

International Conference on

IMMUNOLOGY

&

VACCINES

Rome, Italy

November 18-19, 2024

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CONFERENCE PROGRAMME

DAY 1 - NOVEMBER 18, 2024

Meeting Hall: Ripa	
09:00 - 09:45	Registrations
09:45 - 10:00	Introduction
Keynote Presentations	
10:00 - 10:40	
Title: Quality by Design and Vaccine Development for Commercialisation Lee Smith, GreyRigge Associates Ltd, United Kingdom	
10:40 - 11:20	
Title: Understanding the Role of Macrophages in the Processes associated with the Development of Endometriosis in the Mare Anna Szóstek-Mioduchowska, Institute of Animal Reproduction and Food Research of Polish Academy of Sciences, Poland	
Networking & Refreshments 11:20 - 11:40 @ Lobby Bar	
11:40 - 12:20	
Title: Therapy of Chronic Inflammations David Naor, The Hebrew University - Hadassah Medical School, Israel	
12:20 - 13:00	
Title: Decrease of E-Cadherin Expression in Q Fever Patient: A First Step Progression toward Non-Hodgkin Lymphoma? Ikram Omar Osman, Service des Laboratoires, Djibouti	
Group Photo 13:00 - 13:10	
Lunch: 13:10 - 14:00 @ Ristorante	
14:00 - 14:40	
Title: Exploring the Tumor Microenvironment in Breast Cancer: Uncovering Novel Therapeutic Targets. Abdallah Badou, Hassan II University of Casablanca, Morocco	
Oral Presentations	
Session Chair	Dariusz J. Skarzynski, Institute of Animal Reproduction and Food Research, Poland

Session Co-chair	Abdallah Badou , Hassan II University of Casablanca , Morocco
Sessions	Cancer Immunology and Immunotherapy Host Pathogen Interactions Reproductive Immunology Vaccine Research & Development Vaccine Immunology Novel Vaccination Strategies Vaccine Manufacturing & Quality Assurance
14:40 - 15:05	
Title: New Findings on Immune-endocrine Regulations of Development, Rescue and Regression of the Corpus luteum: Cow as A Model	
Dariusz J. Skarzynski, Institute of Animal Reproduction and Food Research, Poland	
15:05 - 15:30	
Title: The Progression of Glioblastoma is Influenced by the Expression of PD1 and TIM3 on CD4+CD103+ and CD8+CD103+ T cells	
Gabriele Lucia, Istituto Superiore Di Sanità, Italy	
15:30 - 15:55	
Title: Vaccine Tech Transfer : Challenges and Best	
Lokender Kaushik, Walvax Biotechnology Co.Ltd, China	
Networking & Refreshments: 15:55 - 16:30 @ Lobby Bar	
16:30 - 16:55	
Title: Discussion on the Concept of Immunity and How Human Body System Could Build Immunity against COVID 19 and It's Relevance to Public Health Education	
Albert Opoku, Nursing and Midwifery Training College, Ghana	
16:55 - 17:20	
Title: Molecular Characterization of the Genes Encoding Resistance to the Beta- Lactam Family in Strains of <i>Klebsiella pneumonia</i> Isolated at the Brazzaville University Hospital	
Fils Landry Mpelle, Marien Ngouabi University, Republic of the Congo	
Video Presentation	
VP-01	
Title: The Case for Shedding of Spike mRNA Gene Therapy Products Causing Excess Death Globally	
Marian Laderoute, Canada	
VP-02	
Title: Lung Epithelial Signaling Mediates Early Vaccine-Induced CD4+ T Cell Activation and Mycobacterium tuberculosis Control	
Shibali Das, Washington University in St. Louis, USA	
Day-1 Concludes followed by Vote of Thanks	

Day 1

Keynote Presentations



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QUALITY BY DESIGN IN VACCINE DEVELOPMENT C COMMERCIALIZATION

Lee Smith

Grey Rigge Associates Ltd, United Kingdom

Abstract

Quality by Design (QbD) is a process that has been in use in pharmaceutical development since the 1990's. In essence, QbD in vaccine development is ensuring that the pathway of developing a process to produce a vaccine starts with the end in mind, specifically commercial production. Regulatory guidance exists that explains, at a high level, the concept of how the development of a process should be approached and managed. This involves thinking about the profile of the vaccine that needs to be designed, and methodically thinking about what product attributes make the vaccine both efficacious and safe. Furthermore, we consider how the vaccine can be manufactured, so that it produces an identical product each and every time it is made. This involves understanding the production process and through the use of risk-based approaches and statistics, we can identify the critical elements of the process.

Consequently, these can be optimized and brought under control, ensuring robust production of a safe and efficacious vaccine suitable for commercial launch.

