EuroSciCon

DAY 1

Keynote Forum

JOINT EVENT

22nd Edition of International Conference on

Immunology and Evolution of Infectious Diseases

&

12th Edition of International Conference on

Tissue Engineering and Regenerative Medicine

May 10-11, 2018 | Frankfurt, Germany

EuroSciCon

May 10-11, 2018
Frankfurt, Germany

JOINT EVENT
22nd Edition of International Conference on
Immunology and Evolution of Infectious Diseases
&
12th Edition of International Conference on
Tissue Engineering and Regenerative Medicine

Thomas Boldicke, J Transm Dis Immun 2018, Volume 2
DOI: 10.21767/2573-0320-C2-004

INTRABODIES KNOCKING DOWN INTRACELLULAR CANCER ANTIGENS

Thomas Boldicke

Helmholtz Centre for Infection Research, Germany

Intrabodies can be used to target and knock down virtually every protein inside the cell. The knockdown of intracellular cancer antigens by intrabodies is promising. Cancer antigens passing the endoplasmatic reticulum (ER) are inactivated by ER intrabodies retained inside the ER and expressed in the single-chain variable fragment (scFv) format. Cytosolic and nuclear cancer targets are inhibited by neutralizing single domain antibodies which comprises only the variable domain of the heavy chain derived from camels or sharks. This talk will give an overview of *in vivo* targeting of cancer antigens by intrabodies in mouse tumor models and will demonstrate an example of ER intrabodies inhibiting the polysialyltransferases in rhabdyomasarcoma cells in a xenograft tumor mouse model.

Biography

Thomas Boldicke has received his PhD in 1982 at the Max Planck Institute of Molecular Genetics, Berlin. He started his carrier as Postdoc at the German Research Centre for Biotechnology (GBF, Brunswick) in the Department of Genetics and Cell Biology by John Collins. Now he is a Senior Scientist at the Helmholtz Centre for Infection Research and Project Leader for intrabodies. In 2011, he qualified as a Professor in Molecular Biology and Cell Biology at the Technical University of Braunschweig. He is an expert in generating mouse and human hybridomas and in selecting and modifying recombinant antibodies. In the last decade he focused on the construction and characterization of intracellular antibodies. He has published 35 manuscripts.

thomas.boeldicke@helmholtz-hzi.de

EuroSciCon

May 10-11, 2018
Frankfurt, Germany

JOINT EVENT
22nd Edition of International Conference on
Immunology and Evolution of Infectious Diseases
&
12th Edition of International Conference on
Tissue Engineering and Regenerative Medicine

Pascal Rihet, J Transm Dis Immun 2018, Volume 2
DOI: 10.21767/2573-0320-C2-004

FROM GENOME SCANS TO THE IDENTIFICATION OF FUNCTIONAL GENETIC VARIANTS ASSOCIATED WITH MALARIA RESISTANCE

Pascal Rihet

TAGC - Inserm and Aix-Marseille University, France

The contribution of host genetic factors to resistance or susceptibility to *Plasmodium falciparum* malaria has been widely studied. Nevertheless, a few genome scans have been performed, and few of them led to the discovery of loci significant at the genome level and to the identification of functional variants potentially causal. Here we describe loci genetically linked to malaria phenotype at the genome level and genetic variants located within those loci and associated with malaria phenotype in two independent populations. Furthermore, we provide evidence of a cis-regulatory effect of the genetic variants, suggesting that those variants are causal. We mainly focus on genes and genetic variants located within chromosome 6p21, which has been linked to mild malaria. These include TNF and NCR3, which encode a major actor of inflammation and a receptor of natural killer cells involved the cytotoxicity function, respectively. Also, the results are in line with those supporting the role of TNF in malaria on the one hand and allow us to propose a new biological model to explain the association of a cis-regulatory variant of NCR3 with mild malaria, on the other hand. Also, the genetic variation that alters the activation of natural killer cells may influence human malaria resistance.

