SJS/TEN patients showed a significantly higher expression of IL-1β, IL-6, and RIPK3 compared to both lichen planus and normal controls.

Furthermore, plasma from SJS/TEN patients significantly reduced platelet viability and increased mitochondrial and total cellular superoxide ions, as demonstrated by elevated levels of MitoSOX Red and CellROX Red. These findings suggest that IL-1β, IL-6, and RIPK3

may contribute to the pathogenesis of SJS/TEN and highlight their potential as targets for therapeutic intervention.

Biography

Dr. Omer Iqbal is currently a research Professor in the departments of Ophthalmology and Pathology at Loyola University Stritch School of Medicine in Maywood, Illinois, USA. He is an elected fellow of the American College of Cardiology (FACC) and European Society of Cardiology (FESC). For a little over three decades, he has been involved in research in the fields of Hemostasis & Thrombosis, and Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN). He has mentored several medical students in research related to SJS. He has a wide and varied interests in the fields of new oral anticoagulants, Precision and Personalized Medicine, Pharmacogenomics, and Pharmacovigilance.

He is a recent recipient of a Certificate in Artificial Intelligence in Healthcare: Strategies and Implementation from Harvard University. He has participated as an invited speaker in several National and International conferences. He serves on editorial boards of several journals. He is a professional member of various international societies.

Ultrasonic-Assisted Coprecipitation of Magnetite Nanoparticles (Fe_3O_4) and Functionalization of Fe_3O_4@ Sio_2-NH_2 for Potential Magnetic Resonance Imaging Contrast Enhancement

Jahaziel Amaya

Universidad Antonio Nariño, Colombia

This work investigates the synthesis and functionalization of Fe_3O_4 nanoparticles for their potential use as MRI contrast agents. The Fe_3O_4 nanoparticles were synthesized through an ultrasonic-assisted coprecipitation method, with variations in the reducing agent addition rate to producing two distinct samples. These nanoparticles were then coated with a silica shell (SiO_2) using a sol-gel approach, resulting in Fe_3O_4@SiO_2 structures. The physicochemical properties of the nanoparticles were thoroughly characterized through several techniques, including transmission electron microscopy (TEM), selected area electron diffraction (SAED), X-ray diffraction (XRD), and X-ray photoelectron spectroscopy (XPS), which confirmed the uniformity, crystallinity, and composition of the particles. Their magnetic properties were analyzed using vibrating sample magnetometry (VSM), while dynamic light scattering (DLS) provided insights into their hydrodynamic size and colloidal stability. Fourier transform infrared spectroscopy (FTIR) demonstrated successful functionalization with SiO_2, introducing functional groups essential for further modifications. The nanoparticles exhibited an average size of 10.3 ± 1.7 nm, high crystallinity, and excellent structural integrity, suggesting their suitability for biomedical applications.

In addition, the nanoparticles were functionalized with a silica layer (SiO_2-NH_2) and conjugated with a microinflammation biomarker peptide. The resulting particles were characterized by high-resolution TEM (HR-TEM), scanning electron microscopy (SEM), and other techniques, revealing spherical nanoparticles smaller than 10 nm with high superparamagnetic properties and thermal stability. Cytotoxicity assays on Vero cells showed cell viability exceeding 85%, indicating the biocompatibility of the nanoparticles. The promising characteristics of these Fe_3O_4@SiO_2-NH_2 nanoparticles, including their magnetic properties and

functionalization, position them as potential candidates for enhancing MRI contrast, particularly in the detection of mild brain lesions, opening new possibilities in non- invasive diagnostic.

Biography

Dr. Jahaziel Amaya is a prominent researcher specializing in the synthesis and characterization of metallic nanoparticles for applications in bioengineering, particularly in the development of bionanosensors for medical use. His work has significantly contributed to the field of nanoparticle technology, focusing on their potential as diagnostic tools in medical imaging and disease detection. In addition to his research on nanoparticles, Dr. Amaya has extensive experience in the field of hydroconversion reactions and biomass transformation, with a strong emphasis on sustainable practices. His work aims to create environmentally friendly and economically viable biofuels, reducing the reliance on harmful substances. Dr. Amaya's interdisciplinary approach bridges nanotechnology, environmental science, and industrial applications, advancing the development of cleaner fuels and innovative medical technologies. His research in functionalized nanoparticles showcases their potential in enhancing medical diagnostics and therapeutic techniques, positioning him as a key figure in both nanotechnology and sustainable energy research.

