

# Principles of Infection Biology

Granavolden Gjæstgiveri, Gran, Norway June 5<sup>th</sup> – June 9<sup>th</sup> - 2023

A Course Arranged By



National Graduate School in Infection Biology and Antimicrobials

#### **Organizing Committee:**

Michael Koomey, University of Oslo, IBA Director Debra Milton, Umeå University, NDPIA Director Tone Tønjum, Oslo University Hospital, IBA Marte Singsås Dragset, Norwegian University of Technology and Science, IBA Morten Kjos, Norwegian University of Life Science, IBA Kurt Hanevik, University of Bergen, IBA Navdeep Kaur Brar University of Oslo, IBA Tara Daughton University of Oslo, IBA Project Coordinator

Picture on front page: iStock

Production: Tara Daughton (IBA Project Coordinator)

# Welcome to the Principles of Infection Biology Course!

With this course, IBA aims to provide a thorough background in the basic concepts behind infection biology spanning the disciplines of host-microbe interaction and infections caused by bacteria, eukaryotic microbes and viruses. IBA wants to ensure that all IBA PhD members acquire a common knowledge base and start building a national and international scientific network early in their PhD progression. Participation in the course is therefore mandatory for all IBA PhD students that are early in their PhD education. The course is organized by the Norwegian PhD School in Infection Biology and Antimicrobials (IBA) and is open to IBA members and members of the Swedish National Doctoral Programme in Infections and Antibiotics (NDPIA).

You can find review articles for the course lectures, lecture handouts, a pdf file of the program and other files here: <u>PIBs 2023</u>

#### Hyperlinks to navigate in this pdf document:

Schedule Monday Schedule Tuesday Schedule Wednesday Schedule Thursday Schedule Friday Biosketches lecturers Meet the attendees Poster abstracts students Poster instructions Poster groups Course evaluation

All lectures are at Granavolden Gjæstgiveri -Poster group I will be in room Poster group II will be in room

# Monday June 5, 2023

#### Session 1: Introduction, Basic Concepts and History

Chair Mike Koomey

9 -	Registration and poster mounting
10:00-10:30	Welcome / Participant Introductions
10:30-11:15	<b>Concepts of Virulence - the Staphylococcus aureus model</b> José R Penadés, Department of Infectious Disease, Imperial College London, United Kingdom. <u>Biosketch page 9</u> .
11:15-11:30	Questions and Discussion with José R Penadés
11:30-12:15	<b>Highlights of Bacteriology</b> José R Penadés, Department of Infectious Disease, Imperial College London, United Kingdom <u>Biosketch page 9</u> .
12:15-12:30	Questions and Discussion with José R Penadés
12:30-13.30	Lunch
13:30-14:15	Studying Microbial Pathogenesis - Listeria monocytogenes as a model Anat Herskovits, The Shmunis School of Biomedicine and Cancer Research, Tel Aviv Universy, Tel Aviv, Israel Change: <u>Biosketch page</u> <u>10</u> .
14:15-14:30	Questions and Discussion with Anat Herskovits
14:30-14:45	Coffee
14:45-15:30	Animal Models for Drug Development Against Respiratory Infectious diseases (online) Priscille Brodin, University of Lille, Institut Pasteur de Lille, France. <u>Biosketch page 11</u> .
15:30-15.45	Questions and Discussion with Priscille Brodin
	Session 2: Student invited speaker session Chair: Navdeep Kaur Brar
15:45-16:30	Introduction to infectious Disease Modelling and its application Nicky McCreesh, London School of Hygiene & Tropical Medicine, CMMID. <u>Biosketch page</u>

16:30-16:45	Questions and Discussion with Nicky McCreesh
16:45-17:00	Understanding bacterial pathogenesis to address antibiotic resistance Anders Håkannson, Experimental Infection Medicine, Lund University, Sweden. Change: <u>Biosketch page 12</u> .
17:00-17:15	Questions and Discussion with Anders Håkannson
	Tuesday June 6, 2023
	Session 3: Bacterial Pathogens Chair Morten Kjos
8:30-9:15	<b>Studying host-pathogen interactions using single-cell genomics</b> Anotine-Emmanuel Saliba, Helmholtz Institute for RNA-based infection research, Würzburg, Germany Change: <u>Biosketch page 13</u> .
9:15-9:30	Questions and Discussion with Anotine-Emmanuel Saliba
9:30-10:15	Spatial and temporal regulation of Type 6 Secretion System dynamics during interactions with bacterial and eukaryotic cells Marek Basler, The Center for Molecular Life Sciences, University of Basel. <u>Biosketch page</u>
10:15-10:45	Questions and Discussion with Marek Basler
10:45-11:00	Break
11:00-12:00	Poster Session I (10 min presentation + 5 min questions in 2 parallel groups). Group 1: St.Petri. Group 2: Gregerstua.
12:00-12:30	Free poster viewing of the "posters of the day"
12:30-13:15	Lunch
13:15 – 14:15	Historic walk around Granavolden

#### **Session 4: Bacterial Pathogens continued**

Chair: José R Penadés

14:30-15:15	Molecular Genetic Approaches to Unveil the Secrets of Chlamydia's Intracellular Life Barbara Sixt, Department of Molecular Biology, Umeå University, Sweden. Change: <u>Biosketch page 14</u> .
15:15-15:30	Questions and Discussion with Barbara Sixt
15:30-15:45	Break
15:45-16:30	<b>Enterococcal virulence (online)</b> Kimberly Kline, Singapore Centre for Environmental Life Sciencest, University of Geneva <u>Biosketch page 15</u> .
16:30-16:45	Question and discussion with Kimberly Kline

# Wednesday June 7, 2023

Session 5: Virology Chair Debra Milton

8:30-9:15	Concepts of Virology - 12 million solutions in 20 moves? (online)
	Hans H. Hirsch, Department Biomedicine, University of Basel and
	University Hospital Basel, Basel, Switzerland. Biosketch page 16.