Biography

Lee is a Director and Principal consultant with experience spanning biopharmaceutical CMC, process, analytical, formulation pre-clinical and clinical assay development as well as experience in product characterization and regulatory submissions and interactions. He is regularly involved in applying QbD and advising on the use of DoE and data analysis for processes, formulation and assays, with a particular expertise in bioassays. This includes the development, optimization and validation of both biopharmaceutical processes and methods.



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UNDERSTANDING THE ROLE OF MACROPHAGES IN THE PROCESSES ASSOCIATED WITH THE DEVELOPMENT OF ENDOMETROSIS IN THE MARE

Anna Szóstek-Mioduchowska

Institute of Animal Reproduction and Food Research of Polish Academy of Sciences, Poland

Abstract

Equine endometrosis is a chronic degenerative condition involving stromal fibrosis together with degenerative changes in the endometrium of the mare. It leads to the destruction of the endometrial tissue architecture and the impairment of endometrial function resulting in early pregnancy dysfunction and embryo loss. Endometrosis can be categorized according to the degree of endometrial structural changes. Four categories are identified: I (healthy endometrium, no changes), IIA (mild condition), IIB (moderate condition), and III (severe condition). As the severity of endometrosis deepens, the expected foaling rate decreases by even 90% in mares with endometrium category III. The pathophysiologic mechanism of endometrosis remains unknown. Macrophages ($M\phi$) are important immune cells in the defense against pathogens and in tissue homeostasis and repair. Macrophages are generally classified into two broad but distinct subsets, either classically activated ($M\phi 1$) or alternatively activated ($M\phi 2$). Research suggests that $M\phi 1$ and $M\phi 2a$ play an important role in the development of fibrosis. Our previous results showed the infiltration of macrophages in category II A endometria. Therefore, the aim of our study was to (1) characterize the presence of $M\phi 1$ and $M\phi 2a$ in the endometrial tissue at different stages of endometrosis, (2) compare the effect of $M\phi^{(IFN\phi/LPS)}$ and $M\phi^{(IL4/IL13)}$ secretome on the expression of ECM-related genes and the transcriptome of fibroblasts in vitro, (3) investigate the effect of intrauterine administration of $M\phi^{(IFN\phi/LPS)}$ and $M\phi^{(IL4/IL13)}$ on the expression of ECM-related genes and the transcriptome of the endometrium of the in vivo. In vitro treatment of endometrial fibroblasts with $M\phi^{(IFN\phi/LPS)}$ or $M\phi^{(IL4/IL13)}$ secretome resulted in significant differential expression of 437 and 301 transcripts, respectively, while in vivo intrauterine administration of $M\phi^{(IFN\phi/LPS)}$ or $M\phi^{(IL4/IL13)}$ induced significant differential expression of 142 and 341 transcripts, respectively. The findings of this study indicate that, in addition to its role in immunological defence, $M\phi$ may exert direct effects on endometrial cells.

Biography

Anna Szóstek-Mioduchowska, Ph.D. is an Assistant Professor at the Department of Reproductive Immunology and Pathology, Institute of Animal Reproduction and Food Research of the Polish Academy of Sciences. Dr. Szóstek-Mioduchowska has authored more than 60 publications in international journals. She is interested in the molecular and metabolic processes involved in the pathogenesis of equine endometrosis. Dr. Szóstek-Mioduchowska and her collaborators have found alterations in secretory function and transcriptomic changes in fibrotic equine endometrium. Her research now focuses on the role of immune cells and their products in processes associated with the development of endometrosis. In particular, on the effect of different populations of macrophages and T-helper cells on endometrial fibroblasts in the context of tissue remodelling, ECM deposition, fibroblast proliferation and migration, and myofibroblast differentiation under in vitro conditions.



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THERAPY OF CHRONIC INFLAMMATIONS

David Naor

The Hebrew University - Hadassah Medical School, Israel

Abstract

We have reported in our previous communication (J Autoimmune. 2021 Nov; 124:102713) that a 5-MER peptide (5-MP; Methionine, Threonine, Alanine Aspartic Acid, Valine (MTADV) attenuates the pathology of animal models of Rheumatoid Arthritis, Crohn's Disease/Ulcerative Colitis and Multiple Sclerosis. In addition, we report herein the ability of 5-MP to alleviate the lung inflammation and fibrosis of Bleomycin-induced fibrosis (BIF), a mouse model of human Idiopathic Pulmonary Fibrosis (IPF). All these maladies share Serum Amyloid A, that fuels chronic inflammation and fibrosis. In our previous communication we presented evidence that 5-MP targets SAA and consequently interferes with formation of SAA oligomers and aggregated fibrils, which are involved in chronic inflammations and fibrosis in vivo and in release of pro-inflammatory cytokines in vitro. In this report we are focusing, as indicated above, on the suppressive effect of 5-MP on fibrosis in vivo, and on fibroblast biological functions in vitro. Inflammation-induced uncontrolled biological activities of fibroblasts and monocytes generate fibrosis, stressing the linkage between inflammation and fibrosis. The last is much less responsive than inflammation to medical intervention. Indeed, we found that 5-MP inhibits mRNA expression of pro-inflammatory cytokines (IL-6, IL-1 β , TNF α) in fibroblasts and monocytes and their release from these SAA-stimulated cells. In conclusion 5-MP displays therapeutic potential in lung fibrosis-associated diseases, which face unmet remedy at present.

