

4TH EURO-GLOBAL SUMMIT ON

ADVANCES IN CLINICAL AND CELLULAR IMMUNOLOGY

21-22
OCTOBER
2026



AMSTERDAM, NETHERLANDS

SCIENTIFIC PROGRAM

DAY 01
WEDNESDAY

OCTOBER 21, 2026

08:30-08:55	Registrations
08:55-09:00	Inaugural Ceremony
Moderator	Betsy J. Barnes , <i>Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, USA</i>
Sessions: Immunology Autoimmunity Medical Virology Cancer Immunology Immune System Antiviral Research Inflammation Cytokines and Chemokines Hypersensitivity, Asthma and Allergy Innate Immune Responses and Host Defense Vaccines and Immunotherapy Viral Vectors Emerging and Re-Emerging Viral Diseases	
Distinguished Speaker Talks	
Session Chair	Natarajan Aravindan , <i>Oklahoma State University, USA</i>
Session Chair	Katarzyna Napiórkowska-Baran , <i>Nicolaus Copernicus University, Poland</i>
09:00-09:20	Title: Vaccine Hesitancy in Diverse Communities: A Holistic, Culturally Humble Approach to Patient-Centered Care and Decision-Making Farzanna Sherene Haffizulla, <i>Dr. Kiran C. Patel College of Osteopathic Medicine, Nova Southeastern University, USA</i>
09:20-09:40	Title: Beyond Weight Loss: Addressing the Metabolic and Dermal-Aesthetic Consequences of GLP-1/GIP (Ozempic/ Mounjaro) Therapy with the G21 CRMP Protocol the Metabolic–Dermal Gap in Modern Obesity Treatment Sotirios Adamidis, <i>Athens Medical Center, Greece</i>
09:40-10:00	Title: Born Immunocompromised: Developmental Control of T Cell Immunity during Oncogenesis Natarajan Aravindan, <i>Oklahoma State University, USA</i>

10:00-10:20	Title: TLR-Induced STK25 Activation Promotes IRF5-Mediated Autoimmunity Betsy J. Barnes, <i>Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, USA</i>
GROUP PHOTO 10:20-10:30	
REFRESHMENT BREAK 10:30-10:50	
10:50-11:10	Title: Sulindac Modulates the Response of Proficient MMR Colorectal Cancer to Anti-PD-L1 Immunotherapy Yaguang Xi, <i>University of Georgia College of Pharmacy USA</i>
11:10-11:35	Title: T Cell Engaging Antibodies for Treating Cancer and Autoimmune Diseases Patrick Alexander Baeuerle, <i>Cullinan Therapeutics, Inc., USA & Ludwig Maximilians University of Munich, Germany</i>
11:35-11:55	Title: Ozone as an Immunomodulator—New Therapeutic Possibilities in the Treatment of Immunodeficiencies Katarzyna Napiórkowska-Baran, <i>Nicolaus Copernicus University, Poland</i>
11:55-12:15	Title: Education against Anti-Vaccine Disinformation Anna Magdalena Rędzio, <i>Warsaw University of Life Sciences, Poland</i>
12:15-12:45	Title: Beyond Antibiotics: Bacteriophages against <i>Pseudomonas aeruginosa</i> Biofilms and the Future of Orthopedic Device-Associated Infection Control Raquel Monteiro da Silva Lanna, <i>São Luiz Morumbi Hospital, Brazil</i>
12:45-13:05	Title: Selective Inhibition of Fc Receptor Shedding to Promote Anti-Tumor Immunity Lucas Ferrari de Andrade, <i>Icahn School of Medicine at Mount Sinai, USA</i>
GROUP PHOTO 13:05-13:15	
LUNCH BREAK 13:15-14:00	

14:00-14:20	Title: Not All Intermolecular Relationships are Linear: The STING Inflammatory Pathway David Kaplan, <i>CellPrint Biotechnology & Case Western Reserve University, USA</i>
14:20-14:40	Title: Update on Kidney Transplantation in Regard to ABO-Incompatible Blood Groups Maurizio Salvadori, <i>Careggi University Hospital Florence, Italy</i>
14:40-15:00	Title: Local and Systemic Chemerin Responses to Adjunctive Omega-3 Supplementation in Patients with Periodontitis and Type -2 Diabetes: A Randomized Clinical Trial Ayman Ahmed Shawky Elgawish, <i>The British University in Egypt, Egypt</i>
15:00- 15:20	Title: ICOSL Reverse Signaling Coordinates Liver Fibrogenesis, Immune Suppression and Hepatocarcinogenesis during MASH Progression Alessia Provera, <i>University of Piemonte Orientale (UPO), Italy</i>
15:20-15:40	Title: Immune-Mediated and Inflammatory Mechanisms of Post-COVID-19 Small Fiber Neuropathy and Chronic Pain Hana Mucevic, <i>Stony Brook Medical Center, USA</i>
15:40-16:00	Title: Still's Disease in Pregnancy Complicated by Suspected Anakinra-Related Hepatotoxicity and Successfully Managed with Intravenous Immunoglobulin Sara Hamza Elmubarak Abushama, <i>Kettering General Hospital, NHS Foundation Trust, UK</i>
REFRESHMENT BREAK 16:00-16:20	
16:20-16:40	Title: Herbal Protection against OTA-Induced Immunosuppression in Young Rabbits Kalina Vladimirova Zhivkova, <i>Trakia University, Bulgaria</i>

16:40-17:00	Title: Interplay between the Gut Microbiome, Diet and Clinical Outcomes in Multiple Sclerosis Simona Rolla, <i>University of Turin, Italy</i>
17:00-17:20	Title: Antineoplastic and Immunomodulatory Effects of Cabozantinib in Continuous and Primary Human Anaplastic Thyroid Cancer Cells Giusy Elia, <i>University of Pisa, Italy</i>
17:20-17:40	Title: Thyroid Cancer: Role of Neutrophil Extracellular Traps and Neutrophil-Related Mediators Francesca Ragusa, <i>University of Pisa, Italy</i>
17:40-18:00 (POSTERS)	Title: Innovative Hemodialysis and Public Health Policy Implications in CKD Andrés Morón Mendoza, <i>Centro Nacional de Investigación en Imagenología e Instrumentación Médica (CI3M), Universidad Autónoma Metropolitana - Unidad Iztapalapa, Mexico</i>
	Title: Single-Base Polymorphism of the ACE2 and TNF-Alpha Genes in Patients with the Cardiac and Indeterminate Forms of Chagas Disease Cilmery Suemi Kurokawa, <i>Medical School of Botucatu UNESP, Brazil</i>
	Title: Alternative Root Canal Irrigation Solutions and NonCytotoxic High Antibacterial Effectiveness Tahir ATAÖZDEN, <i>Biruni University, Türkiye</i>

CERTIFICATE PRESENTATION

END OF DAY-1

SCIENTIFIC PROGRAM

DAY 02
THURSDAY

OCTOBER 22, 2026

09:25-09:30

Introduction

Moderator

David Kaplan, *CellPrint Biotechnology & Case Western Reserve University, USA*

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

Distinguished Speaker Talks

Session Chair

Yogeeswari Perumal, *Birla Institute of Technology and Science- Pilani, Hyderabad Campus, India*

Session Chair

Maurizio Salvadori, *Careggi University Hospital, Florence, Italy*

09:30-09:50

Title: Mast Cell Activation Disease as a Pathophysiological Driver of Long COVID

Anne Maitland, *Medical University of South Carolina Ehlers Danlos Syndrome (EDS) Center, USA*

09:50-10:10

Title: Lysosomal Cysteine Proteases in Disease Modulation: Insights from Cancer and Pain Research

Yogeeswari Perumal, *Birla Institute of Technology and Science- Pilani, Hyderabad Campus, India*

10:10-10:30

Title: Infection Mitigation by Early Immune Activation

Jan Cuppen, *Wageningen University, The Netherlands*

GROUP PHOTO 10:30-10:40

REFRESHMENT BREAK 10:40-11:00

11:00-11:20	Title: Beta-Glucan Supplementation Improves Clinical Outcomes and Preserves Immune Homeostasis in Patients with Advanced Solid Tumors Receiving Chemotherapy: A Prospective Phase II Randomized Three-Arm Clinical Trial Wen-Chi Yang, <i>National Sun Yat-Sen University, Taiwan</i>
11:20-11:40	Title: Oxidative Stress-Induced Expression Levels of <i>PXDN</i> and <i>NF-κB</i> In Type 2 Diabetic Patients with Nephropathy Dalia Ali Gaber, <i>Gulf Medical University, UAE</i>
11:40-12:00	Title: Carcinoembryonic Antigen Beyond Cancer: Exploring its Associations with Glycaemic Dysregulation, Lipid Profile, and Vitamin D in Diabetes Noha Kamal Abdelmomen, <i>Gulf Medical University, UAE</i> <i>Theodor Bilharz Research Institute (TBRI), Egypt</i>
12:00-12:20	Title: Innate Immunity in Bronchopulmonary Dysplasia: The Role of the IL-1 Cytokine Family and the IL-1β-IL-8 Axis Andreja Nataša Kopitar, <i>University of Ljubljana, Slovenia</i>
12:20-12:40	Title: Development and Characterization of a Mouse Model of Hepatocellular Carcinoma for the Evaluation of Novel Immunotherapeutic Approaches Ekaterina Igorevna Ushakova, <i>Allergy and Immunology Research Centre FMBA of Russia, Russian Federation</i>
12:40-13:00	Title: The Acari Hypothesis Andrew C. Retzinger, <i>West Virginia University, USA</i>
GROUP PHOTO 13:00-13:10	
LUNCH BREAK 13:10-14:00	
14:00-14:20	Title: Autophagy Maintains High Endothelial Venule Identity and Function during Inflammation Kathryn Jacobs, <i>KU Leuven, Belgium</i>
14:20-14:40	Title: <i>In Situ</i> Reprogramming of Myeloid Cells with TLR Agonists in an Experimental Model of Triple-Negative Breast Cancer Ekaterina Igorevna Ushakova, <i>Allergy and Immunology Research Centre FMBA of Russia, Russian Federation</i>

14:40-15:00	Title: Risky Behaviours and Students' Academic Life in Higher Education Institutions in Tanzania Rose Ephraim Matete, <i>The University of Dodoma, Tanzania</i>
15:00- 15:20	Title: Modulating Tumor Mechanics and Vascular Barriers to Enhance Chemotherapy Efficacy in Pancreatic Tumors and Melanoma Stella Angeli, <i>The Cyprus Institute of Neurology & Genetics, Cyprus</i>
15:20-15:40	Title: Effects of Dietary Humic Acid Supplementation on Survival, Immune Response, and Gut Microbiota in Nile tilapia (<i>Oreochromis niloticus</i>) Challenged with <i>Lactococcus garvieae</i> Edén Vladimir Gaitán-Morado, <i>Universidad Michoacana de San Nicolás de Hidalgo, México</i>
15:40-16:00	Title: Immunological Aspects in case of Metabolic Dysfunction-Associated Steatotic Liver Disease Adrián Róbert Gál, <i>Institute of Physiology, University of Pécs Medical School, Hungary</i>
16:00-16:20	Title: Monosodium Glutamate as an Innate Immune Modulator: Evidence from Experimental Models and Implications for Food Safety Fatemeh Sayareh, <i>University of Warmia and Mazury in Olsztyn, Poland</i>
REFRESHMENT BREAK 16:20-16:40	
16:40-17:00	Title: Patient Perceptions and Type 1 Delay in Head and Neck Cancer: A Scoping Review of High- and Low-/MiddleIncome Countries Buhwa Gordon, <i>Makerere University, Uganda</i>

Title: Clq is Recognized as a Soluble Autoantigen by Anti-Clq Antibodies of Patients with Systemic Lupus Erythematosus

Vancho Donev, *Bul Bio—NCIPD EAD, Bulgaria*

17:00-17:30
(POSTERS)

Title: LIFE-CRAFT: A Multi-Agentic Conversational RAG Framework for Lifestyle Medicine Coaching with Context Traceability and Case-Based Evidence Synthesis

Hania Aslam, *Weill Cornell Medicine-Qatar, Qatar*

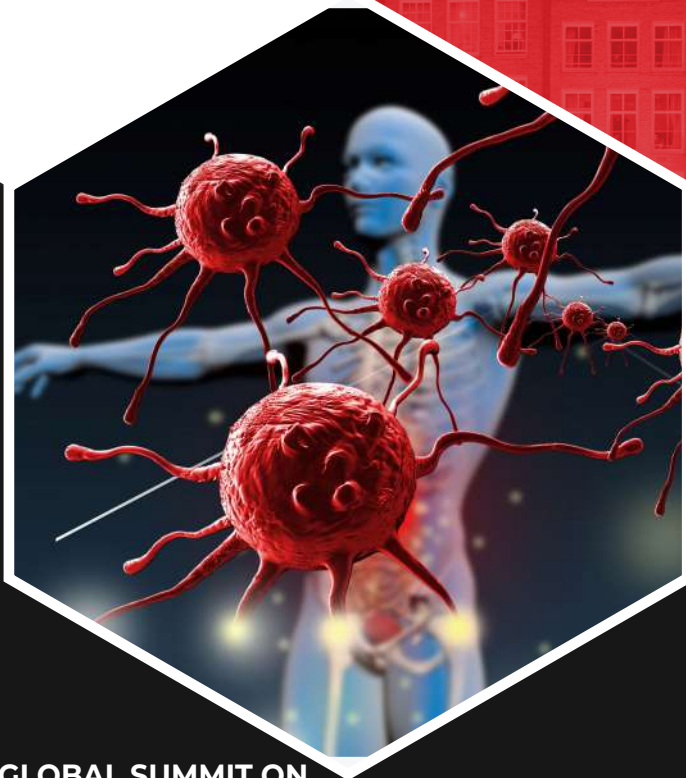
Title: Oncolytic Potential of Newcastle Disease Virus: Modulation of Apoptosis-Related Genes in the TC-1 Cell Line

Reihaneh Ramezani, *Islamic Azad University, Iran*

NETWORKING & CERTIFICATE PRESENTATION

END OF DAY-2

DAY 01



4TH EURO-GLOBAL SUMMIT ON

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SPEAKER TALKS



Vaccine Hesitancy in Diverse Communities: A Holistic, Culturally Humble Approach to Patient-Centered Care and Decision-Making

Farzanna Haffizulla

Dr. Kiran C. Patel College of Osteopathic Medicine
Nova Southeastern University, USA

Vaccine hesitancy remains a formidable barrier to global immunization equity, disproportionately affecting racially and ethnically diverse communities where historical medical abuses, structural racism, and persistent health disparities erode institutional trust. This chapter examines the multifactorial drivers of vaccine reluctance and presents a community-centered framework for strengthening vaccine confidence and uptake among underserved populations.

Drawing on a federally funded mixed-methods study conducted in Lauderdale, Florida. This community is a low-income, predominantly Black, Caribbean, and Hispanic community with a social vulnerability index of 0.76. This work engaged over 2,000 residents, business owners, and community leaders through surveys and focus groups grounded in community-based participatory research. Theoretical foundations include the WHO Behavioral and Social Drivers of Vaccination Framework, the Health Belief Model, and the Healthy People 2030 Social Determinants of Health framework.

Key findings reveal that vaccine hesitancy is shaped by concerns about vaccine safety and development speed, perceived coercion, breakthrough infection skepticism, misinformation from competing sources, and limited provider-initiated vaccine conversations. Participants emphasized the need for "genuine" messengers free of financial motive, cultural and age-appropriate education across multiple media formats, and respect for holistic and faith-based health practices. Top-down public health messaging proved insufficient; instead, empathetic communication using the COACH approach (Curiosity, Open, Appreciative, Compassionate, Honesty), motivational interviewing, and the five-step Collaboration Cycle demonstrated greater potential to build trust and facilitate informed decision-making.