Biography

Pascal Rihet has a long lasting experience of research in the field of genetics and genomics of infectious diseases. He has mapped malaria and sepsis predisposing genes by using genetic linkage or association approaches. Furthermore, he has identified many variants associated with the disease; most of those genetic variants are located within noncoding regions. He has provided evidence that several variants have a cis-regulatory effect, suggesting that the regulation of gene expression is critical for the pathogenesis. In this way, he has investigated gene expression profiles in patients or in mouse models, and identified a number of genes whose expression is up- or down-regulated before or at the onset of the disease. He was the Deputy Director of the TAGC laboratory. Currently he is the Director of TAGC laboratory. The research scope of the laboratory is Genetics, Genomics and Bioinformatics. He is a Professor of Genomics and Immunology at AMU.

pascal.rihet@univ-amu.fr

EuroSciCon

DAY 2

Keynote Forum

JOINT EVENT

22nd Edition of International Conference on

Immunology and Evolution of Infectious Diseases

&

12th Edition of International Conference on

Tissue Engineering and Regenerative Medicine

May 10-11, 2018 | Frankfurt, Germany

EuroSciCon

May 10-11, 2018
Frankfurt, Germany

JOINT EVENT
22nd Edition of International Conference on
Immunology and Evolution of Infectious Diseases
&
12th Edition of International Conference on
Tissue Engineering and Regenerative Medicine

Huang Wei Ling, J Transm Dis Immun 2018, Volume 2
DOI: 10.21767/2573-0320-C2-004

WHY DO PATIENTS STILL CATCH HOSPITAL INFECTIONS DESPITE THE PRACTICE OF INFECTION PREVENTION AND CONTROL PROGRAMS?

Huang Wei Ling

Medical Acupuncture and Pain Management Clinic, Brazil

Statement of the Problem: Very few publications provide sound scientific data used to determine which components are essential for infection prevention and control (IPC) programs in terms of effectiveness in reducing the risk of infection. In recent years, a range of regional best practice or policy principles have been developed that address what could be considered as core components of IPC programs. However there remains a major gap in relation to the availability of international best practice principles for core components of IPC programs.

Purpose: The purpose of this study was to show why patients still catch hospital infections despite IPC programs. A better understanding of a variety of theories is needed that could explain the physiopathology of diverse diseases described in the medical past history, which are usually disregarded clinically today. A broader view seems to show the necessity of seeing the patient as a whole; not only focusing on the disease in the prevention of these hospital infections.

Methodology: A review of these theories such as those presented by Hippocrates (Natural forces within us are the true healers of disease), as well as others from oriental medicine, which explain that diseases originate from three factors: external (exposure to cold, heat, humidity, wind and dryness), internal (emotional) and dietary.

Findings: Having a broader view of the patient as a whole (*Yin, Yang, Qi*, blood energy and heat retention), we can understand better the formation of hospital infection which is a systemic energy reaction of our body undergoing normal hospital treatment.

Conclusion: To understand better why a patient is still catching hospital infections, despite these IPC programs, we need to broaden our view observing all emotional, environmental and dietary factors, as well as studying the patient's energy situation at the moment of admittance identifying the risk of hospital infection.

Biography

Huang Wei Ling has graduated in Medicine in Brazil, specializing in infectious and parasitic diseases, a General Practitioner and Parenteral and Enteral Medical Nutrition Therapist. Once in charge of the Hospital Infection Control Service of the City of Franca's General Hospital, she was responsible for the control of all prescribed antimicrobial medication, and received an award for the best paper presented at the Brazilian Hospital infection Control Congress in 1998. She was coordinator of both the Infection Control and the Nutritional Support Committee in Sao Joaquim Hospital in Franca, and also worked at the infectious Sexually Transmitted Disease Reference Center. She is the owner of the Medical Acupuncture and Pain Management Clinic, and since 1997 she has been presenting her work worldwide concerning the treatment of various diseases using techniques based on several medical traditions around the world.