- 09:15-09:30 Questions and Discussion with Hans H. Hirsch
- 9:30-09:45 Break
- 09:45-10:45 Poster Session II Group 1: St.Petri. Group 2: Gregerstua
- 10:45-11:15 Free poster viewing of the "posters of the day"
- 11:15-11:30 Break
- 11:30-12:15InfluenzaRebecca Jane Cox, Influenza Centre, Department of Clinical Science,<br/>University of Bergen, Bergen, Norway. Biosketch page 17.
- 12:15-12:30 Questions and Discussion with Rebecca Jane Cox
- 12:30-14:30 Lunch

#### Session 6: Virology continued Chair Tone Tønjum

14:30-15:15	<b>SARS-CoV-2</b> Rebecca Jane Cox, Influenza Centre, Department of Clinical Science, University of Bergen, Bergen, Norway. <u>Biosketch page 18</u> .
15:15-15:30	Questions and Discussion with Rebecca Jane Cox
15:30-15:45	Break
15:45-16:30	Neurotropic Flaviviruses and tools to study them Anna Överby Wernstedt, Department of Clinical Microbiologgy, Umeå Universit Sweden <u>Biosketch page 19</u> .
16:30-16:45	Questions and Discussion with Anne Överby Wernstedt
18:30-21:00	Course dinner –
	Thursday June 8, 2023 Session 7: Host Response Chair Marte Singås Dragset
8:30-9:15	Detection of Pathogen Infection by the Host (online) Clare Bryant, Department of Medicine, Department of Veterinary Medicine, University of Cambridge, United Kingdom. <u>Biosketch page</u> <u>20</u> .
09:15-09:30	Questions and Discussion with Clare Bryant
9:30-09:45	Break
09:45-10:30	Host Response to Infection Charlotte Odendall, School of Immunology & Microbial Sciences, Kings College London. <u>Biosketch page 21</u> .
10:30-10:45	Questions and Discussion with Charlotte Odendall
10:45-11:00	Break
11:00-11:45	Host response and infection models

Pere-Joan Cardona, Unitat de Tuberculosi Experimental, Institut Germans Trias i Pujol, Barcelona, Spain. <u>Biosketch page 22</u>.

- 11:45-12:00 Questions and Discussion with Pere-Joan Cardona
- 12:00-13:00 Lunch

#### **Session 8: Parasitology**

#### **Chair Kurt Hanevik**

- 13:00-13:45Parasites why are they cool?Staffan Svärd, Department of Cell and Molecular Biology, Uppsala<br/>University, Sweden.<br/>Biosketch p. 23.
- 13:45-14:00 Questions and Discussion with Staffan Svärd
- 14:00-14:15 Break
- 14:15-15:00Molecular mechanisms of Plasmodium motility and invasion -<br/>perspectives on drug design<br/>Inari Kursula, Department of Biomedicine, University of Bergen,<br/>Norway and Faculty of Biochemistry and Molecular Medicine,<br/>University of Oulu, Finland. Biosketch page 24.
- 15:00-15:15 Questions and Discussion with Inari Kursula
- 15:15-15:30 Break

#### Session 9: Human Infection Studies Chair Mike Koomey

- 15:30-16:15Human Infection StudiesRobert Read, Infectious Disease and Clinical Microbiology, University<br/>of Southampton, UK.<br/>Biosketch page 26.
- 16:15-16:30 Questions and Discussion with Robert Read
- 16:30-16:45 Break
- 16:45-17:45 Poster Session III

Group 1: St.Petri. Group 2: Gregerstua

17:45-18:15 Free poster viewing of the "posters of the day"

## Friday June 9, 2023

Session 10: Microbiota Chair Mike Koomey

08:30-09:15	<b>Gut Microbiota and Metabolic Diseases (online)</b> Valentina Tremaroli, Department of Molecular and Clinical Medicine, University of Gothenburg, Sweden. <u>Biosketch page 27</u> .
09:15-09:30	Questions and Discussion with Valentina Tremaroli
09:30-09:45	Break
09:45-10:30	<b>Gut Microbiota and Health During Lifespan</b> Anne Salonen, Human Microbiome Research Program, Faculty of Medicine, University of Helsinki, Finland. <u>Biosketch p. 28</u> .

- 10:30-10.45 Questions and Discussion with Anne Salonen
- 10:45-11:15 Concluding remarks, <u>evaluation</u>, and course certificates
- 11:30-15:00 Lunch (to bring or eat in)



# José R Penadés

Professor Department of Infectious Disease, Imperial College London, United Kingdom

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José R Penadés is Director of the MRC Centre for Molecular Bacteriology and Infection, and Chair of Microbiology at Imperial College. Research in the Penadés lab has focused on the molecular basis of bacterial virulence, deciphering the mechanisms underlying the transfer of different mobile genetic elements (MGEs) involved in pathogenesis. Seminal contributions from his lab include the identification of a widespread family of MGEs, the Phage-Inducible Chromosomal Islands (PICIs), and discovery of the most powerful mode of phage transduction described to date: lateral transduction. Prior to joining the Imperial College, José was Professor of Microbiology at the University of Glasgow.

More information: <u>https://www.imperial.ac.uk/people/j.penades</u>



# **Anat Herskovits**

#### Professor

The Shmunis School of Biomedicine and Cancer Research, the George S. Wise Faculty of Life Sciences, Tel Aviv Universty, Tel Aviv, Israel anathe@tauex.tau.ac.il

Our lab of bacterial pathogenesis and cell host interactions is located at Tel Aviv University. We are interested in how intracellular bacterial pathogens manage to survive the mammalian niche and cause infection. We study the bacterium Listeria monocytogenes, which is a human facultative intracellular pathogen. We specifically ask how is L. monocytogenes metabolically adapted to grow within mammalian cells? How does it sense host derived metabolites and use this information to regulate virulence gene expression? We also ask, how does L. monocytogenes interact with its inhabiting prophages in the course of mammalian infection? Lysogenic phages are parasites, potential 'molecular time bombs', yet during mammalian infection they cooperate with their bacterial host. We study phage adaptive behaviours that support the survival of pathogens within the mammalian niche. Our lab combines various computational, biochemical and genetic approaches to decipher these mechanisms.