This work concludes that achieving vaccination equity demands a paradigm shift toward culturally humble, relationship-driven strategies co-created with communities. Actionable recommendations include deploying vaccine ambassadors, multi-venue delivery, provider recommendation standardization, and behaviorally informed health messaging. By centering community voices and honoring lived experiences, healthcare systems can design sustainable in-

terventions that advance trust, improve vaccine uptake, and promote a more equitable global health landscape.

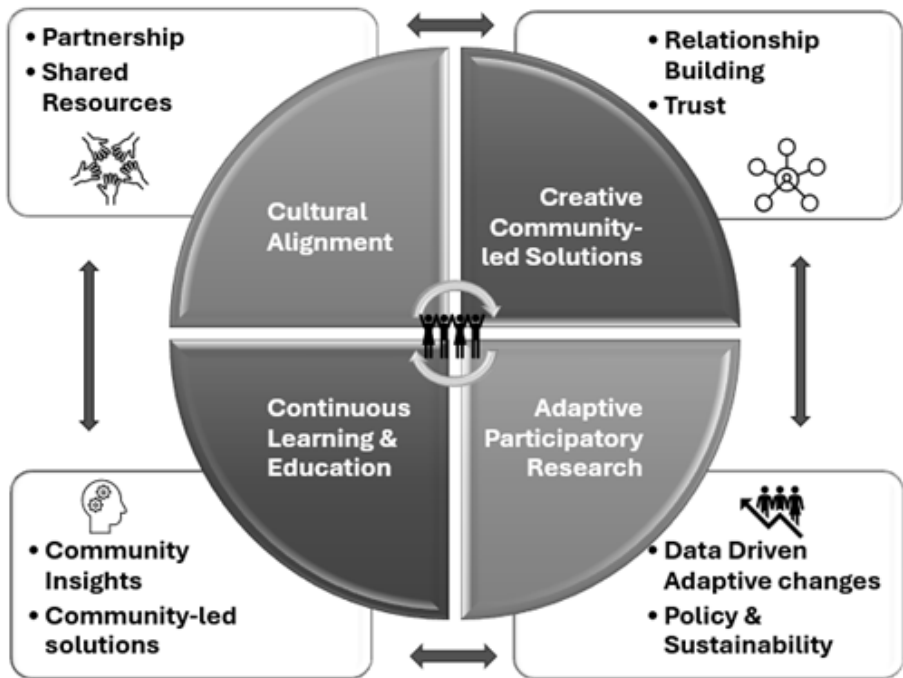


Figure 1: Collaborative Research Model for Health Equity; Haffizulla, Farzanna. (2025). Vaccine Hesitancy in Diverse Communities: A Holistic Approach to Patient-Centered Care. 10.1007/978-3-031-88668-3_9.

Presenter:

Farzanna Sherene Haffizulla

Dr. Kiran C. Patel College of Osteopathic Medicine
Nova Southeastern University, USA



Beyond Weight Loss: Addressing the Metabolic and Dermal-Aesthetic Consequences of GLP-1/GIP (Ozempic/Mounjaro) Therapy with the G21 CRMP Protocol

The Metabolic–Dermal Gap in Modern Obesity Treatment

Sotirios Adamidis¹, Adamidis Nikolaos² and Adamidi Sofia³

¹Director, Department of Internal Medicine
Director, Athens Medical Lifestyle Medicine, Athens Medical Center

President, ESoDiMeSO, Greece

²Internist, Clinical Practice, Berkshire Hospital, USA

³Internist, Charlton Memorial Hospital, Massachusetts, USA

Objectives: The introduction of GLP-1 receptor agonists with dual GLP-1/GIP (tirzepatide) or mono GLP-1 (semaglutide) agents, has transformed modern obesity management by achieving unprecedented metabolic and weight-loss outcomes. However, rapid adipose reduction frequently reveals a parallel clinical challenge characterized by dermal-aesthetic deterioration, including cellulite accentuation, skin laxity, connective tissue remodeling, and altered body contour morphology. This emerging phenomenon may represent a previously underrecognized therapeutic limitation in obesity medicine, described by us as the “Metabolic–Dermal Gap.”

Scope: Current obesity treatment paradigms focus primarily on metabolic endpoints such as weight reduction, glycemic control, and cardiovascular risk improvement, while insufficient attention is given to the dermal, extracellular matrix, microvascular, and lymphatic consequences accompanying rapid fat loss. Increasing evidence suggests that cellulite and dermal / subcutaneous tissue fibrosis are not purely cosmetic entities, but rather manifestations of adipose dysfunction, insulin resistance, chronic low-grade inflammation, and extracellular matrix remodeling.

Methods: The G21 CRMP (Cellulite Removal Medical Protocol) was developed as an integrated metabolic-dermal therapeutic approach designed to complement GLP-1/GIP-based treatment. The protocol combines systemic metabolic optimization with targeted dermal-adipose intervention aiming to improve tissue quality, microcirculation, dermal architecture, and aesthetic outcomes during and after pharmacologic weight reduction.

Results: Based on preliminary clinical observations and one-year follow-up

data, the combined use of tirzepatide/semaglutide with the EU-patented G21 topical formulation demonstrated promising complementary benefits in body contour transition, cellulite appearance, skin texture, and patient satisfaction. The proposed framework introduces a translational model integrating obesity medicine, lifestyle medicine, and aesthetic endocrinology into a unified therapeutic strategy.

Conclusion: This concept proposes that future obesity management should extend beyond weight loss alone and include restoration of metabolic and dermal integrity. Addressing the Metabolic–Dermal Gap may represent an important next step in precision obesity medicine and patient-centered long-term outcomes.

Presenter:

Sotirios Adamidis

Director, Department of Internal Medicine

Director, Athens Medical Lifestyle Medicine, Athens Medical Center

President, ESoDiMeSO, Greece



Born Immunocompromised: Developmental Control of T Cell Immunity during Oncogenesis

Natarajan Aravindan

Department of Physiological Sciences, College of Veterinary Medicine, Oklahoma State University, USA

Effective antitumor immunity is rooted in proper T-cell (Tc) development within the thymus, yet the embryonic programs that preconfigure immune competence remain incompletely understood. Although neural crest cells (NCC) are essential contributors to thymus organogenesis, the intrinsic molecular programs within this lineage that predefine immune function remain poorly defined. We identified retinal degeneration protein 3 (RD3) as a critical developmental regulator of thymic architecture, Tc maturation, and tumor immune control. Loss of RD3 within the NCC resulted in profound defects in thymic organization (size, corticomedullary architecture, stromal composition), indicating impaired thymic patterning rather than complete developmental arrest. While early thymocyte commitment and selection were largely conserved, terminal Tc differentiation was significantly skewed. RD3 deficiency reduced maturation of CD4⁺ Tc while maintained a relative expansion of CD8⁺ Tc, revealing a detour in lineage specification and immune education. These defects were reflected systemically, with a marked reduction in total CD3⁺ Tc in the periphery, directly linking altered thymic programming to diminished immune capacity. Consistent with this impaired cellular immunity, circulating levels of key effector cytokines (IFN γ , TNF α) were reduced, indicating compromised systemic immune activation. Crucially, this developmental immune imbalance translated into functional immune evasion *in-vivo*. Spontaneously arising tumors exhibited features of an immunologically suppressed microenvironment, characterized by diminished T cell infiltration and coordinated dysregulation of immune activation, memory, and inhibitory pathways. Multiplex immune profiling revealed altered expression of Tc identity and activation (CD4, CD8, CD44, GITR, CD95), immune exhaustion and inhibitory signaling (CD244.2), adenosinergic metabolic suppression (A2AR, CD39, CD73), myeloid polarization (CD206), and immune sensing (STING). These outcomes establish RD3 as a developmental determinant of thymus organogenesis and immune surveillance, revealing that immune dysfunction and tumor immune evasion can originate from embryonic NCC intrinsic defects. Our findings provide a developmental framework for immune failure and cancer susceptibility, with relevance to pediatric malignancies.

Presenter:

Natarajan Aravindan

Oklahoma State University, USA



TLR-Induced STK25 Activation Promotes IRF5-Mediated Autoimmunity

Betsy J. Barnes^{1,2,3}, Matthew R. Rice^{1,2}, Bharati Matta², Loretta Wang², Qi Luo⁴, Jeremy De Guzman⁴, Dinesh Srinivasan⁴, Katelyn R. Ludwig⁵, Surya Indukuri^{1,2}, Leianna Brune² and Seng-Lai Tan⁴

¹Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, USA

²Institute of Molecular Medicine, The Feinstein Institutes for Medical Research, USA

³Departments of Molecular Medicine and Pediatrics, Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, USA

⁴Hoffman La-Roche, USA

⁵Department of Integrative Physiology, University of Colorado Boulder, USA

Systemic lupus erythematosus (SLE) is a highly complex and variable autoimmune disease, yet common threads run through it, including loss of self-tolerance, persistent autoantibody production, enhanced type I interferon expression, complement activation, inflammation, and tissue damage. The transcription factor interferon regulatory factor 5 (IRF5) is an essential mediator of innate and adaptive immunity downstream of Toll-like receptor (TLR) signalling and *IRF5* genetic variants have the second highest odds ratio for SLE risk after *HLA* genes. We previously reported that healthy donors carrying the *IRF5*-SLE risk haplotype and SLE patients share a common phenotype of constitutive IRF5 nuclear translocation or hyper-activation. Thus, while dysregulation of IRF5 activity has been implicated in the development of SLE, the factors that modulate IRF5 activation are poorly understood. Here, we performed a kinome-wide siRNA screen and identified serine/threonine protein kinase 25 (STK25) as a positive regulator of proinflammatory cytokine production via phosphorylation of IRF5 at Thr265, leading to IRF5 transcriptional activation. We further found that STK25 undergoes autophosphorylation in response to multiple SLE relevant triggers. Findings were validated in *Stk25*-deficient primary immune cells revealing a significant attenuation in *R848*-induced IRF5 nuclear translocation and proinflammatory cytokine production. Further, littermate wild-type and *Stk25*-deficient mice were injected with pristane and lupus onset and severity determined. *Stk25*-deficient mice were also crossed to TLR7.1 mice as a model of TLR7-driven lupus. Finally, we detected increased levels of STK25 autophosphorylation in immune cells from SLE donors com-

pared to healthy controls. These findings implicate STK25 as a new regulator of TLR7/8 signalling through the modulation of IRF5 activation.

Presenter:

Betsy J. Barnes

Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, USA



Sulindac Modulates the Response of Proficient MMR Colorectal Cancer to Anti-PD-L1 Immunotherapy

Yaguang Xi

Professor, University of Georgia College of Pharmacy, USA

Although immune-checkpoint inhibitor (ICI) therapy has become a cornerstone in treating various human cancers, especially advanced solid tumors, its efficacy in colorectal cancer remains limited, benefiting only around 15% of patients, mainly those with microsatellite instability (MSI) tumors indicating defective DNA mismatch repair (dMMR). Conversely, patients with proficient DNA mismatch repair (pMMR) typically experience little benefit from ICIs. To address this, we investigated the potential of sulindac to augment the response of pMMR colorectal cancer to anti-PD-L1 immunotherapy. Using syngeneic mouse tumor models and humanized Patient-Derived Xenograft (PDX) animal models, we evaluated the individual and combined effects of PD-L1 antibody (Ab), sulindac, and their combination on tumor growth. Our results revealed that the combination therapy significantly reduced tumor volume and promoted infiltration of CD8⁺ T lymphocytes into the tumor microenvironment. Mechanistically, sulindac downregulated PD-L1 expression by inhibiting NF- κ B signaling, consequently reducing exosomal PD-L1 levels. Importantly, we observed that exosomal PD-L1 in circulation can sequester PD-L1 Ab, thus limiting its efficacy. Therefore, by lowering PD-L1 expression, sulindac enhances the availability and efficacy of PD-L1 Ab in combination therapy. Furthermore, we demonstrated that low-dose sulindac did not exert systemic inhibitory effects on prostaglandin E2 (PGE2). In summary, our findings shed light on the immunomodulatory role of sulindac in enhancing anti-PD-L1 therapy for pMMR colorectal cancer, offering potential therapeutic insights.

Presenter:

Yaguang Xi

University of Georgia College of Pharmacy, USA



T Cell Engaging Antibodies for Treating Cancer and Autoimmune Diseases

Patrick A. Baeuerle

Cullinan Therapeutics, Inc., USA
Faculty of Medicine, Institute for Immunology, Biomedical Center, Ludwig Maximilians University of Munich, Germany

T cell-engaging antibodies (TCEs) are bispecific, antibody-based adaptor proteins that can connect every kind of cytotoxic T cells with select target cells for redirected lysis that does not require peptide/MHC complexes or TCR specificity. One arm of a TCE binds a select cell surface antigen on the target cell, the other arm binds to the invariant CD3 epsilon subunit of the T cell receptor complex. This will trigger formation a cytolytic synapse through which perforin and granzymes can eradicate the attached target cell.

A total of twelve TCEs are now approved by the FDA or EMA that very effectively treat as a monotherapy B cell-derived leukaemia, lymphomas and multiple myeloma. Likewise, solid tumors derived from uveal melanoma, small cell lung cancer and ovarian cancer can now be treated with TCEs. I will review the learnings from the twelve approved TCEs in terms of target selection, design and biochemical characteristics. TCEs will also be compared to CAR-T cell therapies.

A spade of CD19- and BCMA-targeted TCEs for deep depletion of autoreactive B cells is currently being tested in early clinical trials for treating rheumatoid arthritis, lupus erythematosus, Sjogren's Disease and several other autoimmune disorders with promising results. Future trends such as the combination of TCEs with standard of care in first line therapy, the use of TCEs in minimal residual disease, the search for new TCE targets and the development of conditionally activated TCEs will be discussed. Cullinan's clinical-stage TCE pipeline targeting CD19, BCMA and FLT-3 will be highlighted that is being developed for treating autoimmune diseases and acute myeloblastic leukaemia, respectively.

Presenter:

Patrick Alexander Baeuerle

Cullinan Therapeutics, Inc., USA
Ludwig Maximilians University of Munich, Germany



Ozone as an Immunomodulator—New Therapeutic Possibilities in the Treatment of Immunodeficiencies

Katarzyna Napiórkowska-Baran

Department of Allergology, Clinical Immunology and Internal Diseases, Collegium Medicum, Nicolaus Copernicus University, Poland

Ozone therapy has emerged as a promising adjunctive strategy with potential immunomodulatory effects, yet its role in the management of immunodeficiencies remains insufficiently explored. The aim of this study was to evaluate the potential of ozone as a therapeutic tool influencing immune system function, with particular emphasis on patients with primary and secondary immunodeficiencies.

Ozone (O₃), administered in controlled medical concentrations, exhibits a dual biological effect—mild oxidative stress induction and subsequent activation of antioxidant defense pathways. This process leads to upregulation of key protective mechanisms, including increased activity of superoxide dismutase (SOD), catalase, and glutathione peroxidase. Moreover, ozone has been shown to modulate cytokine production, enhancing immune balance by influencing both innate and adaptive immune responses.

Experimental and clinical observations suggest that ozone therapy may improve tissue oxygenation, reduce chronic inflammation, and enhance antimicrobial defense mechanisms. In patients with recurrent respiratory infections and impaired immune responses, ozone inhalation and ozonated water therapies may contribute to a reduction in infection frequency and severity, as well as improvement in quality of life.

Additionally, selected clinical case photographs will be presented to illustrate therapeutic outcomes and practical applications of ozone therapy in patients with immunodeficiencies.

Despite promising findings, current evidence is still limited and heterogeneous. Further well-designed randomized clinical trials are necessary to establish standardized protocols, optimal dosing, and long-term safety.

In conclusion, ozone therapy represents a novel and potentially valuable immunomodulatory approach, particularly in the context of immunodeficiencies, where conventional therapies remain insufficient.