Exploring Hepatitis B Virus Replicative Space

Pooja Bhatia[1], Aas Mohd[1], Harshita Katiyar[2], Amit Goel[2] and **Naga Suresh Veerapu[1]**

[1]Virology Section, Department of Life Sciences, Shiv Nadar Institution of Eminence, India
[2]Department of Hepatology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, India

Hepatitis B virus (HBV) variants arise from host-virus interactions and virus-unique replication features. New variants generate, and their establishment depends on replicative space determined by the levels of covalently closed circular DNA (cccDNA). We studied cccDNA levels under normal proliferative and growth-arrested conditions, and NTCP- dependent (external pathway; treated with anti-HBs Abs) and independent (internal pathway; treated with ciclopirox) conditions. Stable and permissive Huh7 cells were engineered with sodium taurocholate cotransporting polypeptide (NTCP) through genome editing. NTCP expression by the Huh7-ihNTCP cells was comparable to HepG2-NTCP cells reported earlier. Growth arrest was induced with DMSO, and the cell proliferation rate was nearly 40% lower than the normal growth-conditioned cells. Huh7-ihNTCP cells were infected with HBV GT-A at GEq 20/cell and cultured over 45 days. Growth-arrested cells had 2-log higher cccDNA and produced 1-log higher genomic DNA compared to cells cultured under normal growth conditions. For the sustenance of the virus, cells were treated with ciclopirox and anti-HBs sera at IC_{25} of 0.25 µM and at dilution of 1:30, respectively. HBV cccDNA levels and genomic DNA decreased during initial treatment times with anti-HBs sera and ciclopirox and later became stable. HBsAg and HBcAg level were also low on treatment with anti-HBs sera and ciclopirox compared to active and growth arrested conditions. Levels of cccDNA or genomic DNA from 1.3x HBV clones generated from the DNA circulating on day 45 of growth-arrested conditions and normal growth conditions didn't differ. However, 1.3x HBV clones isolated from NTCP-dependent conditions produced a high amount of cccDNA and HBsAg, while independent conditions produced high amounts of HBcAg, but not cccDNA and

genomic DNA compared to wild-type clone. Our findings explain the pattern of cccDNA in HBV limited replicative space.

Biography

Skilled in cell cloning, including the development of stable cell lines using genome editing techniques. Experienced in viral cell culture, particularly with hepatitis viruses, for studying infection dynamics and host-virus interactions. Proficient in Southern blotting for nucleic acid analysis, especially in the detection of viral cccDNA, and well-versed in a range of molecular biology techniques such as nucleic acid extraction, PCR, cloning, and gel electrophoresis.

Demonstrated ability to apply these techniques in long-term infection models and antiviral treatment studies. This work provides important insights into HBV persistence, replication control, and antiviral responses, which are critical for both fundamental research and translational healthcare applications.

PEERS ALLEY MEDIA

Joint Event

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY & FUTURE VIROLOGY

September 25-26, 2025 | Berlin, Germany

Lymphocytosis and Heteropenia in Rainbow Agama Lizards: A Haematological Study from Zaria, Nigeria

Olufisoye O. Ojo[1], Buzu B. Shedrack[2] and **James S. Sambo[2]**

[1]Department of Veterinary Public Health and Preventive Medicine, University of Ibadan, Nigeria
[2]Department of Veterinary Pathology, Ahmadu Bello University, Nigeria

There is a lack of information regarding the hematological indices of rainbow agama lizards in Zaria, Nigeria. Therefore, this study aims to determine their hematological values. A total of 100 lizards were randomly sampled across the region. Blood samples were collected from the caudal vein using a 21-gauge needle and syringe and stored in sterile vials containing an anticoagulant. The packed cell volume (PCV) was measured using the capillary micro-hematocrit technique, while the hemoglobin (Hb) concentration was determined via the cyanmethemoglobin method. Differential leucocyte counts were performed using a light microscope (Olympus XSZ-107BN) at high magnification with oil immersion after staining the slides with Wright-Giemsa stain.