Short CV: https://www.herskovitslab.sites.tau.ac.il/anat

More information: https://en-lifesci.tau.ac.il/profile/anathe



# **Priscille Brodin**

Research Director University of Lille, Inserm, Institut Pasteur de Lille, France priscille.brodin@inserm.fr

P. Brodin has been studying *Myobacterium tuberculosis* -host relationships over the last 20 years. P. Brodin was recipient of the ERC grant INTRACELLTB and has been involved in several EU consortium projects (TB-VIR, MM4TB, CycloNHit, MTI4MDRTB and ERA4TB) around axes focused on *M. tuberculosis* colonization of host cells and TB drug discovery. She co-authors more than 100 publications and is inventor on several patents including Telacebec. She was Laureate of the Sanofi Junior Prize and the EMBO Young Investigator Program.

More information: <u>https://www.ciil.fr/teams/chemical-genomics-of-intracellular-mycobacteria-cgim</u>



## **Nicky McCreesh**

Assistant Professor London School of Hygiene & Tropical Medicine, CMMID

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Research field/expertise: I am an epidemiologist and mathematical modeller. I work primarily on tuberculosis and HIV, and am interested in understanding the role of heterogeneity (e.g. in contact patterns, infectiousness, natural history, and health seeking behaviour) in the transmission and control of TB. My research focuses on high TB burden settings, and sub-Saharan Africa in particular. I also work on methods development related to the calibration and analysis of complex models.

Short CV: I am an Assistant Professor in Infectious Disease Biology, based at the London School of Hygiene & Tropical Medicine. My background is in epidemiology, mathematics, and anthropology. Prior to working on TB and HIV, I have worked on understanding the impact of climate change on schistosomiasis and malaria, and respondent-driven sampling methodology.

More information: https://www.lshtm.ac.uk/aboutus/people/mccreesh.nicky



## Anders P Håkansan

Professor Experimental Infection Medicine, Lund University, Sweden.

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My laboratory focuses on understanding host-pathogen interactions, with emphasis on respiratory pathogens, using this knowledge to develop novel preventive and therapeutic strategies to protect against bacterial infection. Our work focuses on both the factors used by bacteria to establish colonization and cause disease and how this is counteracted by the host inflammatory response to colonization and infection. We are working on several major research projects, including developing probiotics and vaccines as well as studying the adjuvant activity of a human milk protein-lipid complex in sensitizing bacteria to a broad range of common antibiotics to provide novel therapeutic strategies against respiratory and other infections with antibiotic-resistant bacteria.

More information: https://portal.research.lu.se/en/persons/anders-p-håkansson



# **Antoine-Emmanuel Saliba**

**Group Leader** 

Helmholtz Institute for RNA-based infection research (HIRI), Würzburg, Germany

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Research Goals/Expertise:

Novel technological leaps are enabling scientists to chart a comprehensive map of the cells across the body, to define their states and to determine their responses to infectious agents in unprecedented detail. Yet how a host either contains the spread of a pathogen, or subsets of pathogens escape host immune surveillance still remains poorly understood.

Emmanuel Saliba's group investigates RNA and RNA processing as a read-out to determine the cell state of both hosts and pathogens at the single-cell level. Their work, involving pathogens such as Salmonella and respiratory viruses with cell culture models, organoids, and clinical samples, analyses, categorizes and clusters cells to decipher cellular microenvironments and understand infectious disease progression.

They employ single-cell RNA seq, spatial transcriptomics, and RNA imaging to capture the RNA transcript census expressed within a host and pathogen. Further temporal single-cell analysis using RNA metabolic labelling provides insights into the history of a cell. These high-resolution analyses potentially enable the prediction of cell behavior and can unlock gene regulatory networks underlying infectious processes. This work is key in the development of precision diagnostics and therapeutics.

#### Short CV:

A.-Emmanuel Saliba studied biochemical engineering at INSA Toulouse (France) and received his PhD in the research group of Jean-Louis Viovy at the Institut Curie (Paris, France). During this time, he developed microfluid-based systems for sorting and analysing rare subpopulations of cancer cells. He then worked as an EIPOD fellow at the European Molecular Biology Laboratory (Heidelberg, Germany) and was involved in the development of innovative systems biology methods for the analysis of protein-lipid interactions. He then joined Jörg Vogel's laboratory in Würzburg as a senior postdoctoral fellow and established single-cell RNA-sequencing to track the fate of individual cells during an infection with Salmonella enterica. Since 2017 he has led the Single-cell Analysis group at HIRI.

More information: https://www.helmholtz-hiri.de/



## **Marek Basler**

Head of Research Group

The Center for Molecular Life Sciences, University of Basel

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My lab at the Biozentrum of the University of Basel has provided seminal contributions to our understanding of T6SS assembly. We have solved structures of many of the critical core components of the T6SS by cryo-electron microscopy and explained how T6SS, and other contractile nanomachines related to bacteriophages, deliver macromolecules across the target cell membranes. Our structural studies showed that T6SS generates unprecedented amount of energy that it uses to puncture membranes of neighboring cells and deliver large protein effectors.

We have significantly advanced live-cell imaging techniques to describe quantitatively T6SS assembly in many bacterial species. We mapped dynamics and composition of T6SS subcomplexes and provided novel insights into T6SS assembly and regulation. In addition, we devised novel imaging assays to show translocation of T6SS effectors into neighboring cells. We showed how delivery of a cocktail of T6SS effectors facilitates target cell lysis, which in turn enables efficient DNA uptake by competent bacteria. Since T6SS is involved in bacterial cell-cell interactions as well as interactions with host cells, my work has impacted research in many areas of microbiology, such as pathogenesis or studies of polymicrobial communities.