Presenter:

Katarzyna Napiórkowska-Baran

Nicolaus Copernicus University, Poland



Education against Anti-Vaccine Disinformation

**Anna M. Rędzio¹, Kamil Izydorczak², Paweł Muniak³
and Dariusz Doliński²**

¹Warsaw University of Life Sciences, Poland

²Faculty of Psychology in Wrocław, SWPS University, Poland

³Faculty of Psychology in Warsaw, SWPS University, Poland

Anti-vaccine propaganda is a pressing societal challenge today. While improvements in vaccine effectiveness and safety continue, their impact is limited when growing numbers of individuals refuse to vaccinate themselves or their children. Due to the widespread availability of the Internet and enormous popularity of social media anti-vaccine mis- and disinformation spreads faster and more effectively than ever before. This is why education aimed at increasing awareness of disinformation is so important and teaching people on this subject is a task for scientists as well as doctors or teachers.

However, it is essential for them to know how to educate people or encourage them to get vaccinated because vaccination is a topic that can arouse strong emotions, and in such situation simple correction of misinformation is ineffective. This presentation summarizes several experimental studies on reducing the effects of anti-vaccine propaganda in Poland and encouraging people to vaccinate.

A popular social influence technique called “But you are free”, which has been effective in many other social situations – such as request for charitable donations – proved to be counter-effective in this context: Parents who were told by a doctor or a nurse “But you are free” (to vaccinate your baby or not) decided to vaccinate their children less often than parents who were not told that.

Manipulation based on playing the online game “COVID-19 Fake News”, inspired by the well-known game “Bad News” (which has been effective as a kind of inoculation against disinformation), had no significant effect on attitudes toward vaccinations when compared to the control condition. But the results of studies in which the participants were informed that most of anti-vaccine online content comes from Russian trolls look a bit more encouraging. The results of a few other studies, both quantitative and qualitative, may also provide guidance on how to encourage patients to get vaccinated.

Presenter:

Anna Magdalena Rędzio

Warsaw University of Life Sciences, Poland



Beyond Antibiotics: Bacteriophages against *Pseudomonas aeruginosa* Biofilms and the Future of Orthopedic Device-Associated Infection Control

Raquel Monteiro da Silva Lanna¹, Amanda Kelly Ferreira Sousa², Viviani Tadioto², Anderson Giehl², Helena Yurevna Caio², Estevão Brasiliense de Souza², Evelin Padilha Corrêa², Mariana Alves Elois², Estevan Rodrigues dos Santos Neto³, Sergio Luiz Alves Junior², Ariadne Cristiane Cabral da Cruz², Mara Cristina Scheffers², Maria Célia da Silva Lanna³ and Gislaine Fongaro²

¹São Luiz Morumbi Hospital, Brazil

²Federal University of Santa Catarina, Brazil

³Federal University of Ouro Preto, Brazil

Orthopedic device-associated infections remain difficult to eradicate because bacteria colonize implant surfaces and mature into biofilms that withstand antimicrobial therapy and host immune responses. This problem affects trauma fixation and joint replacement surgery, where infection may compromise implant survival, bone healing, function and outcomes.

Although *staphylococci* predominate, Gram-negative organisms, including *Pseudomonas aeruginosa* (*P. aeruginosa*), are clinically relevant in severe contaminated trauma, open fractures, repeated surgical procedures and selected implant-associated infections.

This narrative and translational review is centered on “Bacteriophage-mediated reduction of *Pseudomonas aeruginosa* biofilm on titanium surfaces for biomedical applications,” aiming to translate recent *in vitro* evidence on phage-mediated antibiofilm activity against *P. aeruginosa* on titanium into an orthopedic infection-control perspective. The experimental model evaluated *P. aeruginosa* American Type Culture Collection (ATCC) 27853 and a clinical isolate on titanium specimens. Five native lytic bacteriophages, PA1 to PA5, were tested against established four-day biofilms and as prophylactic agents during biofilm formation at multiplicities of infection (MOI) of 0.01, 0.1 and 1. Antibiofilm activity was assessed through viable bacterial counts and scanning electron microscopy (SEM) with semi-quantitative image analysis.

Bacteriophage treatment markedly reduced viable biofilm-associated bacteria and titanium surface coverage. Reduction efficiencies ranged from 50.53% to 99.78% for the reference strain and from 0.01% to 99.94% for the clinical isolate, with PA4 and PA5 showing the most consistent activity and reductions above

99%. Prophylactic application suppressed biofilm formation, while no consistent dose-response relationship was observed.

Orthopedic phage therapy against *P. aeruginosa* is no longer merely theoretical: preclinical implant-related and bone infection models, phage-delivering hydrogels, phage-activated titanium surfaces and compassionate clinical reports support its translational relevance.

However, evidence for controlling mature *P. aeruginosa* biofilms on titanium orthopedic surfaces remains incompletely defined. Bacteriophages should therefore be positioned as emerging adjunctive antibiofilm tools, not substitutes for surgical debridement, implant decision-making or antimicrobial therapy.

Presenter:

Raquel Monteiro da Silva Lanna

São Luiz Morumbi Hospital, Brazil



Selective Inhibition of Fc Receptor Shedding to Promote Anti-Tumor Immunity

Lucas Ferrari de Andrade

Icahn School of Medicine at Mount Sinai, USA

Antibodies protect against infections and cancers in part through the engagement of Fc-activating receptors, such as CD16a that triggers antibody-dependent cellular cytotoxicity (ADCC) by natural killer (NK) cells. On the other hand, CD16a surface expression is downregulated by ectodomain shedding, which is a post-translational modification that transforms surface proteins into soluble polypeptides. Historically, *in vitro* experiments with NK cells that were treated with protease inhibitors or genetically engineered to express a mutant CD16a resistant to cleavage revealed a dual-contrasting role for CD16a shedding in ADCC: it inhibits NK cell effector functions but promotes serial killing. However, the physiological significance of CD16a shedding had remained largely uncharacterized because CD16a shedding is difficult to inhibit with high specificity *in vivo*. We, therefore, generated a novel monoclonal antibody (F9H4) that binds CD16a and inhibits the shedding in a substrate-specific manner, a unique feature that enables *in vivo* studies in immunocompetent hosts. F9H4 is not an agonist or blocker. Our studies in Fc gamma receptor-humanized mice revealed that systemic immunoestimulation is followed by CD16a shedding, which, in turn, is inhibited by F9H4. As a consequence, F9H4 greatly increases the CD16a surface expression in blood NK cells *in vivo*. Furthermore, analysis of tumor explants from patients revealed that tumor-infiltrating leukocytes shed CD16a, but F9H4 inhibits the shedding, and, in preclinical models, F9H4 inhibits tumor growth by an NK cell-mediated mechanism. Therefore, CD16a shedding is a homeostatic mechanism for immune regulation, but it can be selectively inhibited by F9H4 to promote anti-tumor immunity.

Presenter:

Lucas Ferrari de Andrade

Icahn School of Medicine at Mount Sinai, USA



Not All Intermolecular Relationships are Linear: The STING Inflammatory Pathway

David Kaplan^{1,2} and Eric Christian¹

¹CellPrint Biotechnology, USA

²Case Western Reserve University, USA

Understanding intracellular signaling pathways is crucial since they regulate essential functional activities. We have studied the configuration of the STING inflammatory pathway in the major mononuclear cell-types from the blood of patients with varying degrees of atherosclerotic coronary artery disease. Interestingly, we found that the STING pathway is configured differently in B cells compared to T cells or monocytes. Additionally, bivariate relationships have been useful to delineate signaling pathways in clinical samples. In our previous studies we have found many linear associations between pathway components, and we have interpreted these correlations as rheostatic regulators. Increases in an upstream component are correlated with a commensurate downstream increase. Here, we report a quantitative analysis of molecules in the STING pathway by assessing the variance in human peripheral blood mononuclear cell-type specific molecular expression from patients with atherosclerotic coronary artery disease. The induction of the type I interferon track by this pathway is dependent on expression levels of STING in T cells and monocytes and expression levels of phospho-STING in B cells. This relationship in the T cells and monocytes demonstrates definitive linearity so that it is regulated as a rheostat. In B cells the relationships are logarithmic indicating an on-off mechanism of regulation. The STING pathway dependent stimulation of the NFkB track is also controlled by on-off mechanisms that are modeled by non-linear bivariate relationships. These on-off switches occur at the bifurcation of the two branches involving phospho-STING, phospho-TBK1, and phospho-ReIA. Whereas linear bivariate associations are readily captured by an evaluation of the correlation matrix, significant nonlinear relationships are not. Nonlinear correlations modelled logarithmically or exponentially are easily discerned by the assessment of a natural log transformed versus non-transformed correlation matrix.

Presenter:

David Kaplan

CellPrint Biotechnology, USA

Case Western Reserve University, USA



Update on Kidney Transplantation in Regard to ABO-Incompatible Blood Groups

Maurizio Salvadori

Department of Renal Transplantation, Careggi University Hospital, Florence, Italy

Kidney transplantation for patients affected by end stage renal failure is considered the best therapeutic option, but this possibility is limited by deceased donor shortage. Living donor kidney transplantation (LDKD) is a valuable option, but this option is frequently limited by immunological incompatibility between donor and recipient.

Once the transplantation between incompatible donor and recipient was a formal contraindication. Along the years several strategies have emerged documenting the possibility of performing this transplantation. This review will consider the to date possibility of performing living donor kidney donation in the case of ABO blood group incompatibility and the progresses that have been made in this field.

Kidney paired donation is one possibility. Such technique offers the best possibility if the number of pairs available is wide. This is possible by performing national and also international registries.

The technique more diffuse is the desensitization of the recipients. Desensitization may be obtained by several ways that are extensively treated in this review.

In the recent period some published studies document the possibility to enzymatically convert A or B group from the cells of the donor to O group. This possibility is only at the beginning, but may represent the future eventually associated to a mild desensitization.

Presenter:

Maurizio Salvadori

Careggi University Hospital, Florence, Italy



Local and Systemic Chemerin Responses to Adjunctive Omega-3 Supplementation in Patients with Periodontitis and Type -2 Diabetes: A Randomized Clinical Trial

Ayman El-Gawish, Dalia Ghalwash and Ahmed Hamdy

Oral Medicine and Periodontology, Faculty of Dentistry, The British University in Egypt, Egypt

Aims: The present study aspired to evaluate the impact of the adjunctive use of omega-3 with non-surgical periodontal therapy on clinical parameters as well as local and systemic chemerin levels as a marker of cardiovascular disease risk in periodontitis patients with diabetes.

Methods: This randomized clinical trial was performed on thirty periodontitis patients with type II diabetes divided into two equal groups, both treated by nonsurgical periodontal treatment with the adjunctive use of daily 1000mg Omega-3FAs in group I only for 6 months. Patients were reexamined after 2weeks (baseline), 3 and 6 months for recording the clinical parameters as follows: plaque index (PI), gingival index (GI), probing depth (PD), and clinical attachment loss (CAL). Chemerin levels were assessed in both serum and GCF samples, HbA1c levels were also assessed.

Results: Omega-3FAs-treated group recorded a more statistically significant improvement in clinical parameters compared to the control group, particularly concerning PD and CAL. A statistically significant reduction of HbA1c levels between baseline, 3 and 6 m values was encountered in Omega-3FAs treated group, while no significant difference was evident in the control group.

Additionally, Omega-3FAs treated group recorded a more statistically significant reduction of GCF and serum chemerin levels in comparison to the control group after 6 months of therapy.

Conclusion: The adjunctive use of Omega-3FAs with non-surgical periodontal therapy has resulted in significant improvement of clinical periodontal parameters and glycemic control in periodontitis patients with type II diabetes, alongside the additional benefit of reducing both local and systemic chemerin levels, a biomarker for cardiovascular risk.

Presenter:

Ayman Ahmed Shawky Elgawish

The British University in Egypt, Egypt



ICOSL Reverse Signaling Coordinates Liver Fibrogenesis, Immune Suppression and Hepatocarcinogenesis during MASH Progression

A. Provera¹, A.N. Sheferaw¹, E. Kivumbi¹, C. Vecchio¹,
I. Stoppa¹, D. Pantham¹, F. Christaki¹, Erica Novo²,
U. Dianzani¹ and S. Sutti¹

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²Department of Clinical and Biological Sciences, Unit of Experimental Medicine and Clinical Pathology, University of Turin, Italy

Background: Metabolic dysfunction-associated steatohepatitis (MASH) is a leading cause of hepatocellular carcinoma (MASH-HCC), yet the mechanisms linking chronic liver inflammation to tumour development remain poorly understood. Besides acting as the ligand for the inducible T-cell co-stimulator (ICOS), ICOS ligand (ICOSL) also transduces intracellular signals through reverse signaling. We investigated whether ICOSL reverse signaling contributes to fibrogenesis, immune suppression and hepatocarcinogenesis.

Materials and Methods: Wild-type (WT), ICOS-deficient (ICOS-KO) and ICOSL-deficient (ICOSL-KO) mice received a Western diet to induce fibrosis or diethylnitrosamine followed by a choline-deficient amino acid-defined (CDAA) diet to establish MASH-associated HCC. ICOSL reverse signaling was triggered with recombinant ICOS-Fc. Complementary *in vitro* studies were performed using RAW264.7 macrophages and primary hepatic stellate cells (HSCs).

Results: Both ICOS- and ICOSL-deficient mice exhibited reduced fibrosis and hepatocarcinogenesis. Compared with WT mice, both knockout strains showed reduced accumulation of activated myofibroblast-like cells, fewer and smaller HCC nodules, reduced neoangiogenesis, decreased IL-6/STAT3 activation, and enhanced anti-tumour immune surveillance, evidenced by increased CD8⁺ T-cell-enriched tertiary lymphoid structure (TLS)-like aggregates.

Concordant genetic and functional data supported a central role for ICOSL reverse signaling. *In vitro*, ICOS-Fc-induced ICOSL reverse signaling promoted HSC activation and proliferation. Consistently, ICOS-Fc promoted liver fibrosis and tumour progression in WT and ICOS-KO, but not ICOSL-KO, mice. Activation of ICOSL reverse signaling enhanced tumour cell proliferation and neoangiogenesis, reduced TLS formation and intratumoural CD8⁺ T-cell infil-

tration, and established an immunosuppressive hepatic microenvironment. Mechanistically, ICOSL reverse signaling increased macrophage IL-6 and Annexin A1 expression *in vitro* and *in vivo*. Conditioned media from ICOSL-activated macrophages enhanced Hepa1-6 proliferation, supporting macrophage-derived mediators in tumour promotion.

Conclusions: ICOSL reverse signaling coordinates hepatic microenvironment remodeling by integrating fibrogenesis, macrophage-dependent immune suppression, angiogenesis and tumour growth while limiting anti-tumour immune surveillance. These findings identify ICOSL reverse signaling as a therapeutic target to prevent progression from advanced MASH to HCC.

Presenter:

Alessia Provera

University of Piemonte Orientale (UPO), Italy



Immune-Mediated and Inflammatory Mechanisms of Post-COVID-19 Small Fiber Neuropathy and Chronic Pain

Hana Mucevic¹, Anirudh Bhimavarapu¹, Sadiq Rahman² and Amruta Desai²

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USA

²Department of Anesthesiology and Pain Medicine
Stony Brook Medical Center, USA

Chronic neuropathic pain has emerged as a significant and debilitating component of post-acute sequelae of SARS-CoV-2 infection (long COVID). Increasing evidence suggests that small fiber neuropathy (SFN) plays a central role in the development of persistent sensory and autonomic symptoms in affected patients. This review aims to synthesize current evidence on the pathophysiology, diagnostic approaches, and management strategies for chronic pain associated with SFN secondary to SARS-CoV-2 infection.

A comprehensive literature review was conducted using PubMed and Google Scholar, focusing on studies published between January 2020 and January 2025. Peer-reviewed clinical trials, observational studies, case series, and systematic reviews examining SFN and post-COVID-19 neuropathic pain were included. Data were extracted regarding clinical presentation, proposed mechanisms, diagnostic modalities, and treatment outcomes.