The hematological parameters obtained for the rainbow agama lizard were as follows: PCV (34.88 ± 0.72%), Hb concentration (11.57 ± 0.24 g/dL), and total blood protein (6.69 ± 0.12 g/L). The total red blood cell (RBC) count was 5.87 ± 0.12 × 10^{12}/L, while the total white blood cell (WBC) count was 6.56 ± 0.38 × 10^{9}/L. Differential leucocyte counts revealed 0.59 ± 0.05 × 10^{9}/L (heterophils), 5.89 ± 0.36 × 10^{9}/L (lymphocytes), 0.047 ± 0.01 × 10^{9}/L (monocytes), and 0.02 ± 0 × 10^{9}/L (eosinophils).

The hematological parameters of rainbow agama lizards in northern Nigeria differed from those previously reported for their southern counterparts. This variation could be attributed to immunological responses to environmental and climatic differences, as well as endemic disease pathogens present in different regions. The blood analysis revealed lymphocytosis with severe heteropenia, suggesting generalized inflammation or infection. Further investigation is necessary to identify the causal agent of the infection or inflammation to accurately interpret the hematological findings of agama lizards in a clinical context.

Biography

Olufisoye Olusegun Ojo is a veterinarian specializing in poultry and aquaculture. He is currently affiliated with the University of Ibadan and El-Elyon Agric. His research focuses on One Health, molecular microbiology, infectious diseases, environmental toxicology, antimicrobial resistance and residues, as well as food safety and hygiene.

He holds a master's degree in veterinary medicine from the University of Ibadan, Nigeria, and has published several research papers in peer-reviewed journals. With extensive experience in field research and laboratory analysis, he has made significant contributions to advancing knowledge in poultry and aquaculture biosecurity, pathology, and infectious diseases.

At ADV. Immunology 2025, he will present on the hematological indices of rainbow agama lizards in Zaria, Nigeria, comparing the blood parameters of lizards in northern and southern Nigeria.

Passionate about scientific innovation and collaboration, he strives to bridge the gap between research and practical applications for a sustainable future.

Ergosterol & Quercetagetin as Dual-Action Anti-Cancer Agents: Decoding Steroid Metabolism *via* AR/ESR1 Crosstalk

Yujiao Chen[1], Jun Yang[1] and **Pooyan Makvandi[2]**

[1]Department of Physiology, Department of Cardiology of the Second Affiliated Hospital and School of Basic Medical Sciences, Zhejiang University School of Medicine, China.
[2]The Quzhou Affiliated Hospital of Wenzhou Medical University, Quzhou People's Hospital, China

Background: Conventional cancer therapies predominantly rely on synthetic or semi-synthetic agents targeting single molecular pathways often face limitations such as drug resistance, toxicity, and poor immune engagement. Hormone-driven malignancies, including liver and lung cancers, exploit steroid metabolism not only for proliferation but also to remodel the tumor microenvironment and evade immune surveillance. Here, we investigate ergosterol and quercetagetin, two phytochemicals that disrupt steroid metabolism via androgen receptor (AR) and estrogen receptor 1 (ESR1) crosstalk.

In this study, lung cancer and liver cancer cells were used as models. Active compounds were extracted and isolated from natural products through activity orientation, and further high-efficiency and low-toxicity anti-cancer lead compounds were screened through animal efficacy experiments. Based on single-molecule real-time transcriptome sequencing, by establishing a method for screening mutant proteins, the mechanism of action of anti-cancer lead compounds was studied from the perspective of hormone metabolism.

Results: Combining network pharmacology, molecular docking, and Single Molecule Real-Time (SMRT) sequencing, we demonstrate that ergosterol inhibits 3β-hydroxysteroid dehydrogenase (3βHSD), blocking hepatic conversion of dehydroepiandrosterone (DHEA) to androstenedione in liver cancer (HepG2). Quercetagetin suppresses 17β-hydroxysteroid dehydrogenase (17βHSD), destabilizing the estradiol/estrone equilibrium in lung cancer (A549). *In vivo* experiments in Lewis lung and H22 liver tumor models revealed dose-dependent tumor suppression, with quercetagetin mitigating splenomegaly and thymic atrophy—hallmarks of systemic immune dysregulation. Drug-likeness screening identified

8/31 compounds complying with Lipinski's Rule of Five, positioning ergosterol and quercetagetin as dual-action leads.

Conclusion: This study pioneers a natural product-driven strategy to concurrently target hormone metabolism and immune evasion, proposing AR/ESR1 inhibition as a bridge to enhance immunotherapy efficacy. Our findings redefine phytochemicals as multi-modal agents for hormone-dependent cancers.