Focusing on therapy of chronic inflammations, we have reported in our previous communication (J Autoimmune. 2021 Nov; 124:102713) that a 5-MER peptide (5-MP; Methionine, Threonine, Alanine Aspartic Acid, Valine (MTADV) attenuates the pathology of animal models of Rheumatoid Arthritis, Crohn's Disease/Ulcerative Colitis and Multiple Sclerosis. In this report we described the ability of 5-MP to alleviate the lung inflammation and fibrosis of Bleomycin-induced fibrosis (BIF), a mouse model of human Idiopathic Pulmonary Fibrosis (IPF). All these maladies share Serum Amyloid A, that fuels chronic inflammation and fibrosis. Further, in our previous communication we presented evidence that 5-MP targets SAA and consequently interferes with formation of SAA oligomers and aggregated fibrils, which are involved in chronic inflammations and fibrosis in vivo and in release of pro-inflammatory cytokines in vitro. In this report we focus, as indicated above, on the suppressive effect of 5-MP on fibrosis in vivo, and on fibroblast biological functions in vitro. Inflammation-induced uncontrolled biological activities of fibroblasts and monocytes generate fibrosis, stressing the linkage between inflammation and fibrosis. The last is much less responsive than inflammation to medical intervention. To this end, we found that 5-MP inhibits mRNA expression of pro-inflammatory cytokines (IL-6, IL-1 β , TNF α) in fibroblasts and monocytes and their release from the SAA-activated cells. In conclusion 5-MP displays therapeutic potential in lung fibrosis-associated diseases, which face unmet remedy. In addition, we have reported in our previous communication (J Autoimmune. 2021 Nov; 124:102713) that a 5-MER peptide (5-MP; Methionine, Threonine, Alanine Aspartic Acid, Valine (MTADV) attenuates the pathology of animal models of Rheumatoid Arthritis, Crohn's Disease/Ulcerative Colitis and Multiple Sclerosis. In this report we further describe the ability of 5-MP to alleviate the lung inflammation and fibrosis of Bleomycin-induced fibrosis (BIF),



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a mouse model of human Idiopathic Pulmonary Fibrosis (IPF). All these maladies share Serum Amyloid A, that fuels chronic inflammation and fibrosis. We should further indicate that in our previous communication we presented evidence that 5-MP targets SAA and consequently interferes with formation of SAA oligomers and aggregated fibrils, which are involved in chronic inflammations and fibrosis in vivo and in release of pro-inflammatory cytokines in vitro. In this report we focus, as indicated above, on the suppressive effect of 5-MP on fibrosis in vivo, and on fibroblast biological functions in vitro. Inflammation-induced uncontrolled biological activities of fibroblasts and monocytes generate fibrosis, stressing the linkage between inflammation and fibrosis. The last is much less responsive than inflammation to medical intervention. However, we found that 5-MP inhibits mRNA expression of pro-inflammatory cytokines (IL-6 IL-1 β , TNF α) in fibroblasts and monocytes and the release of the corresponding proteins from SAA-activated cells. In conclusion 5-MP displays therapeutic potential in lung fibrosis-associated diseases, which face unmet remedy at present.

Biography

David Naor is a professor of immunology in the Hebrew University and was the head of Milton Winograd chair of cancer studies. Professor Naor served as associate visiting professor in various international universities and institutions, for example Charing Cross Sunley Research Centre, London, September 1987 to November 1988. Professor Naor received grants from many institutions, for example German Cancer Foundation. He published a book entitled "Immunosuppression and Human Malignancy"; Humana Press, New Jersey, 1988 as well as 150 articles. He is supervising 2 post docs. He was a founder of the start-up company Maimonides. He was a member of the editorial board of Journal of Autoimmunity. He received award from Johnson & Johnson, Focused Giving Program "in recognition of outstanding research toward the advances of science and technology in health care." He received the Kaye Innovation Award from the Hebrew University of Jerusalem for Scientific innovation.



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DECREASE OF E-CADHERIN EXPRESSION IN Q FEVER PATIENT: A FIRST STEP PROGRESSION TOWARD NON-HODGKIN LYMPHOMA

Ikram Omar Osman

Service des laboratoires, Djibouti

Abstract

Q fever caused by the obligate intracellular bacterium *C. burnetii*, is a zoonotic disease with world-wide prevalence. In humans, the infection remains asymptomatic in a high percentage of cases and symptomatic infection named Acute Q fever usually resolves spontaneously in a few weeks. Although the infection rarely leads to death, in less than 5% of cases the symptoms do not resolve and become persistent, evolving into endocarditis, vascular infection or osteoarticular infection. As, *C. burnetii* is known to infect myeloid cells such as monocytes and macrophages, several lymphoid disorders have been reported and a diagnostic of Q fever with a persistent focal infection was identified as a greater risk factor in the occurrence of Non-Hodgkin Lymphoma (NHL).