The reviewed literature demonstrates that post-COVID-19 SFN presents with heterogeneous sensory and autonomic manifestations, including burning pain, allodynia, dysesthesias, and gastrointestinal or genitourinary dysfunction. Diagnosis relies on careful clinical evaluation supported by quantitative sensory testing and confirmatory skin biopsy assessing intraepidermal nerve fiber density. Proposed mechanisms include immune-mediated inflammation, molecular mimicry, direct viral neurotropism, cytokine-mediated injury, and unmasking of preexisting neuropathy. These mechanisms are summarized schematically in Figure 1, highlighting the multifactorial nature of COVID-19-associated SFN.

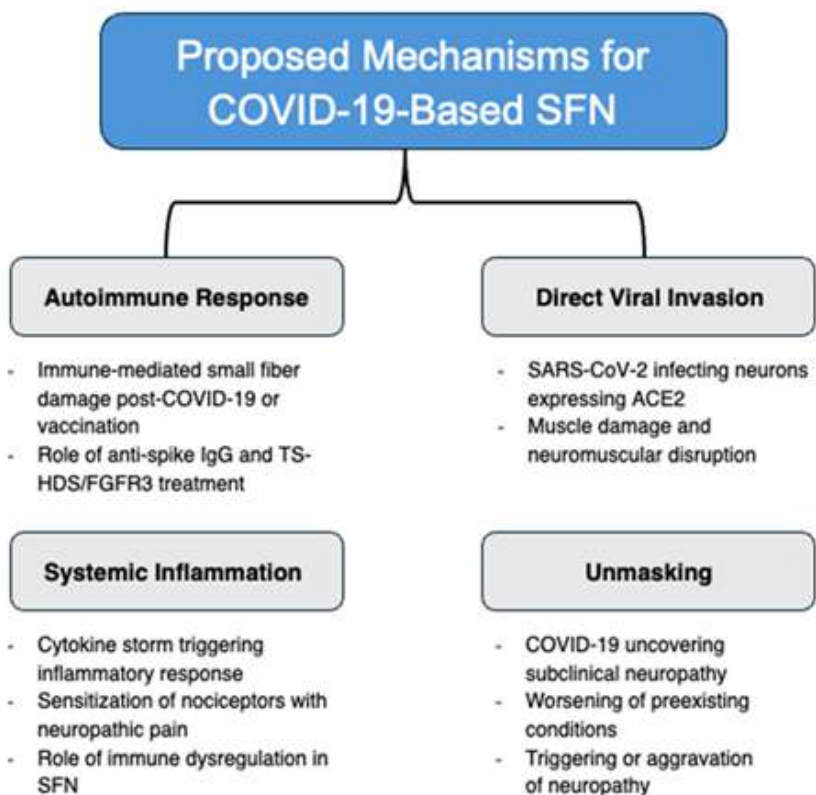


Figure 1. Proposed mechanisms for COVID-19-based SFN: autoimmune response, direct viral invasion, systemic inflammation, and unmasking.

Pharmacologic management primarily targets symptom relief, with gabapentin and antidepressants serving as first-line therapies. Corticosteroids and intravenous immunoglobulin have demonstrated benefit in select immune-mediated cases, while non-pharmacologic interventions remain understudied.

Post-COVID-19 SFN represents a complex, multifactorial condition requiring a multidisciplinary and individualized treatment approach. Further large-scale studies are needed to clarify disease mechanisms, identify predictive biomarkers, and optimize targeted therapeutic strategies to improve long-term outcomes in this growing patient population.

Presenter:
Hana Mucevic
Stony Brook Medical Center, USA



Still's Disease in Pregnancy Complicated by Suspected Anakinra-Related Hepatotoxicity and Successfully Managed with Intravenous Immunoglobulin

**Sara Abushama, Emmanuel Adeleye, Nilay Joshi
Ajanthan Jeyakumar and Shirish Dubey**

Department of Rheumatology, Kettering General Hospital
NHS Foundation Trust, UK

Background: Still's disease (SD) presenting during pregnancy is rare and poses significant diagnostic and therapeutic challenges. Evidence regarding biologic therapies, particularly interleukin-1 (IL-1) blockade, during pregnancy remains limited.

Case: A 34-year-old primigravida developed de novo SD at 27 weeks' gestation with high-spiking fever, evanescent rash, polyarthralgia, neutrophilic leucocytosis, markedly elevated C-reactive protein (342 mg/L), and hyperferritinaemia (27,967 ng/mL). Extensive investigations excluded infection, autoimmune disease, HELLP syndrome, pre-eclampsia, and other pregnancy-related causes. Although her H-score indicated a high probability of haemophagocytic lymphohistiocytosis, she did not fulfil HLH-2004 criteria. She responded initially to intravenous methylprednisolone, oral prednisolone, and anakinra 100 mg daily. One week later, she developed a marked hepatocellular injury (ALT 1138 IU/L). Anakinra was discontinued, with subsequent biochemical improvement. A RU-CAM score of 6 supported a probable drug-induced liver injury. IVIg (0.4 g/kg/day for five days) was administered as rescue therapy, resulting in rapid clinical and biochemical improvement. Caesarean delivery at 34 weeks resulted in a healthy neonate. Postpartum, tocilizumab achieved sustained disease remission.

Conclusion: SD in pregnancy requires prompt multidisciplinary assessment and exclusion of infection, HLH/MAS, and pregnancy-specific complications. Anakinra can provide effective disease control but requires vigilant hepatic monitoring. IVIg represents a potential rescue option when IL-1 blockade is contraindicated or poorly tolerated. Postpartum tocilizumab may provide effective longer-term disease control. Further prospective data are needed to guide treatment and establish maternal–foetal safety.

Presenter:

Sara Hamza Elmubarak Abushama

Kettering General Hospital, NHS Foundation Trust, UK



Herbal Protection against OTA-Induced Immunosuppression in Young Rabbits

K. Zhivkova¹, K. Gospodinova¹, V. Petrov², V. Ivanov³ and S.D. Stoev¹

¹Department of General and Clinical Pathology, Faculty of Veterinary Medicine, Trakia University, Students Campus Bulgaria

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The protective effects of herbal feed additives *Silybum marianum*, *Centella asiatica*, Silymarin and *Withania somnifera* against ochratoxin A (OTA) toxicity were investigated in young New Zealand White rabbits during an experimental period of 80 days. OTA was administered at 2 ppm, whereas *Silybum marianum*, *Centella asiatica*, Silymarin and *Withania somnifera* were used at feed levels 5000 ppm, 4600 ppm, 25000 ppm and 4000 ppm, respectively. All rabbits were immunized at the age of 37 days against Rabbit Hemorrhagic Disease Virus (RHDV) immediately after beginning of the experiment and the immune response was evaluated by measuring the antibody titer using an indirect ELISA technique. A strong immunosuppressive effect of OTA on the humoral immune response against RHDV was observed. The herbal supplements Silymarin and *Withania somnifera* were found to provide reliable protection against OTA-induced immunosuppression. The immunosuppressive effect of OTA caused the development of secondary bacterial infection (pasteurellosis) and the death of two rabbits exposed to OTA only and one rabbit each from the groups protected with *Silybum marianum* and *Centella asiatica*. Body weight was significantly decreased in rabbits exposed to OTA only, but that decrease was slighter in the rabbits, protected with herbal supplements. It was found that the target organs damaged by OTA exposure in rabbits were the liver, kidneys and spleen, except in cases of secondary pasteurellosis, when the strongest damage was found in the lungs. All herbal additives investigated in the present study, especially Silymarin and *W. somnifera*, could be used as a practical method for safely utilising of OTA-contaminated feed, preventing deleterious effects of OTA on health and the immune system, and reducing potential economic losses from possible secondary bacterial infection.

Presenter:

Kalina Vladimirova Zhivkova

Trakia University, Bulgaria



Interplay between the Gut Microbiome, Diet and Clinical Outcomes in Multiple Sclerosis

Simona Rolla

Department of Clinical and Biological Sciences
University of Turin, Italy

Multiple sclerosis (MS) is a chronic immune-mediated neurodegenerative disorder in which environmental factors, particularly diet and gut microbial composition, are increasingly recognized as modulators of disease onset and progression. Emerging metagenomic evidence indicates that, already at diagnosis and subject with MS display alterations in gut microbial composition and diversity compared with healthy individuals. These microbial patterns appear to be associated with relevant clinical and radiological features. At the same time, nutritional factors may influence MS severity through their impact on systemic immunity, oxidative stress, intestinal barrier integrity, and gut-brain axis signaling. In particular, greater adherence to a Mediterranean dietary pattern and higher fiber intake have been associated with lower clinical severity. Overall, these findings suggest that nutritional and microbial profiles may contribute to stratification of patients and to the development of personalized supportive strategies alongside standard therapies.

Presenter:

Simona Rolla

University of Turin, Italy



Antineoplastic and Immunomodulatory Effects of Cabozantinib in Continuous and Primary Human Anaplastic Thyroid Cancer Cells

Elia Giusy¹, Ferrari Silvia Martina¹, Ragusa Francesca¹, Piaggi Simona², Balestri Eugenia¹, Botrini Chiara¹, Rugani Licia¹, Del Carlo Giulia¹, Mazzi Valeria¹, Rizzi Rocco¹, Antonelli Alessandro¹ and Fallahi Poupak²

¹Department of Surgical, Medical and Molecular Pathology and Critical Area, University of Pisa, Italy

²Department of Translational Research and New Technologies in Medicine and Surgery, University of Pisa, Italy

Thyroid cancer (TC) is the most common endocrine malignancy including different histological subtypes. Among these, anaplastic TC (ATC) is the rarest but also the most aggressive form. Anaplastic thyroid cancer is characterized by marked resistance to conventional therapies with a median survival of approximately 6 months. Therefore, new treatments are urgently needed and extremely challenging.

We aim to investigate the antineoplastic and immunomodulatory effect of cabozantinib, a multikinase inhibitor, recently approved (EMA;AIFA) for the treatment of adult patients with locally advanced or metastatic differentiated thyroid carcinoma, not eligible or refractory to radioactive iodine who have progressed during or after prior systemic therapy.

We performed *in vitro* studies in primary human ATC cell cultures, obtained from 6 patients, and in continuous cell lines, 8305C and CAL62. We firstly showed a significant reduction in viability/proliferation, a marked proapoptotic effect and an inhibition of the migration in treated cells compared to controls, in the presence of increasing concentrations of cabozantinib. Moreover, we observed a reduction of CXCL10 and CCL2 (T-helper (Th)1 and (Th)2 chemokines, respectively) production in ATC cells with cabozantinib, suggesting an immunomodulatory effect of the drug.

These findings are highly encouraging and support the future *in vivo* evaluation of cabozantinib in patients with ATC. Moreover, *in vitro* studies may provide an opportunity to assess the chemosensitivity of patient-derived cells, thereby supporting a personalized medicine approach.

In the near future, we aim to further investigate the immunomodulatory effects of cabozantinib by characterizing the tumor microenvironment through the establishment of co-culture models using primary ATC cells and thyroid fibroblast.

Presenter:

Giusy Elia

University of Pisa, Italy



Thyroid Cancer: Role of Neutrophil Extracellular Traps and Neutrophil-Related Mediators

Ragusa Francesca¹, Modestino Luca^{2,3,4}, Cristinziano Leonardo^{2,3}, Poto Remo^{2,3}, Ventrici Annagioia^{2,3,4}, Trocchia Marialuisa^{2,3}, Ferrari Silvia Martina¹, Elia Giusy¹, Fallahi Poupak⁵, Marone Gianni^{2,3,4,6}, Antonelli Alessandro¹, Varricchi Gilda^{2,3,4,6} and Galdiero Maria Rosaria^{2,3,4,5}

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Inflammatory processes and cancers are characterized by neutrophil activation, which can release a broad spectrum of mediators, such as myeloperoxidase (MPO), pentraxin-3 (PTX3), matrix metalloproteinase-9 (MMP-9), and neutrophil extracellular traps (NETs).

Our aim was to investigate the role of NETs and neutrophil-related mediators as potential biomarkers for thyroid cancer (TC).

The study included the following population: 25 healthy controls (HCs), 29 multinodular goiter (MNG) patients, 22 differentiated TC (DTC) patients, and 28 de-differentiated TC (DeDTC) patients.

We evaluated serum levels of circulating free DNA (dsDNA), nucleosomes, citrullinated histone H3 (CitH3), and MPO-DNA complexes as NETs indicators, together with the neutrophil-related mediators MPO, PTX3, MMP-9, and granulocyte-macrophage colony-stimulating factor (GM-CSF).

We observed higher serum levels of all NETs indicators in patients with DeDTC compared to HCs.

In particular, CitH3 serum levels were higher in patients with DeDTC and DTC compared to HCs and patients with MNG; similarly, serum levels of MPO-DNA complexes and nucleosomes were higher in patients with DeDTC compared to HCs and patients with MNG. MPO-DNA complexes levels were also elevated in

patients with DeDTC compared to those with DTC.

Furthermore, MPO serum levels were increased in patients with DeDTC compared to HCs, while serum levels of PTX3, MMP-9, and GM-CSF were higher in patients with DTC and DeDTC compared to HCs.

Finally, patients with metastatic disease at diagnosis exhibited higher serum levels of dsDNA, nucleosomes and MPO-DNA complexes compared to patients without metastases at diagnosis.

Taken together, these findings provide evidence of the neutrophilic inflammation involvement in TC. The increased expression of NETs indicators, particularly in DeDTC and in patients with metastatic disease, suggests that NETs may be linked to tumor malignancy and disease severity.

Presenter:

Francesca Ragusa

University of Pisa, Italy



Innovative Hemodialysis and Public Health Policy Implications in CKD

Andrés Morón Mendoza¹, Raúl Enrique Molina Salazar², José Joaquín Azpiroz Leehan¹ and Itzel Díaz-de-León¹

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²Departamento de Economía, Universidad Autónoma Metropolitana - Unidad Iztapalapa, Mexico

Chronic kidney disease (CKD) represents a growing public health challenge in Mexico, where renal replacement therapy continues to rely predominantly on conventional dialysis strategies. This study evaluated innovative hemodialysis modalities and their potential implications for evidence-based public health policy, comparing high-flux hemodialysis (HDF) with expanded hemodialysis (HDx) in patients with stage 5 CKD.

A mixed-method approach combined prospective clinical measurements with qualitative analysis of health-policy and administrative feasibility. Clinical data from 20 men and women aged 20-75 years receiving maintenance hemodialysis were analyzed before and after treatment. Patients received high-flux hemodialysis during sessions 1-12 and expanded hemodialysis during sessions 13-24. Biochemical outcomes included serum phosphorus, urea, and beta-2 microglobulin, while interviews with experts in hospital management, administrative sciences, and public policy explored barriers and criteria for adoption within the Mexican public health system.

High-flux hemodialysis produced a statistically significant initial reduction in serum phosphorus ($p < 0.0001$) and was effective for removal of small uremic solutes. During the subsequent HDx phase, phosphorus remained metabolically stable without significant additional differences between sessions 12 and 24. HDx showed a significant reduction in beta-2 microglobulin and greater efficiency in the clearance of middle molecules associated with systemic inflammatory processes. Expert analysis identified the need for clinical effectiveness and safety evaluation, cost-benefit assessment, administrative planning, infrastructure compatibility, professional training, and monitoring indicators before large-scale implementation.

These findings suggest that HDF and HDx provide complementary clinical benefits. Incorporating innovative hemodialysis into public policy should therefore

combine therapeutic evidence with economic, organizational, regulatory, and equity considerations to support sustainable renal-care strategies in Mexico.