URL for web site: https://www.biozentrum.unibas.ch/basler/



Photo by Mattias Pettersson

#### **Barbara Sixt**

Research group leader

The Laboratory for Molecular Infection Medicine Sweden (MIMS), Department of Molecular Biology, Umeå University, Sweden

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Research Goals/Expertise:

I am a passionate researcher with keen interest in the field of host-pathogen interaction and cell-autonomous immunity. Cell-autonomous immunity is an evolutionary ancient branch of immunity that provides protection against intracellular pathogens. It acts at the level of each individual infected cell and comprises a set of cellular defense responses that can limit replication and spread of the pathogen. Together with my research team and collaborators, I strive to uncover the hidden protective potential of pathogen-suppressed cellular defense pathways, to identify the molecular determinants of host defense and pathogenic countermeasures, and to find means to disturb their balance to the benefit of the host. Our long-term vision is to find novel ways to exploit the natural defenses of human cells to fight infectious diseases. Our current research focuses primarily on the obligate intracellular bacterial pathogen *Chlamydia trachomatis*, a common cause of sexually-transmitted diseases and blinding eye infections.

More information: https://sixtlab.org/cv-barbara-sixt/



# **Kimberly Kline**

Professor

University of Geneva, Singapore Centre for Environmental Life Sciences Engineering (SCELSE), Visiting Scientist

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Kim earned her PhD from Northwestern University and did her post-doc in the laboratory of Scott Hultgren at Washington University in St. Louis in collaboration with Birgitta Henriques-Normark and Staffan Normark at the Karolinska Institute in Stockholm. During that time, Kim was an American Heart Association Fellow, Carl Tryggers Fellow, and NIH K99 recipient. In 2011, Kim received and NRF Fellowship in Singapore and joined the faculty at Nanyang Technological University (NTU) joined SCELSE, a biofilm-focused research institute, as a Principal Investigator. In 2014 she was awarded an ICAAC Young Investigator Award by the American Society of Microbiology, in 2016 she received the Nanyang Teaching Award, and she was promoted to Professor in 2021. In 2022, Kim joined the University of Geneva. In her free time, Kim enjoys reading, cooking, her pugs, and exploring Switzerland.



## Hans H. Hirsch

MD, Professor

Department of Biomedicine, University of Basel and University Hospital Basel, Switzerland

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Hans H. Hirsch studied medicine at the Albert-Ludwigs-University in Freiburg (Germany) and biochemistry at the Oregon State University (OR, USA). He then specialized in internal medicine, infectious diseases, and medical microbiology at the University of Basel and the University Hospital Basel. In 2004, the Medical Faculty of the University of Basel appointed him as tenure track Professor with promotion to full professor in 2017. Currently, Dr. Hirsch is director of the Clinical Virology diagnostic laboratory and he holds a senior clinical appointment as consultant in Infectious Diseases at the University Hospital Basel. Dr. Hirsch heads the research group "Transplantation & Clinical Virology" and has (co-)authored more than 200 peer-reviewed papers. Dr Hirsch has contributed to relevant textbooks, conferences and guidelines in infectious diseases, transplantation, and virology including the recent 7<sup>th</sup> edition of Fields Virology.

https://shop.lww.com/Fields-Virology--DNA-Viruses/p/9781975112578.

Dr Hirsch has been Co-Editor-in-Chief for Transplant Infectious Disease from 2004 – 2020, associate editor or editorial board member of a number of journals incl. Transplantation, Am J Transplantation, Clin Infectious Diseases, J Virology, J Clin Virology, J Clin Microbiology, and ad-hoc reviewer for several high-impact journals.

#### More information:

https://biomedizin.unibas.ch/en/research/research-groups/hirsch-lab/

https://www.unispital-basel.ch/zuweiser/services/aerztinnen-und-aerzte-von-a-bisz/h/pers/hans-hirsch/

Lecture date: Wednesday 07.06.2023



## **Rebecca Jane Cox**

Professor

Department of Clinical Science, Influenza center, University of Bergen, Norway

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Research Goals/Expertise:

Rebecca's research focuses on development and evaluation of influenza and COVID vaccine with particular focus on human immune responses to infection and vaccination.

#### Short CV:

Rebecca Cox is professor in medical virology and head of the Influenza Centre at the University of Bergen, Norway leading a team of 20 scientists. Rebecca Cox completed her PhD in 1995 at the London Hospital Medical College, University of London, UK before post doc positions at Guys Hospital, UK and the University of Bergen, Norway. She has >25 years of experience of influenza work particularly in development and evaluation of influenza vaccines. She has serves as advisor to the WHO SAGE Immunization Working Group on Influenza, the Norwegian epidemic and pandemic committee and the European Medicines Agency (EMA) . She had headed the Bergen COVID-19 Research Group and conducted studies on SARS Cov-2 infection in household and healthcare settings and investigated the immune response after infection and long-term complications such as long COVID. Her research focuses on development and evaluation of influenza and COVID vaccine with particular focus on human immune responses to infection and vaccination. She is deputy chair of Influenza and Other Respiratory Viruses and senior editor for the journal Influenza and Other Respiratory Viruses. She is author of more than 140 peer-reviewed papers and regularly contributes to the public debate on Influenza, COVID and vaccines through multi-media channels.

More information: <u>https://www.uib.no/en/persons/Rebecca.Jane.Cox.Brokstad</u>

Lecture date: Wednesday 07.06.2023



# Anna Överby Wernstedt

Professor

Department of Clinical Microbiology, Deputy Director of the Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå University

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Anna Överby is a professor at the department of Clinical Microbiology at Umeå University, and deputy director of MIMS, (The Laboratory for Molecular Infection Medicine Sweden), Umeå Unviersiy. She graduated with a Master of Science in Engineering Biology at Umeå University in 2003, and in 2007 she completed her PhD at the Karolinska Institute, Stockholm where she studied the assembly of bunyaviruses. Thereafter, Anna moved to Freiburg, Germany for a post doc in the laboratory of Friedemann Weber were the focus was tick-borne encephalitis virus (TBEV) interaction with the innate immune system. In 2011 she started her own research group within the Laboratory for Molecular Infection Medicine Sweden (MIMS), one of the Nordic EMBL nodes at Umeå University. Her research team has characterized different aspects of what determines tick-borne encephalitis virus pathogenicity and tropism in mice in particular how the local type I interferon response within the central nervous system restricts viral replication. Her lab recently developed tools to study virus infection in mice brain by developing whole brain imaging and combine that with transcriptomics.