weilingmg@gmail.com

EuroSciCon

May 10-11, 2018
Frankfurt, Germany

JOINT EVENT
22nd Edition of International Conference on
Immunology and Evolution of Infectious Diseases
&
12th Edition of International Conference on
Tissue Engineering and Regenerative Medicine

Thomas Grundstrom, J Transm Dis Immun 2018, Volume 2
DOI: 10.21767/2573-0320-C2-004

REGULATION OF DIVERSIFICATION AND AFFINITY MATURATION OF ANTIBODIES

Thomas Grundstrom

Umeå University, Sweden

B-lymphocytes can modify their immunoglobulin (Ig) genes to generate antibodies with a new isotype and enhanced affinity. Activation-induced cytidine deaminase (AID) is the key mutagenic enzyme that initiates these processes. How somatic hypermutation (SH) and class switch recombination (CSR) are targeted and regulated to understand how we achieve good antibodies. The *trans-acting* factors mediating specific targeting of AID and thereby SH and CSR have remained elusive. How AID is recruited was still a big mystery. We show that mutant E2A transcription factor with defect inhibition by the Ca^{2+} sensor protein calmodulin results in reduced B cell receptor (BCR), IL4- plus CD40 ligand-stimulated CSR to IgE. AID is shown to be together with the transcription factors E2A, PAX5 and IRF4 in a complex on key sequences of the *Igh* locus in activated mouse splenic B cells. Calmodulin shows proximity with them after BCR stimulation. Direct protein-protein interactions are shown to enable formation of the complex. BCR signaling reduces binding of the proteins to some of the target sites on the *Igh* locus, and calmodulin resistance of E2A blocks this reduction. Thus, E2A, AID, PAX5 and IRF4 are components of a CSR and SH complex that calmodulin binding redistributes on the *Igh* locus. We present also that initiation of antibody diversification leads to formation of a mutasome, a complex between many proteins that enable repair at high error rate of the uracils made by AID on Ig genes but not on most other genes. We show also that BCR activation, which signals end of successful SH, reduces interactions between some proteins in the complex and increases other interactions in the complex with varying kinetics. Furthermore, we show increased localization of SH and CSR coupled proteins on switch regions of the *Igh* locus upon SH/CSR and that BCR signaling differentially change the localization.

Biography

Thomas Grundström has completed his Doctorate at Umeå University in 1981 and his Medical degree in 1982. He was a Postdoc during 1982-1984 in the laboratory of Professor Pierre Chambon, Institut de Chimie Biologique, Strasbourg, France, where he characterized the first discovered enhancer of transcription. He is a Professor at the Department of Molecular Biology at Umeå University since 1994. He has been studying Ca^{2+} sensor proteins and eukaryotic transcription and discovered the first direct Ca^{2+}/calmodulin inhibition of a transcription factor. He has characterized the Ca^{2+} regulation of many transcription factors and other regulatory proteins with a main focus on the immune system. He is presently studying regulation of production of antibodies. He studies how somatic hypermutation (SH) and class switch recombination (CSR) are targeted and the regulation of the protein complex that performs SH and CSR.

Thomas.Grundstrom@umu.se

EuroSciCon

May 10-11, 2018
Frankfurt, Germany

JOINT EVENT
22nd Edition of International Conference on
Immunology and Evolution of Infectious Diseases
&
12th Edition of International Conference on
Tissue Engineering and Regenerative Medicine

Sasha Berdichevski, J Transm Dis Immun 2018, Volume 2
DOI: 10.21767/2573-0320-C2-004