Presenter:

Andrés Morón Mendoza

Centro Nacional de Investigación en Imagenología e Instrumentación Médica (CI3M), Universidad Autónoma Metropolitana - Unidad Iztapalapa, Mexico



Single-Base Polymorphism of the ACE2 and TNF-Alpha Genes in Patients with the Cardiac and Indeterminate Forms of Chagas Disease

Cilmary Suemi Kurokawa¹, Bruna da Cruz Carvalho¹, Daniela Filadelfo Sanches¹, Rodrigo Mattos Dos Santos¹, Erika Alessandra Pellison Nunes da Costa¹ and Simone Baldini Lucheis²

¹Medical School of Botucatu, UNESP, Brazil

²Veterinary Medical School of Botucatu, UNESP, Brazil

Introduction: Chagas disease (CD) is caused by the protozoan *Trypanosoma cruzi*, which is endemic in 21 Latin American countries and has also been reported in North America and continental Europe. It is estimated that 6 to 7 million people in Latin America are infected. The disease presents two clinical phases: acute and chronic. The chronic phase may be asymptomatic (indeterminate form) or symptomatic, including cardiac, digestive, or mixed forms, depending on clinical manifestations. Few studies have sought to clarify why some infected individuals remain asymptomatic, whereas others develop more severe forms of the disease.

Aims: To evaluate and correlate the frequency of single nucleotide polymorphisms (SNPs) in the *ACE2* and *TNF- α* genes in patients with chronic cardiac and indeterminate forms of CD, and to associate these findings with clinical data and comorbidities.

Methods: The study included male patients with chronic indeterminate (n = 51) and cardiac (n = 22) forms of CD, followed at the outpatient clinic of the Hospital das Clínicas, Botucatu Medical School (HC-FMB). Clinical data were extracted from electronic medical records, and SNP analysis was performed on nucleated peripheral blood cells using Sanger sequencing and restriction fragment length polymorphism (RFLP) methods.

Results: Patients had a mean age of 68 and 70 years in the indeterminate and cardiac groups, respectively, and were classified as elderly. Essential hypertension was the most frequent comorbidity among those evaluated. Analysis of the *ACE2* gene SNP rs2074192 showed no significant difference in the frequencies of the guanine (G) and adenine (A) alleles. Similarly, analysis of the *TNF- α* gene SNP rs1800629 revealed no significant differences in the frequencies of the GG, GA, and AA genotypes.

Conclusion: No significant differences were observed in the frequency of polymorphisms rs2074192 and rs1800629 between the clinical forms of Chagas disease.

Presenter:

Cilmery Suemi Kurokawa

Medical School of Botucatu, UNESP, Brazil



Alternative Root Canal Irrigation Solutions and Non-Cytotoxic High Antibacterial Effectiveness

Tahir ATAÖZDEN¹, Semanur Özüdoğru², Muhammet Yayla³ and Mustafa Çoşkun³

¹Biruni University, Türkiye

²Gelişim University, Türkiye

³Kafkas University, Türkiye

Aim: This *in vitro* laboratory study aimed to evaluate the cytotoxicity and antibacterial effectiveness of alternative root canal irrigation solutions, including N-acetylcysteine (NAC), boric acid, and chitosan, at different concentrations using L929 mouse fibroblast cells and *Q. aureus* biofilms.

Methods: Cytotoxicity was evaluated using cell culture tests. Chitosan was prepared at 10 concentrations ranging from 2048 to 4 µg/mL, NAC at 8 concentrations ranging from 50 to 0.39 mg/mL, boric acid at 10 concentrations ranging from 64 to 0.125 mg/mL, and sodium hypochlorite (NaOCl) at 10.5%, 5.25%, and 2.625%. Antimicrobial testing was performed using chitosan (1–0.002 mg/mL), NAC (25–0.195 mg/mL), and boric acid (32–0.0625 mg/mL). Prepared microplates were incubated at 37°C for 18 hours, and intra- and intergroup comparisons were performed.

Results: NaOCl showed the strongest antimicrobial activity across the tested concentrations and time intervals but was also the most cytotoxic. Chitosan demonstrated an acute toxic effect beginning at 128 µg/mL. The MIC value of *Q. aureus* for chitosan was 0.031 mg/mL. NAC showed an MIC of 1.563 mg/mL; toxicity was observed at 25–50 mg/mL during the first 24 hours, while the antimicrobial threshold was not associated with cytotoxicity. Boric acid showed an MIC of 4 mg/mL and no cytotoxic effect during the first 24 hours, although toxicity increased over time.

Conclusion: Among the tested alternatives, NAC and boric acid demonstrated more favorable antimicrobial-dose/cytotoxicity profiles than the other experimental groups, whereas NaOCl provided the highest antimicrobial effectiveness but the greatest cytotoxicity.

Presenter:

Tahir ATAÖZDEN

Biruni University, Türkiye

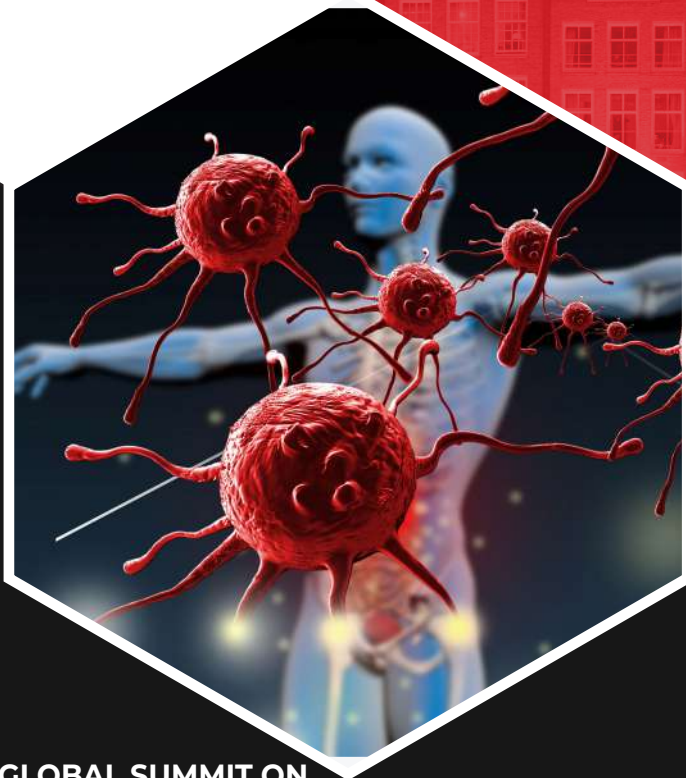
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SPEAKER TALKS



Mast Cell Activation Disease as a Pathophysiological Driver of Long COVID

Anne Maitland

Department of Medicine – Rheumatology Charleston
Medical University of South Carolina Ehlers Danlos Syndrome
(EDS) Center, USA

The introduction of SARS-CoV-2 to the human population has resulted in a staggering global health crisis, with over 775 million confirmed cases. For a significant and growing percentage of survivors—estimated at 15% per individual per infection—the acute phase is followed by a complex, multisystem chronic condition known as Long COVID, which is also known as Post Acute Sequelae of SARS-CoV-2 (PASC). This condition is characterized by persistent immune overactivity, systemic inflammation, and significant functional impairment lasting for at least three months.

Central to understanding this pathology is the disruption of the epithelial barrier and the subsequent dysregulation of the nerve-mast cell interactome. Mast cells (MCs) serve as the body's strategic sentinels, residing at host-environment interfaces and clustering peri-vascularly near nerve endings to survey tissue integrity. While a robust innate immune response is essential to recognize and clear the novel pathogen and initiate tissue repair, an ineffective or unnecessary immune activation leads to the chronic "Goldilocks" failure where the response is "too hot," resulting in ongoing tissue injury. This neuro-immune struggle manifests as an array of multi-organ symptoms, including neuropsychiatric dysfunction ("brain fog," headaches), gastrointestinal distress, and interconnected dysautonomia, such as Postural Orthostatic Tachycardia Syndrome (POTS).

To address this unmet clinical burden, this chapter proposes utilizing the consensus diagnostic criteria for Mast Cell Activation Syndrome (MCAS) to assess Long COVID patients. By documenting multisystem mediator-related symptoms and biomarker elevations, and characterizing MCAS phenotypes—distinguishing between primary (clonal) and secondary (non-clonal/extrinsic) endotypes— as well as disorders that mimic MCAS, such as dysautonomia and joint hypermobility syndromes, clinicians can move toward a new paradigm of personalized medicine. Targeted therapies directed at the disturbed mast cell-nerve axis, including mast cell stabilizers, mediator blockades, and epithelial barrier restoration, offer a strategic pathway to quench the sentinel's storm and restore homeostasis.

Presenter:

Anne Maitland

Medical University of South Carolina Ehlers Danlos Syndrome (EDS) Center, USA



Lysosomal Cysteine Proteases in Disease Modulation: Insights from Cancer and Pain Research

Yogeeswari Perumal and **Madhu Babu Battu**

Department of Pharmacy, Birla Institute of Technology and Science-Pilani, Hyderabad Campus, India

Objectives: This study aims to investigate Cathepsin S (CatS), a lysosomal cysteine protease with unique activity at neutral pH, as a viable therapeutic target in cancer and neuropathic pain. The primary objective is to design and evaluate selective small-molecule inhibitors that can effectively modulate CatS activity. Additionally, the work seeks to establish the dual therapeutic relevance of these inhibitors in glioblastoma and chronic pain conditions.

Scope: The scope of this research encompasses the exploration of CatS biology in both intracellular and extracellular environments, particularly its role in tumor progression and neuropathic pain pathways. The study integrates computational drug design, cellular validation, and animal model evaluation to ensure translational applicability, focusing on glioblastoma as a model cancer and neuropathic pain as a chronic condition.

Methods Used: A multiple pharmacophore modeling approach was employed to rationally design CatS inhibitors. These compounds were subjected to *in-vitro* antiproliferative assays using glioblastoma cell lines, alongside CatS expression analysis and pathway studies to confirm mechanism of action. Further, *in-vivo* evaluation was conducted in animal models of neuropathic pain to assess antihyperalgesic and antiallodynic effects.

Results: The designed ligands demonstrated significant antiproliferative activity against glioblastoma cells and effectively modulated CatS activity as confirmed through biochemical and pathway analyses. *In-vivo* studies revealed substantial attenuation of neuropathic pain symptoms, highlighting both antihyperalgesic and antiallodynic properties. These findings underscore the dual therapeutic efficacy of the developed compounds.

Conclusion: The study validates CatS as a compelling therapeutic target and presents novel inhibitors with strong potential in both oncology and pain management. The integrated approach establishes a foundation for future drug development targeting lysosomal proteases, offering promising avenues to address unmet medical needs in cancer and chronic pain.

Presenter:

Yogeeswari Perumal

Department of Pharmacy

Birla Institute of Technology and Science-Pilani, Hyderabad Campus, India



Infection Mitigation by Early Immune Activation

Jan J.M. Cuppen and **Huub J.F. Savelkoul**

Wageningen University, The Netherlands

Many bacterial and viral pathogens have evolved mechanisms to delay the host immune response, enabling early-stage proliferation and disease progression. This immune evasion often targets the innate immune system, particularly during the critical initial phase of infection.

We have developed a technology that activates neutrophils, thereby enhancing and accelerating the innate immune response. In animal models, this approach reduced mortality and tissue damage while improving overall vitality during infection. *In vitro* and *ex vivo* studies demonstrated significantly enhanced immune function. Furthermore, a study in healthy human volunteers showed increased neutrophil activation following a 30-minute treatment.

The intervention consists of a 30-minute exposure to a specific electromagnetic field (EMF) stimulus. The EMF operates well within established safety guidelines and is imperceptible to users. Its intensity is comparable to that of stray electromagnetic fields from common household devices, but it employs a distinct signal waveform. The treatment is delivered by a simple, low-cost, battery-powered device suitable for home use. Scalable versions for mass deployment (e.g., in public venues during pandemics) are also available.

The next step is a clinical study in patients with recurrent cystitis (urinary tract infections, UTI) to evaluate whether early immune activation can mitigate symptoms and substantially reduce antibiotic use, thereby contributing to the reduction of antimicrobial resistance (AMR).

Given that immune delay is also a feature of infections such as influenza and coronaviruses, this technology may offer a broadly applicable strategy to reduce disease burden and societal disruption in future pandemics. Wider independent validation will be essential, and we welcome collaboration with other research groups.

Presenter:

Jan Cuppen

Wageningen University, The Netherlands





Beta-Glucan Supplementation Improves Clinical Outcomes and Preserves Immune Homeostasis in Patients with Advanced Solid Tumors Receiving Chemotherapy: A Prospective Phase II Randomized Three-Arm Clinical Trial

Wen-Chi Yang¹ and Ming-Shyan Huang²

¹School of Medicine, College of Medicine
National Sun Yat-Sen University, Taiwan

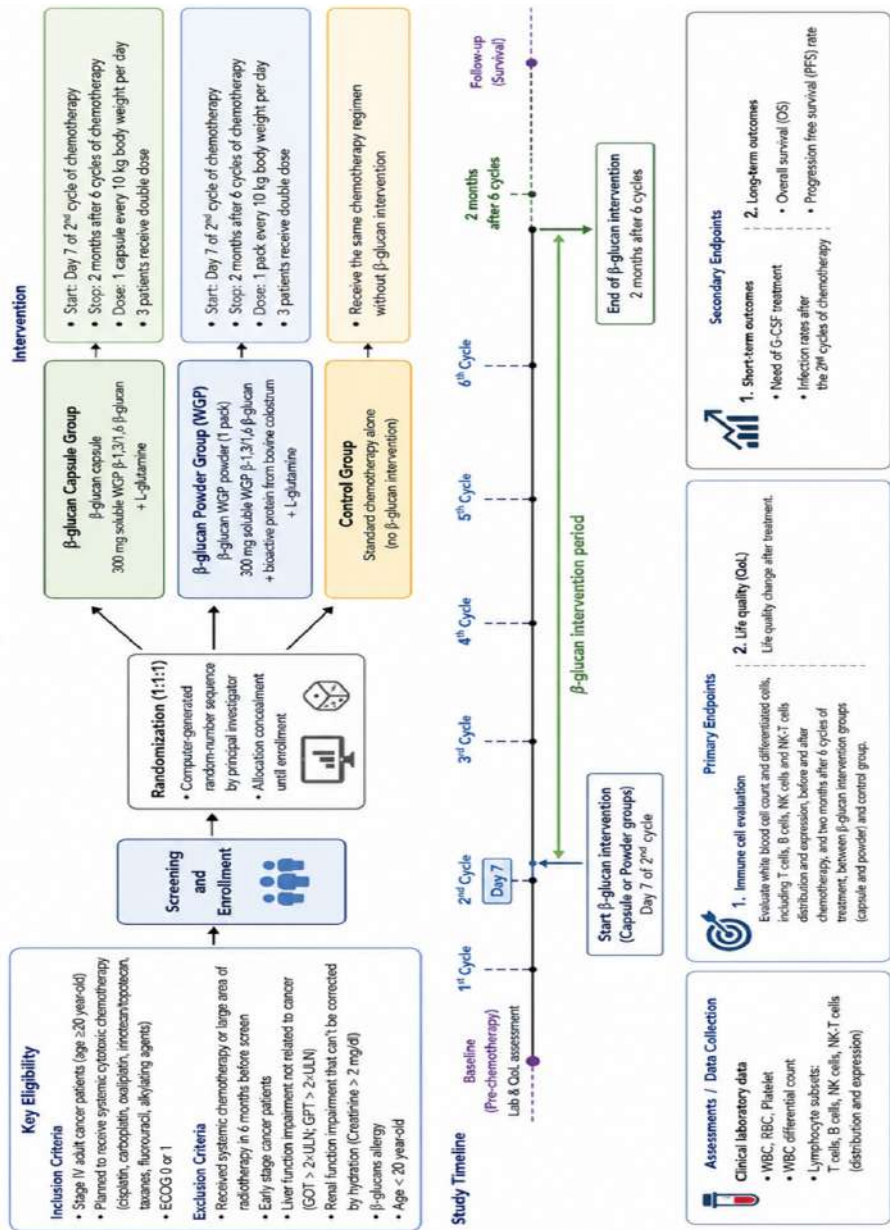
²Department of Internal Medicine, E-DA Cancer Hospital
Taiwan

Background: Chemotherapy-induced immune dysregulation remains a major challenge in patients with advanced solid tumors. Although β -glucan has immunomodulatory properties, its longitudinal effects on immune homeostasis during chemotherapy remain unclear.