## **Clare Bryant**

Professor of Innate Immunity

University of Cambridge, Department of Medicine, Cambridge, United Kingdom

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Research field/Expertise:

Clare Bryant is Professor of Innate Immunity at the Departments of Medicine and Veterinary Medicine in the University of Cambridge. She studies innate immune cell signalling in response to Pathogen Associated Molecular Pattern Receptor (PRR) activation during bacterial infection using cutting edge multi-disciplinary approaches (collaborating with mathematicians, physicists, physical chemists and structural biologists) to answer fundamental questions about host-pathogen interactions and how to modify them therapeutically. She also applies these innovative approaches to study PRR-induced inflammatory signalling in chronic diseases of humans and animals. In particularly her work using super resolution and single molecule fluorescent imaging approaches to study Toll-like receptor and NOD-like receptor signalling within cells have revealed novel mechanisms in how these receptors signal. She has been on secondments in Genentech and GSK, has extensive collaborations with many pharmaceutical companies, is on the scientific advisory board of several biotech companies, has a drug discovery project with Apollo Therapeutics and helped found the natural product company Polypharmakos. During the COVID-19 pandemic she founded, and still runs, the Inflammazoom international seminar series and was elected as a Fellow of the British Pharmacology Society in 2018.

Short CV: https://www.vet.cam.ac.uk/directory/ceb27%40cam.ac.uk

More information: <a href="https://www.vet.cam.ac.uk/directory/ceb27%40cam.ac.uk">https://www.vet.cam.ac.uk/directory/ceb27%40cam.ac.uk</a>



## **Charlotte Odendall**

PhD

School of Immunology & Microbial Sciences, Kings College, London

Charlotte Odendall received her PhD in Cellular Microbiology from Imperial College London, where her work focused on *Salmonella* pathogenesis. Charlotte then moved to Boston Children's Hospital/Harvard Medical School to study innate sensing, in particular the regulation of type I and III interferons (IFNs). Charlotte then started her lab as a Sir Henry Dale Fellow funded by the Wellcome Trust and Royal Society at King's College London, where she is now a Senior Lecturer. The Odendall lab investigates both sides of the interaction between pathogens and their host. Charlotte is especially interested in uncovering the anti-bacterial functions of type I and III IFNs, and how enteric bacterial pathogens manipulate IFN signaling pathways to promote virulence. In particular, the Odendall lab identified that a family of Shigella virulence factors blocks IFN receptor signaling, favouring bacterial infection. Other projects include identification of IFN stimulated genes that target intracellular bacteria.

More information: <u>https://www.kcl.ac.uk/people/charlotte-odendall</u> and <u>https://www.kcl.ac.uk/research/odendall-group</u>



Research Goals/Expertise:

## Pere-Joan Cardona

Professor of Microbiology

Department of Genetics and Microbiology, Universitat Autònoma de Barcelona (UAB), and Hospital Universitari "Germans Trias i Pujol", Spain

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Due to my previous experience developing a humanized therapy for endocarditis experimental infection in rabbits soon after I finished my MD, I was challenged to generate an experimental TB model when I earned my internship in Clinical Microbiology in the "Hospital Universitary Germans Trias i Pujol" (HUGTiP). That's why I could spent 6 months as Visitor Scientist in the Mycobacterial Research Laboratories, with Prof. Ian Orme (1995-1996) to start my research. When I came back, we built a small replica of Ian Orme's lab. This was the origin of the "Experimental Tuberculosis Unit" (UTE) at the Research Institute Germans Trias i Pujol (IGTP) (1997). Since then, I started my work mainly in the C57BI/6 model to evaluate humoral response against glycolipids (earning my PhD), the study of intragranulomatous necrosis in several mice strains, and artificially induced with LPS (through the "Schwartzman phenomen"), to finally discover the role of foamy macrophages (FM) carrying non-replicating bacilli. This finding led to elaborate the "dynamic hypothesis" to understand the phenomenon of latent TB infection (LTBI) as a process of constant endogenous reinfection supporting the empirical use of INH in chemoprophylaxis, epidemiological data and "TB Spectrum". As a consequence, I designed the therapeutic vaccine RUTI, based in the inoculation of nanoparticles of fragmented Mtb bacilli grown in stressful conditions to trigger immune surveillance of non-replicating bacilli. This let me to the tech transfer world cofounding Archivel Farma and becoming CSO and CMO. Due to the emergence of MDR TB, the concept of RUTI vaccination with chemotherapy as a therapy against active TB has been undertaken. Nowadays, a clinical trial is ongoing in India (under the STRITUVAD Horizon 2020 Consortium) and one about to start in Argentina. The one running in Ukraine was abruptly terminated after the brutal aggression that is still ongoing. In 2009 I also started to work in a new question: How LTBI turns to active TB, after the observation of the granuloma encapsulation in the minipig model. I decided to work with

C3HeB/FeJ finding that a quick infiltration of neutrophiles was key, leading to a fast enlargement of the lesions to overcome the encapsulation process and induce liquefaction of the intragranulomatous necrosis. With the help of a group of physicists we could built the "bubble model" through "in silico" modelling, which has been demonstrated already in the minipig model and in NHP. The "bubble model" supported Host-derived therapies, after demonstrating the usefulness of AINEs, and raised the strategy of potentiating a Treg response in order to balance the Th17 one, using the oral administration of heat-killed environmental, a concept transferred to a new spin-off, Manremyc.

By 2015 I started to work in coevolution, exploring the *Drosophila* model infected with *M*. *marinum*. Publications are expected to be available from 2022, together with the latent Mtb model in *Drosophila*.

On 2021, I was appointed to be the Head of the Microbiology Department at the HUGTIP. Thanks to that I've incorporated a clinical team able to work in TB in the field of new diagnostic tools, and also to help the Public Health System by starting a new project on sequencing all Mtb strains in Catalonia to refine TB active surveillance.

Short CV: https://www.validate-network.org/people/pere-joan-cardona

Lecture date: <u>Thursday 08.06.2023</u>



## Staffan Svärd

Professor in Microbiology

Department of Cell and Molecular Biology, Uppsala University, Sweden staffan.svard@icm.uu.se

Research field/Expertise:

*Giardia intestinalis* is a major contributor to the enormous burden of diarrheal diseases with 180 million symptomatic human infections per year. The focus of our research group is to understand the biology and pathogenesis of *Giardia* but also other diarrhea-inducing protozoa.