By exploring the complex distribution of genes whose expression is modulated in Q fever patients with different clinical outcomes, we reported an association between the infection and a decrease of the expression of cell-surface protein E-cadherin (E-cad), which behave as a tumor suppressor, as well as a greater release of its soluble fraction (sE-cad) into the bloodstream. An *in vitro* model of *C. burnetii*-infected cells was characterized, allowing us to demonstrate that the bacterium triggers sE-cad release after cleavage of E-cad by a sheddase, thereby altering cell-to-cell interaction and protein signaling. Using a straightforward bioinformatics approach to scan the complete genomes of four laboratory strains of *C. burnetii*, we provide evidence that *C. burnetii* encodes a 451 amino acid sheddase (CbHtrA) belonging to the HtrA family and differently expressed according to the bacterial virulence. Finally, we revealed that cleavage of E-cad by and an artificial CbHtrA on macrophages-THP-1 cells leads to an M2 polarization of the target cells and the induction of their secretion of IL-10, which 'disarms' the target cells and improves *C. burnetii* replication.

These data allow a better understanding of the physiopathological mechanisms induced by *C. burnetii* in infected patients and opened a new area of research for pro-carcinogenic co-factor accounting for progression of Q fever toward NHL, since the disruption of E-cad and release of sE-cad was previously reported to be associated to breast, gastric and colorectal solid tumors.

Biography

Ikram OMAR OSMAN has expertise in the field of host-pathogen interaction and is passionate about understanding the pathophysiology of infectious diseases in order to improve health and well-being. During her PhD in infectious diseases with a specialization in Immunobiology of host-pathogen interactions: from the cell to the clinic at the IHU Méditerranée Infection in Marseille, she was able to publish a large number of research articles on *Coxiella burnetii* and SARS-CoV-2-induced disease and its pathophysiological consequences. Among her research projects, she succeeded in providing epidemiological information on the COVID-19 virus to Djibouti's public health department for the first time to bridge the gap of knowledge regarding the SARS-CoV-2 strains spreading in Djibouti during two epidemic waves. Currently, she is the head of the laboratory service of the CNSS healthcare department in Djibouti and continues to collaborate with her former team at IHU Marseille to elaborate an explanation for a better understanding of the pathophysiological mechanisms induced by *C. burnetii* infection and its progression toward a NH lymphoma, as well as a COVID-long explanation based on the effect of SARS-CoV-2 infection on the integrity of the intestinal barrier.



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DECODING THE TUMOR MICROENVIRONMENT IN BREAST CANCER

Abdallah Badou

Hassan II University, Morocco

Abstract

Breast cancer is a diverse and widely distributed form of cancer that represents a major threat to women worldwide. The cross-talk between immune and malignant cells appears to be crucial in the eradication of breast cancer. Despite progress in treatment modalities, persistently high rates of recurrence, metastasis, and treatment resistance underscore the urgent need for new therapeutic approaches especially in aggressive forms of the disease. Here, we performed differential expression analysis in patients with tumor-infiltrating CD8 T cells between the most (triple negative breast cancer, TNBC) and less aggressive forms of the disease and identified a member of the Fatty Acid Binding Proteins family, FABP, which was significantly and specifically upregulated in TNBCs with CD8+ T cell infiltration. High levels of FABP correlated with patient's poor survival. Regression analysis showed that FABP5 effect was intrinsic and of independent prognostic significance. The analysis of data from a Single-Cell Breast Cancer database confirmed that FABP is highly expressed in exhausted and dysfunctional CD8 T cells. Moreover, gene expression analysis in the Moroccan cohort corroborated that FABP was significantly associated with the most aggressive molecular and histological subtype of the disease. Interestingly, FABP knockdown in TNBC cancer cell lines co-cultured with human CD8 T cells, resulted in enhanced CD8 T cell effector functions. Our data, and for the first time, provided insights into an impaired mechanism behind CD8 T cell dysfunction/exhaustion in TNBC by revealing FABP as a novel therapeutic target. FABP blockade alone, or in combination, could be of major significance in TNBC treatment.