Methods: In this prospective, randomized phase II study, patients receiving chemotherapy were assigned to β -glucan powder with L-glutamine and bio-active protein from bovine colostrum, β -glucan capsules with L-glutamine, or standard care. Clinical outcomes, serial hematologic parameters, and longitudinal profiles of T-cell, natural killer (NK)-cell, natural killer T (NKT)-cell, and killer-cell immunoglobulin-like receptor (KIR; CD158)-expressing populations were evaluated. Direct biological activity was assessed using an *in vitro* Jurkat T-cell viability assay.

Results: Compared with standard care, β -glucan supplementation was associated with a higher disease control rate, faster neutrophil recovery, and greater stability of the neutrophil-to-lymphocyte ratio during chemotherapy. Longitudinal immune profiling demonstrated greater stability of T-cell, NK-cell, NKT-cell, and KIR-expressing populations in the β -glucan groups, suggesting attenuation of chemotherapy-associated immune remodeling. Both formulations showed broadly comparable immunomodulatory effects, although CD158b(+) and CD158i(+) NK-cell subsets remained more stable in the powder group during later treatment cycles. *In vitro*, β -glucan significantly reduced Jurkat T-cell viability, supporting direct biological activity.

Conclusions: β -Glucan supplementation may help preserve immune homeostasis during chemotherapy while supporting neutrophil recovery and disease control in patients with advanced solid tumors. These findings support its potential role as an adjunctive immunonutritional strategy and warrant confirmation in larger studies.



Presenter:
Wen-Chi Yang
National Sun Yat-Sen University, Taiwan



Oxidative Stress-Induced Expression Levels of *PXDN* and *NF-κB* in Type 2 Diabetic Patients with Nephropathy

Dalia A. Gaber¹, Zakira Hanin², Aisha Anees Ahmed², Shuhd Salem Al Nahdi², Sumayya Althaf Kasim², Mahir Khalil Jallo³ and Rajaram Rhambau Jagdale³

¹Department of Biomedical Sciences, College of Medicine
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Background: Diabetic nephropathy (DN) is the leading cause of end-stage renal disease in patients with diabetes mellitus (DM). Early detection remains difficult, but essential for timely management. In this study, we assessed Peroxidase (PXD) gene expression as a biomarker for early detection of DN and for reflection of oxidative stress status. We also compared *PXDN* and Nuclear factor κ B (*NF-κB*) expression levels in DN patients, aiming to identify a reliable diagnostic biomarker.

Methods: This prospective cross-sectional study included 40 patients with T2DM with and without nephropathy, and 20 matched healthy controls. *PXDN* mRNA expression was quantified by real-time polymerase chain reaction (qRT-PCR). Biochemical analyses of malondialdehyde (MDA) and total antioxidant capacity (TAC) were also done to assess the oxidative stress status. Laboratory parameters, including glycated hemoglobin, urinary albumin-to-creatinine ratio, estimated glomerular filtration rate, and microalbuminuria, were measured and correlated with gene expression levels and the oxidative stress status.

Results: *PXDN* mRNA expression was significantly upregulated in patients with DN compared to diabetic cases without nephropathy and control subjects. MDA levels were significantly elevated in both diabetic groups relative to controls, but no significant difference was found between DN and DM without renal impairment. TAC was significantly lower in DN cases compared to DM and controls. The *NF-κB* gene was upregulated in both diabetic groups compared to control subjects but no significant difference between DN and DM without nephropathy.

Furthermore, *PXDN* expression did not correlate with MDA, TAC, or *NF-κB* levels. Receiver operating characteristic (ROC) analysis identified *PXDN* as a highly specific and sensitive marker for DN, outperforming conventional oxidative stress biomarkers in distinguishing DN from both diabetic and healthy individuals.

Conclusion: *PXDN* is significantly upregulated in DN and may serve as a sensitive and specific molecular marker for early detection of nephropathy compared to *NF-κB*.

Presenter:

Dalia Ali Gaber

Gulf Medical University, UAE



Carcinoembryonic Antigen Beyond Cancer: Exploring its Associations with Glycaemic Dysregulation, Lipid Profile, and Vitamin D in Diabetes

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¹Gulf Medical University (GMU), UAE

²Theodor Bilharz Research Institute (TBRI), Egypt

Background: Diabetes mellitus is associated with a high prevalence of chronic metabolic dysregulation, dyslipidaemia, and vitamin D deficiency. Carcinoembryonic antigen (CEA) mild elevation has been reported in diabetic patients. The mechanisms and clinical significance of this elevation, and its relationship to lipid profile and vitamin D status, remain incompletely understood, particularly in Gulf region populations.

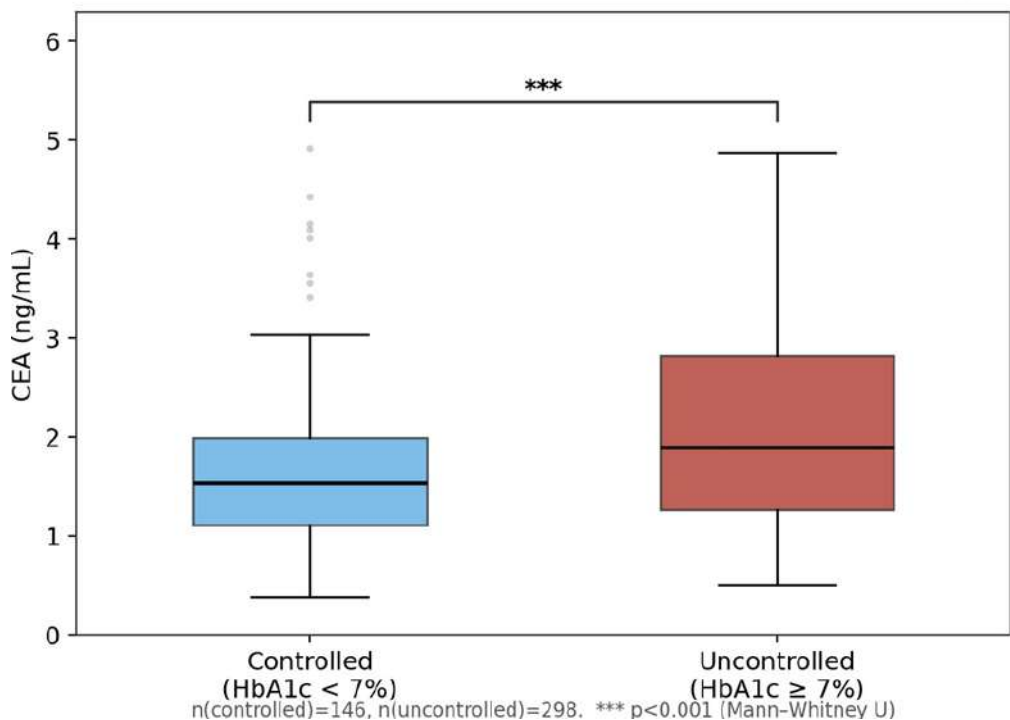
Objectives: This study aimed to compare serum CEA levels between diabetic and non-diabetic subjects attending Thumbay Hospital, UAE, and to examine its relationship with glycaemic control, lipid profile, and 25-hydroxyvitamin D [25(OH)D] status.

Materials & Methods: A retrospective cross-sectional study was conducted using laboratory records of 3,118 adult patients (1,719 non-diabetic, 955 pre-diabetic, and 444 diabetic).

Results: Median serum CEA increased progressively across the glycaemic groups, from 1.15 ng/mL in non-diabetic subjects to 1.32 ng/mL in pre-diabetic subjects and 1.75 ng/mL in diabetic subjects ($p < 0.001$). Within the diabetic group, CEA was significantly higher in patients with uncontrolled diabetes than in those with controlled diabetes (1.90 vs 1.54 ng/mL, $p < 0.001$), and CEA correlated positively with HbA1c and fasting glucose. Lipid parameters correlated weakly with CEA in the non-diabetic group but showed no association in the diabetic group. No statistically significant differences were observed between CEA and vitamin D status, despite a high prevalence of vitamin D deficiency or insufficiency across all three groups (69.2% non-diabetic; 69.3% pre-diabetic; 60.6% diabetic). CEA was consistently higher in males than females across all groups.

Conclusion: Serum CEA is significantly and progressively elevated with worsening glycaemic status among the study population and should be interpreted in the context of glycaemic control rather than co-existing dyslipidaemia or vitamin D deficiency. These findings, the first of their kind from the UAE, support cautious interpretation of mildly elevated CEA in diabetic patients before pursuing further investigation for malignancy.

CEA by Glycaemic Control Within the Diabetic Group



Presenter:
Noha Kamal Abdelmomen
Gulf Medical University, UAE
Theodor Bilharz Research Institute (TBRI), Egypt



Innate Immunity in Bronchopulmonary Dysplasia: The Role of the IL-1 Cytokine Family and the IL-1 β –IL-8 Axis

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Larisa Janžič¹ and Štefan Grosek^{3,4}

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⁴Department of Medical Ethics, Faculty of Medicine, University of Ljubljana, Slovenia

Objective: Bronchopulmonary dysplasia (BPD) is a major chronic lung disease of extremely preterm infants, characterized by impaired alveolar and vascular development. This work aims to examine the role of innate immunity in BPD, with particular emphasis on the IL-1 cytokine family and the IL-1 β –IL-8 (CXCL8) axis, and to relate current evidence to our ongoing prospective study in extremely preterm infants.

Methods: Evidence from human and experimental studies on innate immune mechanisms in BPD was evaluated, focusing on IL-1 β , IL-8/CXCL8 and other members of the IL-1 cytokine family. In parallel, our ongoing prospective study evaluates longitudinal blood profiles of IL-1 family members and IL-8 in extremely preterm infants in relation to BPD development and severity, calculated BPD risk, neutrophil involvement, and the need for postnatal glucocorticoid treatment.

Results: Current evidence indicates that damage- and pathogen-associated molecular patterns activate pattern-recognition receptors, NF- κ B signaling, and inflammasomes in the immature lung. IL-1 β acts as an upstream inflammatory mediator inducing IL-8 expression, while IL-8 promotes neutrophil recruitment and activation. Persistent neutrophilic inflammation contributes to oxidative stress, protease release, and epithelial and endothelial injury, thereby interfering with alveolarization and pulmonary vascular development. Increased IL-1 β and IL-8 levels are consistently associated with BPD development and severity. Other IL-1 family members may further shape these responses: IL-1 receptor antagonist (IL-1Ra) counterbalances IL-1-mediated signaling, whereas IL-18 and IL-33 contribute to inflammasome-associated and tissue-derived immune responses.

Conclusion: The IL-1 cytokine family, particularly the IL-1 β -IL-8 axis, represents an important interface between inflammatory injury and disrupted lung development in BPD. Defining the temporal relationships among these mediators may improve biomarker-based risk stratification and support more precisely targeted anti-inflammatory strategies while preserving antimicrobial defence and tissue-repair mechanisms.

Presenter:

Andreja Nataša Kopitar

University of Ljubljana, Slovenia



Development and Characterization of a Mouse Model of Hepatocellular Carcinoma for the Evaluation of Novel Immunotherapeutic Approaches

Ushakova E.I.¹, Gladkaya A.N.¹, Gurskiy D.A.¹
Pichugin A.V.¹, Shilovskiyy I.P.¹ and Khaitov M.R.^{1, 2}

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²Pirogov Russian National Research Medical University of the
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University), Russian Federation

Background: Hepatocellular carcinoma (HCC) is characterized by high mortality and a profoundly immunosuppressive tumor microenvironment (TME). Immunologically relevant *in vivo* models are essential for investigating tumor-immune interactions and evaluating novel immunotherapeutic strategies. This study aimed to develop and characterize a reproducible murine HCC model suitable for preclinical assessment of immunomodulatory therapies.

Methods: C3H mice were subcutaneously inoculated with 2.5×10^5 MH-22a murine HCC cells. Tumor growth and survival were monitored, and primary tumors were surgically resected on day 19. The immune composition of tumors and tumor-draining lymph nodes was characterized by flow cytometry. Metastatic tumor cells in different tissues were assessed using a limiting dilution assay. The model was subsequently used to evaluate combined TLR3/TLR4 activation with Poly(I:C) (5 µg/mouse) and Immunomax® (15 µg/mouse), administered intraperitoneally every 2–3 days for a total of seven injections. Antitumor efficacy was compared with standard therapy using anti-VEGF (5 mg/kg) and anti-PD-L1 (10 mg/kg) antibodies.

Results: MH-22a inoculation resulted in solid tumor formation in 100% of mice. Mortality associated with progressive tumor growth began 35–40 days after inoculation. No spontaneous metastases were detected in the lungs or lymph nodes. The TME was strongly myeloid-dominated (~75%), comprising tumor-associated macrophages, dendritic cells, myeloid-derived suppressor cells, and monocytes, with low representation of activated T cells. Combined TLR3/TLR4 stimulation markedly inhibited tumor growth. On day 26, mean tumor volume was 474.6 mm³ compared with approximately 1,000 mm³ in untreated controls and 288.3 mm³ following anti-VEGF/anti-PD-L1 therapy. TLR-based immunotherapy was accompanied by increased accumulation of effector and memory T cells in tumors and regional lymph nodes.

Conclusions: The MH-22a model combines reproducible tumor growth with a myeloid-dominated immunosuppressive TME and absence of spontaneous metastasis. It represents a useful platform for evaluating immunotherapies targeting the tumor immune microenvironment. Combined TLR3/TLR4 activation inhibited tumor growth and promoted local and regional T-cell responses.

Funding: The study was supported by State assignment for 2026-2028 (agreement No. 388-03-2026-078 dated 28.01.2026 with Federal Medical-Biological Agency).

Presenter:

Ekaterina Igorevna Ushakova

Allergy and Immunology Research Centre, FMBA of Russia, Russian Federation



The Acari Hypothesis

Andrew C. Retzinger¹ and **Gregory S. Retzinger²**

¹Camden Clark Medical Center, West Virginia University, USA

²Feinberg School of Medicine, Northwestern University, USA

The global prevalence of allergic disease has increased dramatically over the past century, yet its pathogenesis and the biological principles that determine why certain molecules provoke allergic responses remain poorly understood. The Acari Hypothesis (I–VIII) proposes a unified evolutionary and immunological framework for the origin, function, and dysregulation of allergic immunity. Across eight interconnected manuscripts, this framework argues that IgE mediated allergic responses evolved primarily as a defense against acarian ectoparasites, and that IgE functions as an anti-vector antibody class that detects biological signals derived from the digestive tract of these parasites.

Within this model, mammalian exocrine glands are interpreted as components of an integrated defensive system that supports protective microbiota. In humans, the microenvironments generated by eccrine, sebaceous, apocrine, and mammary glands foster microbial mutualists capable of producing metabolites and immune signals that are proposed to help detect and deter acarian parasites, particularly dust mites (Pyroglyphidae). Modern behaviors, including the hygienic disruption of integumentary gland secretions, widespread antibiotic use, and replacement of maternal milk with formula, may disrupt these glandular ecosystems or the microbial communities they support and thereby increase vulnerability to acarian ectoparasitism.

This framework provides mechanistic explanations for several epidemiological patterns recognized by clinicians, including the modern global rise in allergic disease, the association between hygiene and allergy, and the predominance of allergic disease in childhood. Importantly, the hypothesis addresses a central question in allergy biology: what determines the allergic potential of a molecule or molecular family? The model proposes two principal classes of allergens. The first is a functional class consisting of molecules derived from the acarian digestive tract that become associated with microbes transferred by the parasite. The second is a dysfunctional class consisting of acarian dietary components inadvertently transferred alongside pathogens.