We do this by using several different approaches that are complementary. Genome sequencing of different isolates is one approach, which is complemented by gene expression analyses using proteomics, and RNA sequencing. Gene expression in *Giardia* is studied during different conditions like host cell interactions, differentiation and stress-conditions. This has identified several new potential virulence genes but also taught us much about the biology of the parasite. Potential virulence factors are studied further in our *in vitro* system with cell lines and enteroids that mimics host cell interaction in the human small intestine. These *in vitro* studies are complemented by studies using giardiasis patient material collected in Sweden, Bolivia and Mocambique. We try to study differences and similarities in the genomes of the parasites and we also study differences in the immune responses to the parasite. We also study the biology of other Diplomonads like *Giardia muris, Spironucleus* and *Hexamita*.

The main focus has been the cytoskeleton, differentiation and the hydrogenosome. Other research areas in the group are drug resistance in protozoan parasites and worms, microbiomes in malaria mosquitos, diagnostics of tape worm infections

Short CV: https://www.scilifelab.se/researchers/staffan-svard/



Research field/Expertise:

# Inari Kursula

Professor

Department of Biomedicine, University of Bergen, Norway; Faculty of Biochemistry and Molecular Medicine, University of Oulu, Finland

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Malaria is one of the world's most devastating infectious diseases. Each year, nearly half a million people die of malaria. The disease is caused by *Plasmodium* spp., which comprise a group of unicellular, eukaryotic, intracellular parasites, belonging to the phylum Apicomplexa. They use an actomyosin motor complex, termed glideosome, for rapid gliding motility and host cell invasion. The motor components are to a large extent unique to these parasites or highly diverged from the corresponding human proteins. Parasite actin filaments are short, and their rapid treadmilling is regulated by an unusually small number of actin-binding proteins. Our work is focused on understanding malaria parasite gliding motility and the molecular machinery behind at the molecular/atomic level. We employ a broad range of biochemical, biophysical and hybrid structural biology methods for creating a complete molecular picture of the parasite actin-myosin motor and the entire glideosome. We also want to understand the evolution of apicomplexan gliding motility and eukaryotic actin-myosin motors in a broader sense. Understanding the mechanistic differences in cell motility between parasites and humans may, furthermore, open up new avenues for treatment and/or prevention of malaria.

#### Short CV:

https://www.uib.no/en/persons/Inari.Talvikki.Kursula#uib-tabs-cv https://www.oulu.fi/en/researchers/inari-kursula

#### More information:

https://www.uib.no/en/rg/inari; https://www.oulu.fi/en/research-groups/molecular-mechanisms-parasite-motility

Lecture date: Thursday 08.09.2023



# **Robert Charles Read**

Professor

Director and Honorary Consultant Physician Southampton NIHR Biomedical Research Centre, University of Southampton and Southampton University Hospitals, United Kingdom

R.C.Read@soton.ac.uk

Research field/Expertise:

Professor Robert Charles Read is Head of Clinical and Experimental Sciences within Medicine at the University of Southampton, and Director of the NIHR Southampton Biomedical Research centre. Robert Read trained in Medicine at the University of Sheffield, UK and completed his Doctorate Degree at Imperial College London, UK. He is Professor of Infectious Diseases and Honorary Consultant Physician in Infectious Diseases at University Hospital Southampton, and is Director of the NIHR Southampton Biomedical Research Centre. Professor Read trained in Infectious Disease and Internal Medicine in various posts in Leeds, Bristol, London and Nottingham, at the National Heart and Lung Institute, Imperial College London, and at the Division of Infectious Diseases, University of California, San Francisco (UCSF) at San Francisco General Hospital, USA. He was previously Professor of Infectious Diseases at the University of Sheffield before moving to Southampton in 2012. His research interests include the pathogenesis and prevention of infections arising within or involving the respiratory tract. He has a major interest in the use of controlled human infection to investigate pathogenesis, immunology and prevention of upper respiratory tract colonization by pathogens. Professor Read has previously had a leadership role as Head of School of Clinical & Experimental Sciences, University of Southampton (2014-2019). Nationally he was Chair of the Infectious Disease and Clinical Microbiology NIHR Clinical Research Network and Chaired the Postdoctoral Awards panel for the NIHR Trainee Coordinating Centre (2010-2015). Internationally, he was a member of Council of the European Society for Clinical Microbiology and Infectious Diseases (2005-2012), and chaired the Program Committee of the Infectious Disease Society of America (IDSA) (2013). He has been an appointed member of expert advisory groups for the UK Medicines and Healthcare Products Regulatory Agency, the UK Department of Health and the European Medicines Agency. He is a full member of the UK Joint Committee for Vaccines and Immunisation (JCVI). He is Editor-in-Chief of the Journal of Infection, and Current Opinion in Infectious Diseases.

Short CV: http://www.ibaschool.no/wp-content/uploads/2022/05/Read-MRC-CV-2022.pdf



## Valentina Tremaroli

PhD, Team Leader for gut microbiota metagenomics

Wallenberg Laboratory, Department of Molecular and Clinical Medicine, University of Gothenburg, Sweden

valentina.tremaroli@wlab.gu.se

Research field/Expertise:

Valentina Tremaroli is an expert in molecular microbiology and works in the field of gut microbiota in relation to obesity, type 2 diabetes, non-alcoholic fatty liver disease and cardiovascular disease. Her research goal is to contribute to the understanding of how microbial interactions may be targeted for human nutrition, metabolic regulation and preservation of health.

Dr Tremaroli has contributed to original articles describing the normal human gut microbiota and alterations associated to metabolic diseases, in particular type 2 diabetes. She has received recognition as highly cited researcher in 2019, 2020 and 2021: <u>https://publons.com/researcher/3226741/valentina-tremaroli/</u>

#### Short CV:

Dr Tremaroli studied Industrial Biotechnology and obtained a Ph.D. in Environmental Microbiology and Microbial Physiology at the University of Bologna, Italy. During her PhD and a one-year post-doctoral fellowship at the University of Calgary, Canada, she characterized soil communities and bacterial strains able to transform organic and inorganic pollutants. In 2009 she joined the laboratory of Prof. Fredrik Bäckhed at the University of Gothenburg as postdoctoral fellow to study microbe-microbe and host-microbe interactions in the human gut. She is now team leader for gut metagenomics research in the Bäckhed lab since 2013.