Biography

Abdallah Badou is currently a Professor of Immunology and Molecular Biology and the head of the Immuno-Genetics and Human Pathology laboratory at the Hassan II University (Faculty of Medicine and Pharmacy), Casablanca, Morocco. Dr. Badou has completed his Master's degree in René Descartes University (Paris, France) in 1994, then his PhD in Immunology in 1998 at Paul Sabatier University (Toulouse, France). Afterwards, He joined the Immunobiology department at Yale University School of Medicine (Connecticut, USA) from 1999 to 2007, as a post-doc then as an associate research scientist. In 2007, He joined Cadi Ayad University in Morocco as an assistant then qualified professor. Since 2014, Dr. Badou was affiliated to the Faculty of Medicine and Pharmacy, where He was promoted as full professor in 2018. From January 2023 to February 2024, He was affiliated to Mohammed VI Center for Research and Innovation as Scientific Director then as General Director. His research topics are related to the study of the tumor microenvironment mainly in breast cancer and gliomas. Dr. Badou is serving in the editorial board of several international journals. He is currently the President of the Moroccan Society of Immunology (SMI), the Secretary General of the Federation of African Immunological Societies (FAIS) and a member of the International Union of Immunological Societies (IUIS) Council.

Day 1

Oral Presentations



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NEW FINDINGS ON IMMUNE-ENDOCRINE REGULATIONS OF DEVELOPMENT, RESCUE AND REGRESSION OF THE CORPUS LUTEUM: COW AS A MODEL

Dariusz J Skarzynski

Institute of Animal Reproduction and Food Research, Poland

Abstract

The corpus luteum (CL) is a transient organ consists different luteal cell types, both steroidogenic and accessory luteal cells: immune cells, endothelial cells, pericytes and fibroblasts). CL secretes progesterone (P4), a hormone essential for control of reproductive cyclicity, and establishment of pregnancy. Mechanisms controlling CL development and function may involve factors produced both within and outside this gland. Arachidonic acid metabolites including prostaglandins (PG), growth factors and cytokines were shown to complement gonadotropins action in the process of CL development. In absence of pregnancy, CL undergoes regression (luteolysis), which is crucial to prepare for next cycle. Luteolysis, initiated in bovine species by uterine PGF₂, is usually divided into two phases, i.e. the decline in P4 concentrations (functional luteolysis) and the elimination of luteal cells/tissue from ovary (structural luteolysis). Crucial role in elimination of cells which constitute the bovine CL during structural luteolysis plays programmed cell death (PCD). Cell death is a fundamental pathophysiological process and also an essential event in normal life and development. However this is not the sole form of programmed cell death (PCD) and many other alternative or atypical pathways have now been described. Concerning CL regression the study was focused mainly on three types PCD: type 1 - caspases-dependent apoptosis, type 2 - autophagic cell death via autophagy related gene (Atg) family and type 3 - receptor-interacting protein kinases (RIPKs)-dependent programmed necrosis (necroptosis). These PCD pathways are not completely independent and interact with each other in a complex manner. Intraluteal mediators of PGF₂ α , such as tumor necrosis factor- (TNF), interferon- (IFNG), Fas ligand and nitric oxide, have been shown to activate intracellular cell death pathways in CL. Recently, we have shown that the expression of RIPK proteins, which are the crucial mediator for necroptosis, is increased in the bovine CL during luteolysis, and they can be up-regulated by TNF and IFNG in the bovine CL. Interestingly, the genes related to cell death pathways exhibit stage-specific responses to PGF₂ α administration depending on its local or systemic actions. Locally-acting PGF₂ α may play a luteoprotective role by inhibiting apoptosis and necroptosis in the early CL. The effects of PGF₂ α and cytokines on luteal function are very complex, and its different effects depend on cell composition and contact as well on number of immune mediators.

The research was supported by the OPUS Grant of the Polish NSC (2018/29/B/NZ9/00391).

Biography

Dariusz J. SKARZYNSKI (DVM, Ph.D., Dr.Sci) is a Head of Department of Reproductive Immunology and Pathology, IAR&FR PAS in Olsztyn, Poland and Professor of Department of Reproduction with Large Animal Clinic of Faculty of Veterinary Medicine, UE&LSci in Wrocław, Poland. His research area is immunology and pathology of reproduction in domestic animals, especially immuno-endocrine, cellular and molecular regulations of the estrus cycle and pregnancy and pathogenesis of uterine disorders in cows and mares. Current research include: (1) Immuno-endocrine, cellular and molecular regulations of the release and action of cytokines and arachidonic acid metabolites in the female reproductive tract; (2) Immuno-endocrine mechanisms controlling embryo-maternal interactions; (3) Regulation of proliferation, angiogenesis and cell death mechanisms in the female reproductive tract; (4) Pathogenesis



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of *endometritis* and *endometrial fibrosis* in mares and cows; (5) Experimental models for the study of embryo-maternal interactions and early embryo mortality in human and animals.



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THE PROGRESSION OF GLIOBLASTOMA IS INFLUENCED BY THE EXPRESSION OF PD1 AND TIM3 ON CD4+CD103+ AND CD8+CD103+ T CELLS

Gabriele Lucia

Istituto Superiore di Sanità , Italy

Abstract

Glioblastoma (GB), specifically the IDH-wildtype variant, is the most common and aggressive high-grade primary malignant brain tumor, characterized by an extremely poor prognosis. While immune checkpoint inhibitors (ICIs) have yielded disappointing results in GB treatment, the discovery of a small subset of patients who respond suggests that the intratumoral immune environment-particularly tumor-infiltrating lymphocytes (TILs) plays a crucial role in influencing disease outcomes. This study aims to identify the characteristics of TIL subpopulations associated with GB prognosis, while also evaluating the predictive significance of peripheral T cell subsets.