Biography

Assistant Director, The National Engineering Research Center of Supercritical Fluid Technology and Equipment, China Aerospace Science and Industry Corporation. Postdoctor, *Zhejiang University*. Deputy General Manager and Chief Engineer, *Guizhou Gui'an Academy of Precision Medicine Co. Ltd*. Deputy Chief Engineer, *Guizhou Aerospace Intelligent Agriculture Co. Ltd*. Director of college of biological engineering alumni association of chongqing university. Deputy Secretary-General, chinese medicine modernization community, China Association of Chinese Medicine (2017-2022). Mainly engaged in the modernization of *Traditional Chinese Medicine*, gene sequencing in the field of scientific research and research results transformation related work.

Joint Event
ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY
&
FUTURE VIROLOGY
September 25-26, 2025 | Berlin, Germany

A Rare Case of Sepsis caused by *Klebsiella oxytoca* and *Aeromonas hydrophila*

Lixin Hua[1] and **Ruirui Yang**[2]

[1]Department of General Surgery, Affiliated Huishan Hospital of Xinglin College, Nantong University, Wuxi Huishan District People's Hospital, China
[2]Department of Science and Education, Affiliated Huishan Hospital of Xinglin College, Nantong University, China

Background: To the best of our knowledge, this is the first report of sepsis cases resulting from mixed infections of *Klebsiella oxytoca* and *Aeromonas hydrophila* worldwide.

Methods: We report a rare case of sepsis caused by a mixed infection of *Klebsiella oxytoca* (*K. oxytoca*) and *Aeromonas hydrophila* (*A. hydrophila*) encountered in China.

Results: The patient's disease progressed rapidly, posed a significant threat, and presented challenges throughout the diagnosis and treatment process. Fortunately, the patient was eventually cured and discharged from the hospital.

Conclusions: The peculiarity of this report lies in the spontaneous rupture of the patient's pancreatic cyst, leading to abdominal contamination and subsequent sepsis caused by a mixed infection of *K. oxytoca* and *A. hydrophila*. The prompt administration of rapid and effective antibiotics, along with supportive therapies, played a pivotal role in the successful recovery of our patient.

Biography

Lixin Hua obtained a doctorate in surgery from Sun Yat-sen University from 2012 to 2015. He engaged in clinical frontline work in general surgery for a long time. As a result, he has rich clinical experience in surgical treatment of severe abdominal infections, sepsis, inflammatory bowel disease, gastrointestinal malignancies, and retroperitoneal malignancies. He actively participates in scientific research work and timely publishes academic achievements. What is more, he often participates in the review work of journals such as International Journal of Surgery, International Journal of Oncology, Surgical Oncology, Oncology Letters, International Journal of Microbiology and Biotechnology, and World Journal of Gastroenterology, etc. He is currently a member of the editorial board of the journal Microbiology and Biotechnology.

Influenza Virus Regulation of Smad Signaling in Macrophages: Theoretical and Experimental Approach

Kareem Awad[1,2,3]

[1]Faculty of Medicine, Institute of Biomedicine, University of Turku, InFLAMES, Finland
[2]National Research Centre, Institute of Pharmaceutical and Drug Industries Research, Egypt
[3]Medical Faculty, Ruprecht-Karls University of Heidelberg, Germany

Influenza virus causes seasonal severe outcomes in human patients such like pneumonia, respiratory distress and cytokine storms. Transforming growth factor beta 1 (TGFβ1) multifaceted signaling in immune cells is complex based primarily on its specific Smad type phosphorylation. We aimed to study the effect of influenza/parainfluenza viruses' infection on the TGFβ1/Smad signaling in human monocytes derived macrophages. Monocytes were isolated from human buffy coats according to a standard procedure and infected with influenza viruses. Glycolytic pattern of infection was determined by monitoring the release of lactate and phosphofructokinase (PFK) activity in infected and uninfected cells. qRT-PCR was used to check the expression of essential viral and cell cytokines gene expression in cultured cells. Specific inhibitors were used to control TGFβ1/Smad signaling. Results show how different influenza strains may modulate TGFβ1-Smad signaling in immune cells and to what extent this differentially affects the expressed genes such like Smad2,3 and 7, HAMP and PLAUR. In conclusion, TGFβ1 signalling has dual pathways in human immune cells. Interference with specific Smad proteins could determine the outcome of influenza and parainfluenza viruses' infection in human.