#### More information: https://backhedlab.org/team/valentina-tremaroli/

#### Lecture date: Friday 09.06.2023



## **Anne Salonen**

Group leader, Principal investigator

Human Microbiome Research Program, Faculty of Medicine, University of Helsinki, Finland

anne.salonen@helsinki.fi

Research field/Expertise:

The research activities of Dr. Anne Salonen are focused on the composition and activity of the intestinal microbiota in health and disease, especially in early life and regarding the interactions between diet, gut microbiota and metabolic diseases; she is a pioneer on addressing the microbiota as a driver of individual dietary responses. Dr. Salonen is a co-principal investigator of the Finnish Health and Early Life Microbiota (HELMI) birth cohort, involved on maternal fecal microbiota transplantation studies in C-section infants, and also investigating how the female reproductive track microbiota relates to clinical outcomes in obstetrics and gynecology.

#### Short CV:

Dr. Anne Salonen is a principal investigator, university researcher and adjunct professor at University of Helsinki, Finland. She has multidisciplinary training in biosciences and PhD in microbiology (2004).

More information: <u>https://www2.helsinki.fi/en/researchgroups/microbes-inside</u>

Lecture date: Friday 09.06.2023



#### **Amanda Singleton**

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My work aims to better understand bacterial responses to antibiotic stress. I am also part of a group developing a novel antimicrobial peptide which has shown to reduce mutagenesis when combined with other antibiotics. See abstract page 38



# Asal Ahmadi

asal.ahmadi@nmbu.no

I started my PhD in bacteriology from 1st of November 2022. In my PhD project we are trying to enlighten the role of Pasteurella multocida in the pathogenesis of bovine respiratory disease in Norway. <u>See abstract page 39</u>



## **Beathe Kiland Granerud**

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I am at PhD student in molecular virology at UiO, working with SARS-CoV-2 at Oslo University Hospital (Rikshospitalet and Ullevål). I have a background as a Biomedical laboratory scientist (bioingeniør), with a MSc in clinical microbiology and am also a qualified teacher. My PhD project includes methods in molecular biology, e.g WGS and qRT-PCR, immunology and biobanking. <u>See abstract page 40</u>



# **Bente Sved Skottvoll**

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In my project we seek to identify hidden reservoirs for ARGs from different environments including samples from subgingival pockets, faeces and wastewater. I will use common methods in the field and machine learning to analyze metagenome sequences of the samples to achive this. See abstract page 41



#### **Charlotte Krog**

charlie.krog@gmail.com

I am a PhD student at Oslo University Hospital. My PhD project focus on genetic elements in bacteria known as Toxin – Antitoxin (TA) systems with a main focus is on the small hydrophobic toxin called TisB. TisB expression has been linked to the formation of persister cells, and TisB expressing cells show higher tolerance against several antibiotics. The main question to answer is how does TisB, and other hydrophobic peptides modulate membrane function to resist stress, especially antibiotic induced stress. What is the biological function of TisB and does the toxin have interaction partners to perform its effect? <u>See</u> <u>abstract page 42</u>



## **Charlotte Årseth**

charlotte.arseth@ntnu.no

I am a 24 year old PhD student in the MYCOVIR-group at NTNU. I have a background (MSc) in bionanotechnology. My project focuses on the role of intracellular complement in Mycobacterium tuberculosis infections of macrophages. <u>See abstract page 43</u>



# **Collins GK Atuheire**

atuheirecollinsgrace@gmail.com

I am working on Burden of Rabies in Rural Uganda; I want to determine community reported incidence and mortality of rabies in 3 regions of rural Uganda. <u>See abstract page 44</u>



## Hesham Amin Mohamed Abouelhana

Hesham.amin@uib.no

I am Hesham. My background is mainly medical microbiology and molecular biology. In my PhD project I study the association between exposure to indoor bacterial communities, and lung function and eosinophilic airway inflammation, using high-throughput sequencing (16S rRNA), qPCR, and endotoxin measurements. See abstract page 46



#### **Ingeborg Mathiesen**

ingeborg.mathiesen@uit.no

I am research track student who has completed the first three years of medicine at UiT. Right now, I am in my research year. I am working with Enterococcus faecium and trying to find new virulence factors. <u>See abstract page 47</u>



Ingeborg Yddal ingeborgyddal@outlook.com

I have completed a bachelor's and master's degree in Molecular Biology and am currently doing my Ph.D. at The Influenza Centre at the University of Bergen. We are working on a project evaluating the effectiveness of the seasonal influenza vaccine in pregnant women and young children in Bangladesh. <u>See abstract page 48</u>



#### Jorge Agramont

jorge.agramont@chalmers.se

Hi, my name is Jorge Agramont, I'm from La Paz Bolivia, I'm a PhD student working on the surveillance of antibiotic resistance and diarrheagenic pathogens in water in La Paz Basin. I'm interested into learn more about infection and pathogenicity mechanisms to have a better comprehension of diarrhoeal infections and inflammation. <u>See abstract page 49</u>



#### Josue Mamani Jarro

jarro@chalmers.se

My name is Josue Mamani, I am a PhD student in the laboratory of Johan Bengtsson-Palme at Chalmers University, I am interested in the topic of pathogen surveillance and antibiotic resistance from the perspective of the One Health approach. I consider that this course will be very important for me to better understand the interactions between pathogens-humans and very useful for my thesis topic related to diarrheal diseases and the environment in Bolivia. See abstract page 50



#### **Klara Andersson**

andersson.klara.linnea@ki.se

My name is Klara and I am a PhD student at Karolinska Institutet, in a collaboration with a startup company called Biomedrex. We are taking inspiration from the bacterial defense mechanism CRISPR-Cas to develop antivirals with the hope to cure viral diseases. Currently we are focusing on SARS-CoV-2, but the approach can be applied to other viruses as well. <u>See abstract page 51</u>