In this single-cohort observational study, patients undergoing GB resection and standard therapeutic and follow-up protocols in Neuro-Oncology were included. Among the 45 patients with histologically confirmed WHO grade 4 GB, 26 (60.0%) had MGMT promoter methylation, 24 were male (53.3%), and the median ages were 57 for younger patients (≤ 63 years) and 71 for older patients (> 63 years). Key predictors of overall survival (OS) included the Karnofsky Performance Status (KPS), with a median OS of 14.7 months for those with $KPS \geq 70$ compared to 7.5 months for $KPS < 70$ ($p=0.0007$), and gross total resection (GTR), which had a median OS of 15 months versus 9 months for non-GTR patients ($p=0.004$). While patients with a methylated MGMT promoter exhibited a non-significantly longer median OS compared to those with an unmethylated promoter (14.5 vs. 10.5 months), the study further characterized the expression of lineage, differentiation, memory, activation, and inhibition markers on intratumoral and peripheral CD4+ and CD8+ T cell subpopulations. The correlation between various CD4+ and CD8+ T cell subsets and OS or progression-free survival (PFS) was analyzed using multiparametric flow cytometry, along with univariate and multivariate analyses.



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VACCINE TECH TRANSFER: CHALLENGES AND BEST PRACTICES

Lokender Kaushik

Walvax Biotechnology, China

Abstract

The transfer of vaccine technology from research and development to manufacturing facilities is a critical process that ensures the timely and efficient production of life-saving vaccines. This presentation aims to share experiences and insights gained from vaccine tech transfer projects, highlighting the challenges faced and the best practices employed to overcome them.

Vaccine tech transfer involves the successful transfer of manufacturing processes, analytical methods, and intellectual property between organizations or facilities. It is a complex undertaking that requires meticulous planning, effective communication, and collaboration among multiple stakeholders, including researchers, technology providers, and manufacturing teams.

This presentation will delve into the key stages of vaccine tech transfer, including process characterization, knowledge transfer, facility preparation, and validation. Particular emphasis will be placed on the importance of robust project management, risk assessment, and mitigation strategies to ensure a seamless and compliant tech transfer process.

Furthermore, the presentation will explore the regulatory landscape surrounding vaccine tech transfer, addressing the requirements and guidelines set forth by regulatory bodies such as the World Health Organization (WHO) and national regulatory authorities. Adherence to these regulations is crucial for ensuring the safety, efficacy, and quality of the vaccines produced.

Real-world case studies will be presented, showcasing successful vaccine tech transfer projects and the lessons learned from overcoming challenges related to scale-up, process optimization, and quality control. These case studies will provide valuable insights and practical recommendations for stakeholders involved in vaccine tech transfer initiatives.

By attending this presentation, participants will gain a comprehensive understanding of the vaccine tech transfer process, its challenges, and best practices for ensuring successful and efficient technology transfer. This knowledge will contribute to the timely availability of high-quality vaccines, ultimately benefiting global public health initiatives.

Biography

Lokender Kaushik, an expert in Quality Management Systems (QMS) and Good Manufacturing Practices (GMP), holds a Master's degree in science with extensive experience in the vaccine industry. He is skilled in leadership, quality audits, CAPA analysis, and regulatory compliance strategy. With a track record of achievements including managing a Bill & Melinda Gates Foundation (BMGF) funded WHO Prequalification (PQ) project, reducing technology transfer timelines by 30%, and setting up Quality Management Systems, Mr. Kaushik is a dynamic and motivated professional. In his current role, he provides high-level strategic and operational regulatory direction, oversees global regulatory submissions, and manages critical issues related to technology transfer and regulatory affairs.



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DISCUSSION ON THE CONCEPT OF IMMUNITY AND HOW HUMAN BODY SYSTEM COULD BUILD IMMUNITY AGAINST COVID 19 AND IT RELEVANCE TO PUBLIC HEALTH EDUCATION

Albert Opoku

Nursing and Midwifery Training College, Ghana

Abstract

Introduction; Every second of day, an enemy of aggressive pathogens (disease causing organisms) such as bacteria, fungi, and viruses crowd on our skin and we stay remarkable healthy most of the time. The body seems to have evolved a single-minded approach to such rivals if you are not with us, you are against us. To implement that stance, it relies heavily on the two intrinsic defense systems and that act both independently and cooperatively to provide resistance or immunity against disease causing pathogens. The above introduction therefore suggest that one's immune system is very vital to fight the new COVID 19 pandemic.