By integrating ecological, evolutionary, and immunological perspectives, the Acari Hypothesis provides a coherent framework for interpreting allergic disease. In this view, allergic pathology represents the misfiring or maladaptation of an evolved anti-vector defense system that becomes dysregulated when

ecological relationships among hosts, microbes, and parasites are disrupted by modern environmental conditions.

Presenter:

Andrew C. Retzinger

West Virginia University, USA



Autophagy Maintains High Endothelial Venule Identity and Function during Inflammation

Kathryn Jacobs^{1,2,3}, Yichao Hua^{1,3}, Apostolos Nikaolakopoulos^{2,3,4}, Swati Gupta^{1,2,3,4}, Gerlanda Vella^{1,3}, Alpasan Tasdogan⁵, Colinda Scheele^{3,4}, Patrizia Agostinis^{2,4} and Gabriele Bergers^{1,3}

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⁵Department of Dermatology, University Hospital Essen & German Cancer Consortium, Germany

Lymph nodes (LNs) sequester antigens to prime immune cells and mount immunological responses. High endothelial venules (HEVs) in LNs play a crucial role in extracting naïve lymphocytes from the blood stream into the LN where they get primed and educated by antigen-presenting cells. To do so, HEVs display high levels of specifically-modified lymphocyte adhesion molecules, referred to as peripheral node addressins (PNAd). Upon inflammation, HEVs expand and upregulate additional leukocyte adhesion molecules, reminiscent of inflamed endothelial cells, to accommodate a proper T cell influx and immune response. Here, we present an unexpected and pivotal role of autophagy in LN HEV function and maintenance during inflammation. Using mouse models of inflammation, we observed that genetic deletion of essential autophagy genes (ATG12 /ATG7) in HEVs led to reduced levels of lymphocyte adhesion molecules and via intravital microscopy, a loss of the typical HEV “plump” morphology, accompanied by reduced recruitment of lymphocytes to the LN. Single-cell transcriptomics and Proteomics revealed reduced expression and activity of key HEV signaling pathways, including the LTbR-RelB axis, which diminished the expression of HEV-specific enzymes and molecules necessary to sustain an HEV phenotype. Autophagy loss was also associated with reduction in ER related stress responses, which have been directly linked to PNAd production. Blocking autophagy or LTbR signaling in HEVs in a mouse model of psoriasis lead to less water loss in the skin, as well as reduced HEV formation, cytokine presence, and immune cell infiltration. Together these data underline that autophagy is an essential pathway for maintaining HEV identity and function under inflammatory stress.

Presenter:

Kathryn Jacobs

KU Leuven, Belgium



***In situ* Reprogramming of Myeloid Cells with TLR Agonists in an Experimental Model of Triple-Negative Breast Cancer**

Ushakova E. I., Pichugin A.V., Lebedeva E.S. and Ataulakhonov R.I.

Allergy and Immunology Research Centre, FMBA of Russia
Russian Federation

Background: The immunosuppressive tumor microenvironment is shaped by both malignant and nonmalignant cells, as well as the substances they produce. Myeloid cells – including tumor-associated macrophages, intratumoral dendritic cells (DCs), and tumor-recruited myeloid-derived suppressor cells – play a key role in establishing this immunosuppressive milieu. However, activation of macrophages and DCs with TLR3, TLR4, or a combination of these agonists confers cytotoxic properties against tumor cells *in vitro*.

Material and methods: Solid tumors were surgically resected 11 days after subcutaneous inoculation of 15,000 4T1 cells into BALB/c mice. Immunotherapy was administered using TLR3 and TLR4 agonists. Metastatic burden was quantified by limiting dilution assay. Naïve mice were injected with TLR agonists, and lung macrophages and DCs were sorted. Antitumor activity of lung myeloid cells against 4T1 targets was assessed using a cytotoxicity assay with FACS-sorted CD45⁺CD11c⁺F4/80⁺Ly6C⁺Ly6G⁺I-A/I-E⁺ DCs and CD45⁺CD11b⁺CD11c⁺F4/80⁺Ly6C⁺Ly6G⁺I-A/I-E⁺ macrophages.

Results: TLR agonists induce systemic reprogramming of macrophages and DCs throughout the body, including in tissues and organs harboring metastatic cells. *In vitro* co-culture of 4T1-GFP carcinoma cells with TLR-activated CD11c⁺ DCs or F4/80⁺ macrophages significantly suppressed tumor cell viability and proliferation starting at a 3:1 effector-to-target ratio. *In vivo* agonist administration in naïve mice activated DCs and macrophages into an antitumor state, with pronounced cytotoxicity observed for DCs at a 25:1 ratio and for macrophages at a 50:1 ratio. Pulmonary myeloid cells from naïve mice lacked such activity. Concurrently, the emergence of cytotoxic macrophages and DCs in lung tissue correlated with a marked reduction in metastatic colony-forming units in tumor-bearing mice, some mice showed complete clearance of metastases.

Conclusion: TLR3 and TLR4 agonist injections reprogram tissue macrophages and DCs in the lungs, transforming them into potent killers of metastatic tumor cells.

Presenter:

Ekaterina Igorevna Ushakova

Allergy and Immunology Research Centre, FMBA of Russia, Russian Federation



Risky Behaviours and Students Academic Life in Higher Education Institutions in Tanzania

Rose Ephraim Matete¹ and Ombeni William Msuya²

¹Seniour Lecturer, University of Dodoma, College of Education, Tanzania

²The University of Dodoma, Tanzania

Risky behaviours remain a widespread global challenge affecting both developed and developing nations. This study explored the risky behaviours and students' academic life in Higher Education Institutions (HEIs) in Tanzania. Employing a qualitative research approach and exploratory case study design, data were gathered through open-ended questionnaires, interviews, and documentary review. A total of 97 participants were involved in the study, comprising 94 undergraduate students and three (3) hostel wardens from a selected university. The findings reveal that students engaged in behaviours such as maintaining multiple sexual partnerships, excessive alcohol consumption, and unprotected sexual practices, which increased their vulnerability to Sexually Transmitted Diseases (STDs) and negatively affected their academic performance. Among these behaviours, having multiple sexual partners emerged as the most prevalent and concerning. It was also found that female students were particularly affected, facing challenges, such as early pregnancies, carry-over courses, and poor performance in University Examinations (UEs). The key argument in this study is that for HEIs to protect students from risky behaviours, there is a need of reinforcing the orientation programmes, strengthening Guidance and Counselling (G&C) services, and implement gender-relations training to help students manage academic and social pressures during and after university life. Nevertheless, strict enforcement of institutional rules and regulations, along with the provision of comprehensive Human Immunodeficiency Virus (HIV)/ Acquired Immunodeficiency Syndrome (AIDS) and sexually transmitted infections (STIs) prevention programmes, is essential to safeguarding students' well-being and academic success.

Presenter:

Rose Ephraim Matete

Seniour Lecturer, University of Dodoma, College of Education, Tanzania



Modulating Tumor Mechanics and Vascular Barriers to Enhance Chemotherapy Efficacy in Pancreatic Tumors and Melanoma

Stella Angeli¹, Constantina Neophytou¹, Antonia Charalambous², Chrysovalantis Voutouri², Triantafyllos Stylianopoulos² and Fotios Mpekris¹

¹Cancer Genetics, Therapeutics & Ultrastructural Pathology Department, The Cyprus Institute of Neurology & Genetics Cyprus

²Department of Mechanical and Manufacturing Engineering, Cancer Biophysics Laboratory, University of Cyprus, Cyprus

Background: Pancreatic ductal adenocarcinoma (PDAC) is characterized by a dense, fibrotic stroma and dysfunctional vasculature that impair drug delivery and immune cell infiltration. Melanoma, although less fibrotic, remains highly metastatic and resistant to therapy. Strategies that normalize the tumor microenvironment (TME) may enhance therapeutic efficacy across these tumor types.

Objective: To evaluate whether a combinatorial approach using the antihistamine Ketotifen and ultrasound-mediated sonopermeation can remodel the TME and improve chemotherapy response in murine models of PDAC and melanoma.

Methods: Orthotopic murine models of PDAC and melanoma were treated with a combination of ketotifen, ultrasound-mediated sonopermeation, and chemotherapy. Tumor growth, vascular function, perfusion, hypoxia, stiffness, and immune cell infiltration were assessed. Cytokine profiling and machine learning analyses were performed to characterize immune responses and identify predictors of therapeutic outcome.

Results: The triple therapy significantly suppressed tumor growth in both models, with more pronounced effects in PDAC. In PDAC tumors, treatment reduced stiffness, restored vascular function, improved perfusion (Figure 1), and alleviated hypoxia, leading to enhanced CD8⁺ T-cell infiltration. Immune activation was confirmed by increased IL-12p70 and IFN- γ expression, indicating a shift toward an immunostimulatory TME (Figure 2). In contrast, melanoma exhibited stromal remodeling but a broad reduction in both pro- and anti-inflammatory cytokines, suggesting limited immune activation despite matrix normalization. Baseline cytokine profiling revealed tumor-type-specific immune signatures, while machine learning identified tumor stiffness as a shared predictor of therapeutic response.

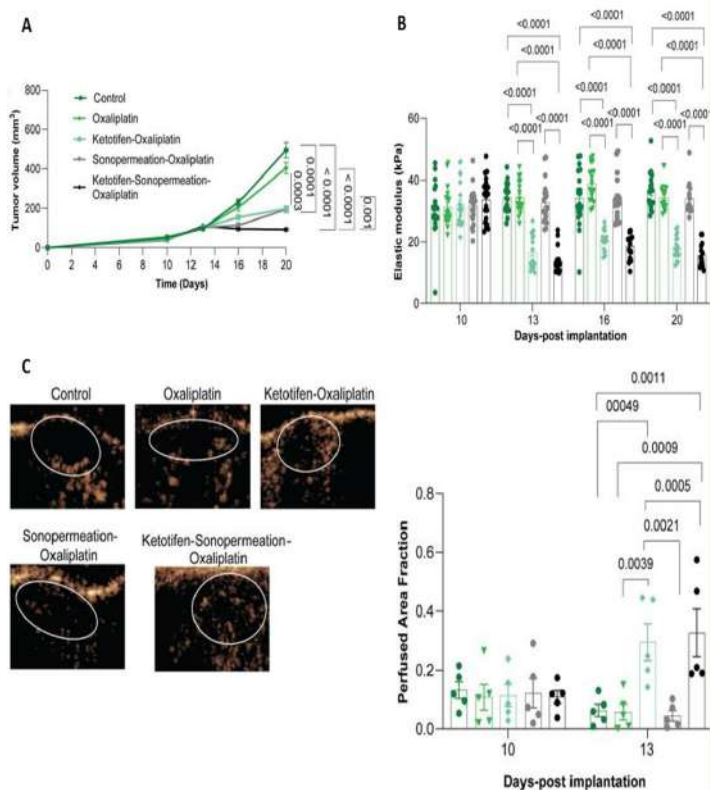


Figure 1. Combination of ketotifen and sonopermeation, reduces tumor volume and stiffness, increases tumor perfusion and improves chemotherapy efficacy in PDAC model.

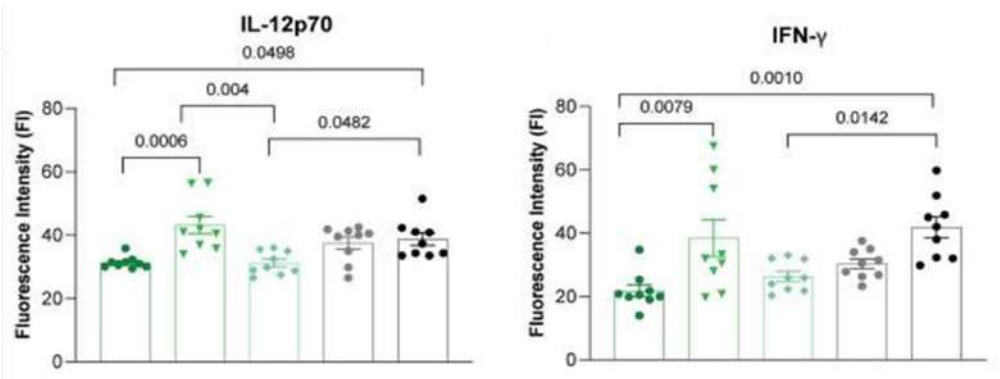


Figure 2. Cytokine profiling of PDAC tumors following microenvironmental modulation and chemotherapy.

Conclusion: The combination of ketotifen and ultrasound-mediated sonoporation enhances chemotherapy efficacy by modulating tumor mechanics, vascular function, and immune responses, with particularly strong effects in fibrotic PDAC. This approach represents a promising strategy to overcome therapeutic resistance in stroma-rich tumors.

Presenter:

Stella Angeli

The Cyprus Institute of Neurology & Genetics, Cyprus



Effects of Dietary Humic Acid Supplementation on Survival, Immune Response, and Gut Microbiota in Nile tilapia (*Oreochromis niloticus*) Challenged with *Lactococcus garvieae*

Edén Vladimir Gaitán-Morado¹, Ruth Escamilla-Montes², Antonio Luna-González³, Ruth Escamilla-Montes³, Genaro Diarte-Plata³ and Rodolfo Pérez-Rodríguez⁴

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Aquaculture of Nile tilapia (*Oreochromis niloticus*) has faced challenges from pathogens such as the emerging bacterium *Lactococcus garvieae*. Humic acid (HA) was used as a prophylactic measure to evaluate its effect on the intestinal microbiota, immune system, and survival in tilapia challenged with *L. garvieae* under laboratory conditions. Four concentrations of HA were tested in feed over 83 days. The intestinal microbiota was analyzed in treatment IV (1.0 g/ kg of feed) and control through 16S rRNA gene sequencing (V3). Liver and gill tissue were collected for immune system gene expression analysis (COX-2, TGF, IL-1 β , and TNF- α) through real-time qPCR methods. On day 71, the fish were inoculated by immersion with *L. garvieae* (CL50 = 8.5×10^5 CFU/mL). Gene expression in the liver (COX-2 and TNF α) and gills (COX-2, TGF- β , and IL-1 β) increased significantly in treatment IV. *Cetobacterium* was among the most abundant genera in the tilapia gut. Fusobacteriota (phylum) and the genera *Aeromonas*, *Rhodobacter*, *Gemobacter*, and *Ideonella* were enriched in HA, whereas the phylum *Patescibacteria* and genera *Pelomonas*, *Nitrobacter*, and *Clostridioi-*des decreased significantly. Dietary HA supplementation significantly altered gut microbiota diversity and the interaction network core. In channel catfish (*Ictalurus punctatus*), dietary HA supplementation significantly altered gut microbiota alpha diversity (Oladipupo et al. 2024). Our results in tilapia showed that, while Shannon and Simpson indices (reflecting both richness and evenness) showed no significant differences between HA and control group, the Chao1 and ACE indices (estimating species richness) were significantly higher

in HA- treated fish. Humic acid supplementation significantly improved tilapia survival against *L. garvieae* by enhancing immune gene expression and modulating gut microbiota—enriching *Fusobacteriota* and beneficial genera (*Rhodobacter*, *Ideonella*) while reducing pathogens (*Pelomonas*, *Clostridioides*). These findings support HA as a prophylactic alternative against lactococcosis in Nile tilapia (*O. niloticus*) farming.

Presenter:

Edén Vladimir Gaitán-Morado

Universidad Michoacana de San Nicolás de Hidalgo, México



Immunological Aspects in case of Metabolic Dysfunction-Associated Steatotic Liver Disease

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MASLD represents the hepatic manifestation of systemic metabolic dysfunction. MASLD is initiated by excess lipid accumulation driven by increased dietary fatty acid intake and accelerated de novo lipogenesis. Beyond lipid dysregulation, impaired hepatic insulin signaling leads to hyperglycemia and compensatory hyperinsulinemia, further stimulating lipogenesis and reinforcing a self-perpetuating metabolic cycle. These disturbances extend beyond the liver, contributing to atherogenic dyslipidemia and chronic inflammation. In parallel, gut dysbiosis and increased intestinal permeability amplify immune activation.