Biography

Kareem Awad research concerns human immune cells responses to different pathogenic and non-pathogenic stimuli as well as the interaction of these cells with the surrounding nerves or vascular neighbouring cells. So, his previous work within years of experiences in different scientific schools in Finland, Egypt and Germany focused on the responses of these cells to pathogens such as influenza viruses' strains as well as signals from abnormal environmental contexts such like hyperglycaemia or tumour cells. In this sense, he targets diseases such as diabetes, influenza virus infection and cancers specifically the brain tumour glioblastoma. His last degree obtained from Cairo University is a PhD in Pharmaceutical Sciences "Biochemistry". More about his publications can be found on https://orcid.org/0000-0003-1007-9632.

Development of *ex vivo* Analysis for Examining Cell Composition, Immunological Landscape, Tumor and Immune Related Markers in Non-Small-Cell Lung Cancer

Vadim V. Kozlov[1,2], Elena G. Ufimtseva[3] and **Lyudmila F. Gulyaeva[4]**

[1]Novosibirsk State Medical University, Russian Federation
[2]Novosibirsk Regional Clinical Oncology Dispensary, Russian Federation
[3]Laboratory of Medical Biotechnology, Institute of Biochemistry, Federal Research Center of Fundamental and Translational Medicine, Russian Federation
[4]Laboratory of Molecular Mechanisms of Carcinogenesis, Research Institute of Molecular Biology and Biophysics, Federal Research Center of Fundamental and Translational Medicine, Russian Federation

Non-small-cell lung cancer (NSCLC) is a very aggressive solid tumor, with a poor prognosis due to post-surgical recurrence. Analysis of the specific tumor and immune signatures of NSCLC samples is a critical step in prognostic evaluation and management decisions for patients after surgery. Routine histological assays have some limitations. Therefore, new diagnostic tools with the capability to quickly recognize NSCLC subtypes and correctly identify various markers are needed. We developed a technique for *ex vivo* isolation of cancer and immune cells from surgical tumor and lung tissue samples of patients with NSCLC (adenocarcinomas and squamous cell carcinomas) and their examination on *ex vivo* cell preparations and, parallelly, on histological sections after Romanovsky–Giemsa and immunofluorescent/immunochemical staining for cancer-specific and immune-related markers. As a result, PD-L1 expression was detected for some patients only by *ex vivo* analysis. Immune cell profiling in the tumor microenvironment revealed significant differences in the immunological landscapes between the patients' tumors, with smokers' macrophages with simultaneous expression of pro- and anti-inflammatory cytokines, neutrophils, and eosinophils being the dominant populations. The proposed *ex vivo* analysis may be used as an additional diagnostic tool for quick examination of cancer and immune cells in whole tumor samples and to avoid false negatives in histological assays.

Biography

Dr. Vadim V. Kozlov, Medicine Doctor (Ph.D.-Medicine), is now the Head of the Thoracic Oncology Department in Novosibirsk Regional Clinical Oncology Center, Russian Federation, Researcher in the Research Institute of Molecular Biology and Biophysics, Federal Research Center for Fundamental and Translational Medicine, Member of the Russian Society of Clinical Oncology, Associate Professor of the Department of Oncology, Novosibirsk State Medical University. Currently, Dr. Vadim Kozlov's research is focused on studying the epigenetic mechanisms of lung cancer carcinogenesis depending on smoking status and histotype, as well as bioinformatics analysis and the search for new markers for lung cancer prognosis.

INDEX

ADV. IMMUNOLOGY 2025 & FUTURE VIROLOGY 2025

ADV. IMMUNOLOGY 2025 & FUTURE VIROLOGY 2025

PEERS ALLEY MEDIA

ADV. IMMUNOLOGY 2025
FUTURE VIROLOGY 2025

Peers Alley Media
1126 59 Ave East, V5X 1Y9, Vancouver BC, Canada
WhatsApp No: +1 (506) 909-0537

BOOKMARK YOUR DATES

4th EURO-GLOBAL SUMMIT ON

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY

&

4TH GLOBAL VIROLOGY CONGRESS

SEPTEMBER, 2026 | SPAIN