Objectives: The aim of this article was to explain different types of immunity and discuss how the human body could build immunity against COVID 19. This would also serve as a guide for public health education on immunity in this era of COVID 19 pandemic.

Methodology: This is a scoping review to explain different types of immunity and discuss how the human body could build immunity against COVID 19. The LILACS-BIREME, SCIELO, PUBMED, AC-ADEMIA, SCIENCE DOMAIN databases, some textbooks and google scholar were accessed for the study. Scientific papers published in English were reviewed. A total of 35 reports published were identified and reviewed. Twenty five (25) publications meeting the inclusion criterion were selected for this review.

Discussions: The study explained immunity and types of immunity; Innate Immunity and adaptive immunity. Innate immunity may be called nonspecific, does not create memory, and its responses are always the same irrespective of the target. The innate immune system consist of Physical barriers, Defense mechanisms, General immune and complements. Adaptive immunity is very specific as to its target, may involve antibodies, does create memory, and may become more efficient. The source of immunity, give two major categories: genetic immunity and acquired immunity (Adaptive immunity).

Conclusion: From the above discussions it is clear that, there are different dimensions to immunity and therefore various means of acquiring each of the immunity explained. This could be used by health professionals to educate the public on the prevention of the spread of COVID19.

Biography

Albert Opoku is a Fellow of Ghana College of Nurses and Midwives (GCNM) and registered nurse, hold Mphil in Physiology from Kwame Nkrumah University of Science and Technology, had his First degree in Nursing with Psychology from University of Ghana, Legon, He hold a Certificate in Minimally Invasive Surgical Techniques from Shandong Province Foreign Trade Vocational College, Qingdao, China, and currently a PhD candidate in Medical Physiology at Adesh University, Bathinda, India. He has 22 publications in reputable international journals and has received a number of Excellence Awards in Reviewing from Asian Journal of Research in Infectious Diseases, International Research Journal of Public and Environmental Health, International Journal of Research and



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Reports in Hematology, Asian Journal of Immunology and Journal of Pharmaceutical Research International. He has delivered a lot of lectures at both local and international conferences and seminars. Notable among them was when he led a group of health professionals from Ghana to give a country report in Minimally Invasive Surgical Technique at Qingdao, China in 2016. He has also been a Panelist on several television and radio stations for health programmes especially during the COVID 19 Pandemic.



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MOLECULAR CHARACTERIZATION OF THE GENES ENCODING RESISTANCE TO THE BETA- LACTAM FAMILY IN STRAINS OF *KLEBSIELLA PNEUMONIA* ISOLATED AT THE BRAZZAVILLE UNIVERSITY HOSPITAL

Fils Landry Mpelle

Marien Ngouabi University, Brazzaville Congo

Abstract

Objective: Genotypically characterize ESBLs and OXA-48 type carbapenemases in *Klebsiella pneumoniae* isolated in carriage and in infectious processes at the University Hospital of Brazzaville.

Material and methods: The study was carried out for 7 months. Samples (urine, pus and blood cultures) were collected from hospitalized and non-hospitalized patients at the Brazzaville University Hospital. *K. pneumoniae* strains were identified by API20E and by MALDITOF. An antibiogram was carried out on the strains isolated by the disk diffusion method on MH agar. The ESBL and carbapenemase-OXA-48 phenotype were detected by the synergy technique according to CA-SFM and during a reduction in inhibition diameter around the Ertapenem disc and confirmed by PCR and sequencing. MLST *K. pneumoniae* genotyping of OXA-48 strains was carried out.

Results: 23 strains of *K. pneumoniae* were isolated from 23 patients, including 11/23 (47.83%) came from outpatients and 12/23 (52.17%) from inpatients. There PCR revealed that 23/23 (100%) produced ESBLs of which 2/23 (8.69%) were ESBL/OXA-48. The blaSHV gene was the most frequent 20/23 (86.95%), followed by blaCTX-M1 14/23 (60.87%), then blaTEM 10/23 (43.48%), then 2/23 (8.69%) bla-OXA-48. The majority came from urine 82.60%. Sequencing of the amplification products showed that strains positive for blaCTX-M1 were all CTX-M15; 13 enzyme variants were detected for blaSHV. Four types for TEM. The two OXA-48 strains were of the OXA-181 variant not carried by the plasmid. These strains were Co resistant to gentamycin and fluoroquinolone. MLST *K.pneumoniae* OXA-48 revealed two different sequence types known in the literature ST464 and ST15.

Conclusion: This study, revealed the presence of the TEM, SHV, CTXM and OXA-48 genes at worrying frequencies within the *K. pneumoniae* strains from the Brazzaville University Hospital. It proves the need to promote an infection prevention program with regulation of antibiotic therapy in hospitals in Congo Brazzaville.

Biography

Fils Landry Mpelle, Biologist hospital Practitioner; PhD in Microbiology- Molecular Biology bio-Informatics and Immunology from Marien Ngouabi University Brazzaville Congo in 2020 and a Master's degree in Applied immunology and molecular Biology from the Marien Ngouabi University Brazzaville Congo in 2014.