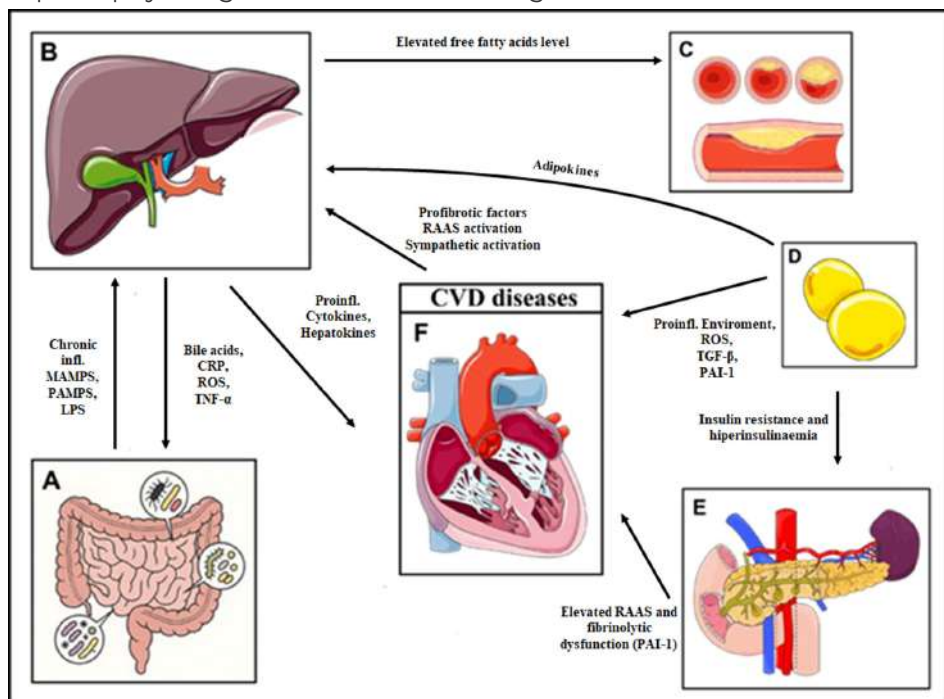
In MASLD, persistent subclinical inflammation is characterized by elevated circulating biomarkers, including tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), C-reactive protein (CRP), and fibrinogen. This inflammation, coupled with oxidative stress, impairs endothelial function and promotes subclinical atherosclerosis.

Immun-related pathologies in MASLD involves vascular immune activation and gut dysbiosis:

- TLR-NLRP3 signaling axis activates the caspase-1 (interleukin-1 β converting enzyme) and triggers proteolytic maturation of pro-inflammatory cytokines, including IL-1 β , thereby initiating the IL-1 β -IL-6-CRP cascade that drives vascular inflammation and atherosclerotic CVD. Collectively, the TLR-NLRP3 signaling axis links the atherogenic lipid milieu of MASLD to vascular immune activation.
- Dysbiosis, represents an important contributor to cardiometabolic dysfunction by perturbing both metabolic and immune pathways. A defining feature of dysbiosis is impairment of the intestinal barrier, resulting in increased mucosal permeability ("leaky gut"). This facilitates the translocation of microbial components and metabolites, including microbe- or patho-

gen-associated molecular patterns such as lipopolysaccharide (LPS) and peptidoglycans, as well as DAMPs released from injured enterocytes, into the systemic circulation. These signals activate innate immune pathways and promote a chronic, low-grade inflammatory state that represents a key mechanistic link between gut dysbiosis and cardiometabolic diseases, including atherosclerosis.

The pathophysiological mechanisms linking MASLD to CVD:



This presentation summarises the main immune-metabolism features, which play a pivotal role in MASLD progression.

Presenter:

Adrián Róbert Gál

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Monosodium Glutamate as an Innate Immune Modulator: Evidence from Experimental Models and Implications for Food Safety

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Monosodium glutamate (MSG) is a widely used food additive whose potential effects on immune function remain controversial. This review synthesizes current evidence on MSG as a modulator of innate immunity, with a focus on experimental models and mechanistic pathways. A targeted literature search (2015–present) was conducted using PubMed and Google Scholar, prioritizing studies evaluating innate immune endpoints, including cytokine production, oxidative stress and immune cell function.

Experimental evidence from *in vivo* and *in vitro* studies indicates that MSG exerts dose-dependent immunomodulatory effects. Chronic exposure in animal models has been associated with alterations in innate immune cell populations, including leukopenia, neutrophilia, and eosinophilia, alongside dysregulation of cytokine profiles, with increased levels of pro-inflammatory mediators such as TNF- α , IL-1 β , and IL-6. Concurrently, reductions in anti-inflammatory cytokines (e.g., IL-10) and immunoglobulin levels suggest impaired immune homeostasis.

Mechanistically, MSG-induced immunomodulation is closely linked to oxidative stress and redox imbalance. Increased reactive oxygen species production, mitochondrial dysfunction, and activation of inflammatory pathways—particularly TLR4/NF- κ B signaling—have been consistently reported. These processes contribute to cellular damage, apoptosis, and disruption of epithelial barrier integrity, particularly in the gastrointestinal tract. Alterations in gut microbiota composition further mediate immune effects, with high-dose MSG promoting dysbiosis and pro-inflammatory signaling, while low doses may exert minimal or even modulatory effects.

Despite consistent findings in experimental systems, translational relevance to humans remains uncertain. Controlled human studies generally report limited or no significant immunological effects at typical dietary intake levels, highlighting the importance of dose, exposure duration and experimental context.

Overall, MSG acts as a context-dependent modulator of innate immunity, primarily through oxidative stress and inflammatory signaling pathways. These findings underscore the need for human-relevant, low-dose and long-term studies to better inform food safety assessment and regulatory frameworks.

Presenter:

Fatemeh Sayareh

University of Warmia and Mazury in Olsztyn, Poland



Patient Perceptions and Type 1 Delay in Head and Neck Cancer: A Scoping Review of High- and Low-/Middle-Income Countries

Gordon Buhwa¹, Meredith A. Anderson², Connor Mayes², Atharva Desai³, Kyle Polen⁴, Jeffrey L. Black⁵, Isabella Reitz², Gamaliel Taengwa⁶, Jose Chacon⁷, Anna Davide^{8,9} and Joshua P. Wiedermann^{9,10}

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Objective: To identify and compare how patient perceptions contribute to Type 1 Delays, defined as delays in deciding to seek care, among individuals with head and neck cancer (HNC) in high-income countries (HICs) and low- and middle-income countries (LMICs).

Methods: We conducted a scoping review and systematic search of MEDLINE, Embase, Cochrane Library, and Scopus from inception through November 20, 2023. Eligible studies included patients with HNC in HICs or LMICs who reported patient perceptions contributing to Type 1 Delays. Data extraction was performed in duplicate, and barriers were categorized thematically. Fisher's exact test was used to compare the frequency of reported barriers between HIC and LMIC studies.

Results: Seventeen studies met inclusion criteria, of which 64.7% (n = 11) were conducted in HICs and 35.3% (n = 6) in LMICs, representing 9 countries and 1410 participants. The most frequently cited perceptions contributing to delay were symptom un-awareness, misinterpretation, and minimization. Additional barriers included fear of diagnosis, stigma, alternative medicine use, financial chal-

lenges, and geographic barriers. Transport/geographic barriers showed a trend toward being more common in LMIC studies ($p = 0.05$). Descriptive patterns suggested LMIC patients more often reported structural and sociocultural barriers, whereas HIC patients more commonly cited psychosocial or perceptual barriers.

Conclusion: Type 1 Delays in HNC are common across both HICs and LMICs, with overlapping and region-specific contributors. Tailored interventions, such as patient education, stigma reduction, and improved access to diagnostic services, may promote earlier detection and improve global HNC outcomes.

Presenter:

Buhwa Gordon

Makerere University, Uganda



C1q is Recognized as a Soluble Autoantigen by Anti-C1q Antibodies of Patients with Systemic Lupus Erythematosus

Vancho Donev¹, Alexandra Anatolieva Atanasova², Ginka Ilieva Cholakova², Alexandra Panagiotis Kapogianni², Delina Ivanova³, Rumen Malinov Stoilov³, Anna Dimitrova Yordanova⁴, Vanya Petkova Bogoeva⁴ and Ivanka Georgieva Tsacheva²

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Background and aims: C1q is an autoantigen in different autoimmune disorders, Systemic Lupus Erythematosus (SLE) and Lupus Nephritis (LN) among them. The two functional domains of C1q, the collagen-like region (CLR) and the globular head region (gC1q), are frequently recognized by autoantibodies in SLE and LN when C1q is immobilized. We studied whether autoantibodies to C1q in SLE and LN patients recognized C1q as a soluble autoantigen and whether the act of immobilization was a prerequisite for the recognition of C1q autoepitopes localized on gC1q domains.

Methods: The interaction of soluble C1q and its globular fragments ghA, ghB, and ghC with immobilized IgG autoantibodies (and vice versa) from sera of 48 patients with SLE and LN was studied with ELISA. Data were compared using Spearman correlation coefficient. Fluorescence spectroscopy was used to study the interaction between C1q and LN IgG autoantibodies both presented in solution.

Results: We found that anti-C1q autoantibodies from SLE and LN patients specifically bound C1q and gC1q fragments, ghA, ghB, and ghC, both as immobilized and soluble antigens. Correlation analysis indicated a negative correlation between the levels of autoantibodies against immobilized and soluble C1q and immobilized and soluble gC1q fragments which indicate different epitopes when these proteins were recognized as autoantigens in soluble and immobilized conformations.

Conclusions: Serum C1q in patients with SLE is a target molecule for binding from anti-C1q autoantibodies. The gC1q region undergoes a conformational change in an immobilized and a soluble form, thus exposing different epitope-binding sites.

Presenter:

Vancho Donev

Bul Bio—NCIPD EAD, Bulgaria



LIFE-CRAFT: A Multi-Agentic Conversational RAG Framework for Lifestyle Medicine Coaching with Context Traceability and Case-Based Evidence Synthesis

Hania Aslam, Gousia K. Malak, Max Renault and Rajat M. Thomas

Weill Cornell Medicine-Qatar, Qatar

Lifestyle medicine offers a proactive and behavior-centric approach to managing chronic diseases, yet scalable, personalized coaching remains a significant challenge. We present LIFE-CRAFT (Lifestyle Intervention Facilitation Engine-Conversational RAG Framework with Traceability), a novel multi-agentic framework that leverages Large Language Models (LLMs) and Retrieval-Augmented Generation (RAG) for personalized, lifestyle medicine related guidance. Central to LIFE-CRAFT is an orchestrator agent that interprets user queries and dynamically routes them to specialized supporting agents with expertise in prominent subdomains of lifestyle medicine. LIFE-CRAFT distinguishes itself from prior LLM-based assistants by introducing adaptive RAG pipeline with specialized sub-domain expert agents alongside dynamic domain routing and iterative feedback-based self-correcting refinement mechanisms for fine-grained context traceability as each agent's response is grounded in semantically relevant document chunks retrieved from a well-curated, expert-reviewed PDF corpus of over 7,000 PubMed articles. For every generated response, the system logs the source document context that contributed to the response, providing transparency and explainability crucial for health-related decision support. The framework is evaluated across 864 GPT-4-generated lifestyle case scenarios using both LLM-based and human-in-the-loop evaluation. Initial results indicate that LIFE-CRAFT achieves higher context alignment and perceived trustworthiness, owing to its modular architecture, agentic decision-making, and transparent evidence grounding. This work demonstrates the feasibility and promise of multi-agentic, traceable RAG systems in supporting scalable, explainable, and personalized digital lifestyle medicine coaching—laying the foundation for future health AI systems that blend reasoning, retrieval, and real-world relevance. We intend to release the code in a GitHub repository upon acceptance of the paper to promote transparency and reproducibility.

Presenter:

Hania Aslam

Weill Cornell Medicine-Qatar, Qatar



Oncolytic Potential of Newcastle Disease Virus: Modulation of Apoptosis-Related Genes in the TC-1 Cell Line

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Central Tehran Branch, Iran

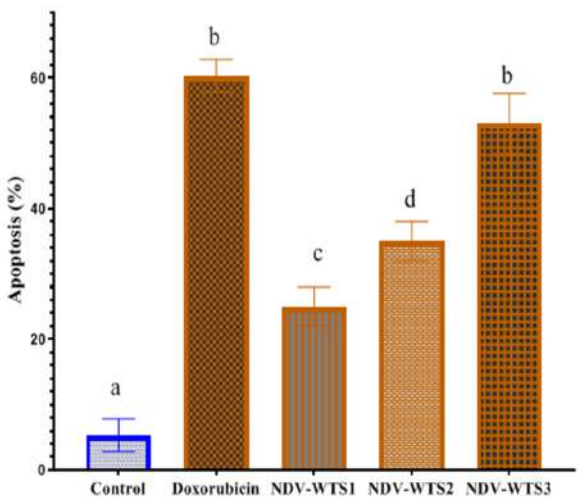
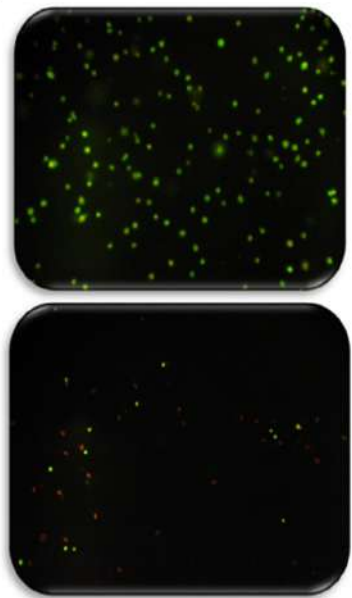
²Applied Virology Research Center, Biomedicine Technologies
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New therapeutic approaches are needed for the management of lung and human papillomavirus (HPV)-associated malignancies due to the limited efficacy and poor selectivity of conventional anticancer treatments. TC-1, a transformed murine lung epithelial cell line expressing HPV-16 E6 and E7 oncoproteins, is a well-established experimental model for evaluating novel anticancer strategies. The present study aimed to investigate the oncolytic potential of wild-type Newcastle disease virus (NDV-WTS) against TC-1 cells and to determine its effects on cell viability and apoptosis-related pathways.

In this *in vitro* experimental study, TC-1 cells were infected with NDV-WTS at multiplicities of infection (MOIs) of 1, 2, and 4. The scope of the study included the evaluation of cytotoxicity, oxidative stress, membrane damage, and apoptotic responses following viral treatment. Cell viability was assessed using the MTT assay. Lactate dehydrogenase (LDH) release was measured as an indicator of membrane damage, while intracellular reactive oxygen species (ROS) levels and caspase-8 and caspase-9 activities were analyzed to examine apoptosis-related mechanisms. In addition, the expression of apoptosis-associated genes, including Bax, Bcl2, Casp8, and Casp9, was determined.

The results demonstrated that NDV-WTS significantly decreased the viability of TC-1 cells in a dose-dependent manner. Viral treatment also led to a marked increase in LDH release, ROS production, and caspase-8 and caspase-9 activities compared with untreated controls. Furthermore, gene expression analysis revealed significant upregulation of the pro-apoptotic genes Bax, Casp8, and Casp9, together with downregulation of the anti-apoptotic gene Bcl2, indicating activation of both intrinsic and extrinsic apoptotic pathways.

In conclusion, NDV-WTS exhibited significant oncolytic activity in TC-1 cells through induction of cytotoxicity and apoptosis. These findings suggest that NDV-WTS may serve as a promising candidate for the development of virotherapy-based approaches against HPV-related and lung malignancies. Further *in vivo* studies are required to confirm its therapeutic applicability.



Presenter:
Reihaneh Ramezani
Islamic Azad University, Iran

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