DESIGNING SCAFFOLDS FOR TISSUE ENGINEERING: 3D GEOMETRY-FUNCTION RELATIONSHIP

Sasha Berdichevski

University of Cambridge, UK

One of the main goals in producing engineered tissues at clinically relevant dimensions is creating perusable vascular networks, since cell viability and function cannot be sustained through diffusion alone. Therefore a great deal of research in the field of regenerative medicine has been devoted to establish *in vitro* pre-vascularization approaches. In this context, we propose to create capillary-like networks using human umbilical cord endothelial cells, cultured with human osteoblasts, as these cells were demonstrated to have both direct and indirect pro-vasculogenic effects, within freeze-dried collagen scaffolds with tailored pore architecture. We guided scaffold pore architecture by manipulation of the freeze-drying conditions; producing porous scaffolds with randomly oriented (isotropic) or uniaxially aligned (anisotropic) pore architectures. We characterized the scaffolds' structural, permeability and mechanical properties and showed that pore architecture affected the invasion, morphology and self-organization of the endothelial cells, in both mono- and co-cultures. Results showed that cell proliferation and metabolic activity were affected by pore architecture as well. Pore anisotropy promoted more uniform cell infiltration deeper within the scaffold, and improved cell organization into multi-cellular vessel-like networks. Co-culture conditions further improved the network quality. We suggest that deeper cell infiltration, along with more efficient medium perfusion within the anisotropic scaffolds account for these findings. However, the exact mechanism and conditions for optimal 3D vascular network formation as function of pore architecture have yet to be established.

Biography

Sasha Berdichevski is a Post-doctoral Research Associate in Engineering Department at University of Cambridge, UK. She has obtained a Blavatnik Fellowship by the Blavatnik Family Foundation, British Council and University of Cambridge, and currently she holds a Marie Curie Fellowship. She has been awarded as outstanding Researcher in Engineering and Science Award and Prize for Excellence in Nano-science and Nanotechnology during her PhD in the Technion, Israel. She has published her research in leading journal papers, and co-authored publications in three books. Her research interests include "Biomaterials, tissue engineering, scaffold-tissue/cell interactions, and scaffolds' 3D geometry function relationship".

sashenka125@gmail.com

EuroSciCon

May 10-11, 2018
Frankfurt, Germany

JOINT EVENT
22nd Edition of International Conference on
Immunology and Evolution of Infectious Diseases
&
12th Edition of International Conference on
Tissue Engineering and Regenerative Medicine

Roman Bauer, J Transm Dis Immun 2018, Volume 2
DOI: 10.21767/2573-0320-C2-004

COMPUTATIONAL MODELLING OF NEURAL TISSUE GROWTH

Roman Bauer

Newcastle University, UK

Biological tissue often exhibits extraordinary complexity. For example, neural tissue comprises large numbers of neurons with cell-type specific axonal and dendritic arborisation, highly structured synaptic connectivity, and fine-tuned electrical activity. A better understanding of how such tissue complexity develops is often essential for tissue engineering purposes. To this end, author will present some of his computational models of neural tissue development, demonstrating how complex structure and function can be generated based solely on simple genetic rules. These multi-scale models comprise intracellular as well as extracellular dynamics in a detailed, physical 3D environment. In particular, author will elaborate on computational models of cortical and retinal structure and function, ranging across different spatial scales. By modelling the biological self-organization of such tissue, predictions are made and so novel hypotheses are generated, which can be experimentally validated. Moreover, these models can inform and guide tissue engineering protocols. Finally, author will discuss modern computational approaches, including the BioDynamo software, which is a collaborative project with project partner CERN Openlab. Overall, author will emphasize the importance of computer models as a tool to advance tissue engineering approaches.

Biography

Roman Bauer is an MRC Research Fellow at Institute of Neuroscience-Newcastle University, with joint affiliation with the School of Computing. His research involves computational models to better understand how neural tissue evolves during development. He received his Bachelor's and Master's Degree in Computational Science and Engineering from ETH Zurich, Switzerland. Afterwards, he did his Doctoral studies at Institute for Neuroinformatics (INI)-ETH and University Zurich, working on simulations of cortical development. After Postdoctoral work at Newcastle University from 2013 to 2016, he took up a prestigious MRC fellowship. His research interests include "Neural development, neural degeneration, neural disorders, gene regulatory networks and cryopreservation".

roman.bauer@ncl.ac.uk