Day 1

Video Presentations



International Conference on Immunology & Vaccines

Rome, Italy | November 18-19, 2024

THE CASE FOR SHEDDING OF SPIKE MRNA GENE THERAPY PRODUCTS CAUSING EXCESS DEATH GLOBALLY

Marian Laderoute

Retired Ottawa, Canada

Abstract

In 2022, Dr. Helene Banoun of France reported anecdotal evidence that the spike mRNA COVID-19 vaccines, carried the risk of shedding putatively of spike-laden exosomes which could transmit microclotting-type disease to the unvaccinated. To explore this possibility further, a novel index of the rate of non-COVID-19 mortality over COVID-19 mortality (per person-years) was calculated for population data for England available from the Office for National Statistics. Remarkably, this index in the unvaccinated mirrored the highs and lows demonstrated in the mRNA vaccinated over time (2021 to end of 2022) and was consistent with shedding from the recently vaccinated causing death. Moreover, as expected for spike-laden exosomes, these mortality index peaks averaged about 3 months and began only after the second dose in the vaccinated with the onset of spike antibodies. The potential impact of shedding during the first 17 months of the almost exclusive Pfizer mRNA vaccine roll out in England was estimated at 551,048 shedding deaths (including 72,034 shedding deaths in the unvaccinated) and about 43,088 direct vaccination deaths (ie., with death onset under 21 days). During this time there were only 87,472 deaths ascribed to COVID-19. The portion of iatrogenic (mRNA vaccine) deaths due to shedding was found to be 90.8 %. Others have estimated over 2021 to 2023 that there were on average 1.7-fold higher vaccine deaths than COVID-19 deaths for 29 countries. Thus, it is proposed that spike mRNA shedding possibly contributed more to excess deaths than SARS-CoV-2 infections, meaning globally the cure was more deadly than the disease. In conclusion this work strongly warrants the absolute and permanent banning of the mRNA gene therapy technology for the purpose of immunization (animals and humans).

Biography

Marian Laderoute received her Ph.D. in Medical Sciences-Immunology from the University of Alberta in 1991 and her post-doctoral work was in molecular carcinogenesis at the Cross Cancer Institute also in Edmonton, Alberta, Canada.

She has made several notable contributions to medical science following her discovery of the 67 kD alpha-fetoprotein (AFP) receptor on tumors and macrophages for her Ph.D. thesis. On June 1, 2024, Dr. Laderoute was the first to publicly reveal population data consistent with the shedding of spike mRNA gene therapy shots causing significant levels of excess deaths worldwide during 2021 to 2023 and as related to mass immunization.



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LUNG EPITHELIAL SIGNALING MEDIATES EARLY VACCINE-INDUCED CD4⁺ T CELL ACTIVATION AND MYCOBACTERIUM TUBERCULOSIS CONTROL

Shibali Das

Washington University in St. Louis, USA

Abstract

Tuberculosis (TB) is one of the leading causes of death due to a single infectious agent. The development of a TB vaccine that induces durable and effective immunity to *Mycobacterium tuberculosis* (*Mtb*) infection is urgently needed. Early and superior *Mtb* control can be induced in *M. bovis* Bacillus Calmette-Guérin (BCG)-vaccinated hosts when the innate immune response is targeted to generate effective vaccine-induced immunity. In the present study, we show that innate activation of DCs is critical for mucosal localization of clonally activated vaccine-induced CD4⁺ T cells in the lung and superior early *Mtb* control. In addition, our study reveals that Th1/Th17 cytokine axis play an important role in superior vaccine-induced immunity. Our studies also show that activation of the nuclear factor kappa-light-chain enhancer of activated B cell (NF- κ B) pathway in lung epithelial cells is critical for the mucosal localization of activated vaccine-induced CD4⁺ T cells for rapid *Mtb* control. Thus, our study provides novel insights into the immune mechanisms that can overcome TB vaccine bottlenecks and provide early rapid *Mtb* control.

Biography

Das has done research focusing on testing different vaccines for efficacy studies. During her previous work with the Washington University School of Medicine, she demonstrated the critical role of Innate lymphoid cells in immunity to tuberculosis and also defined immune landscape that contributed to the disease outcome during tuberculosis. Her current research interest is to define immune mechanisms that contribute to the pathogenesis and progression of myocarditis and heart failure. Specifically, she is interested in studying the role of macrophage subsets in myocarditis and resultant heart failure, elucidating the role of trained immunity against Cardiac disease using cutting edge mice model and development of vaccine strategy against cardiac infections. Her key interests are innate and adaptive immunity, innate lymphoid cells, vaccine induced immunity, infectious diseases and cardiac disease. Her research areas are elucidating the mechanism that contribute to the pathogenesis and progression of myocarditis and heart failure and developing vaccination strategies to improve host immunity.



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