#### **Lise Benette Hovd**

lisebenette@hotmail.com

Im a master student in the parasittology research group here at NMBU. My thesis is essentially about trying to find a way to utilize bovine organoids as an in vitro system for researching the parasite Cryptosporidium. In particular we're hoping to be able to fullfill the lifecycle in this system. <u>See abstract page 52</u>

#### Lukas Hoen

Lukashoen@hotmail.com My name is Lukas, and I work with Influenza, immunology, and vaccinology. See abstract page 52

# Maria Naqvi

marianaq@oslomet.no

Im a second year PhD student at OsloMet. My research focuses on the ocular microbiome and its potential relevance to dry eye disease. See abstract page 53



## Marita Pérez Syltern

marita.p.syltern@uit.no

I work with clinical E. coli strains, comparing variations in the metabolome in sensitive and resistant strains. I use high resolution-LC MS to obtain data and I apply non-targeted approach to achieve a comprehensive depiction of the metabolomic profile. <u>See abstract page 54</u>



# Mitchellrey Magbanua Toleco

mitchellrey.toleco@uis.no

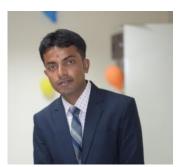
I am a nerd molecular biologist/(bio)chemist from the Philippines. I am a PhD student in Prof. Mark van der Giezen's molecular and biochemical parasitology lab at the University of Stavanger. I am fascinated by metabolite and carbon skeleton fates in the context of short-distance molecular transport across biological membranes and its role(s) in the organism's physiology. For my PhD project, I am working with a non-model anaerobic parasite called Blastocystis and potentially other stramenopiles, as part of the bigger Norwegian Research Council-funded mitochondrial glycolysis project of Prof. van der Giezen. <u>See abstract page 55</u>



# **Ninsiima Lesley Rose**

Ininsiima04@gmail.com

My Ph.D. is about bat ecology and social epidemiology of filoviridae hemorrhagic fevers in Uganda. One of my objectives is detecting the viruses (Ebola and Marburg viruses) in fecal matter which this course is important to me. <u>See abstract page 56</u>



## **Rajneesh Kumar**

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I am doing my PhD from University of Bergen, Norway, in my PhD I am studying the causes and influences of heterogeneous energy supply in cell and evolutionary biology, capturing the dependence of life's essential process on ATP. My research uses mathematical models and simulation to explore how ATP variability arises within and between cells, population dynamics, and the evolution of cooperation and competition. <u>See abstract page 57</u>



Rebekka Rolfnes

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I am working to discover the mechanism of action as well as the frequency and mechanism of resistance, of a new dual betalactamase inhibitor. <u>See abstract page 58</u>



#### Simen Hermansen

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I am a prospective PhD student at NMBU currently working as a project engineer on antimicrobial discovery at the Laboratory of Microbial Gene Technology (LMG). My primary interest is in membrane proteins and bacteriocins, and during my PhD I will focus on the mechanism of the LsbB family of bacteriocins and their interaction with the intramembrane zinc metalloprotease RseP. <u>See abstract page 59</u>



# Vilde Irene Nilsson

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I graduated as a veterinarian in December 2021 at the Veterinary Faculty at NMBU. Since then I have worked as a clinician in a small animal clinic in Oslo. I started my PhD in March this year, in infectious biology - Leptospirosis and One Health. <u>See abstract page 60</u>

## Meet the attendees



## Vismai Naik Thuppe

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My PhD project is on the host-parasite interaction between Atlantic Salmon and crustacean ectoparasite Salmon lice with emphasis on the immune modulation capabilities of Salmon lice. See abstract page <u>61</u>

## Signalomic and metabolomic analysis of a beta-clamp inhibitor and putative thymidylate monophosphate kinase inhibitor

<u>Amanda H. Singleton<sup>1#</sup></u>, O. E. T. Bergum<sup>1#</sup>, C. K. Søgaard<sup>1</sup>, L. M. Røst<sup>2</sup>, C. E. Olsen<sup>3</sup>, F. H. Blindheim<sup>3</sup>, B. H. Hoff<sup>3</sup>, P. Bruheim<sup>2</sup>, and M. Otterlei<sup>1</sup>

<sup>1</sup>Department of Clinical and Molecular Medicine, Norwegian University of Science and Technology (NTNU), NO-7489 Trondheim, Norway

<sup>2</sup>Department of Biotechnology and Food Science, Norwegian University of Science and Technology (NTNU), NO-7491 Trondheim, Norway

<sup>3</sup>Department of Chemistry, Norwegian University of Science and Technology (NTNU), NO-7491 Trondheim, Norway

World-wide increase in antimicrobial resistance far outpaces the development of new antimicrobial drugs. A putative thymidylate monophosphate kinase (TMPK) inhibitor (JK274) combined with a beta-clamp inhibitor (betatide) exhibited an 8-fold reduction in MIC for *Staphylococcus aureus*. To better understand the synergistic effect between the two antimicrobials and further elucidate the MoA of the two antimicrobials separately, we conducted a proteomic and metabolomic study using *S. aureus*.

Logarithmic phase *S. aureus* was treated with either JK274, betatide, or a combination. Two doses of each antimicrobial were included – a high and low dose. The high dose mimicked the growth curve of the combination treatment. The combination treatment consisted of low dose JK274 and betatide. Signalomics samples were taken at 10, 25, 50 and 180 minutes after treatment and processed for signalome analysis using a multiplexed inhibitor bead assay. Metabolomics samples were taken 180 min post-treatment. A control with no treatment was also sampled for both sample types.

The effects of betatide were seen in the 10-minute samples while the effects of JK274 treatments were not significantly detected until the 50-minute samples. After 10 min, proteins involved in translation and modification of proteins and RNAs showed reduced enrichment compared to the control. Additionally, the essential cell division protein FtsA was strongly reduced in all timepoints. At 25 min, the protein FtsZ also exhibited reduced pull-down with high dose MDR26. On the other hand, JK274 strongly reduced enrichment of TCA cycle proteins. JK274 did not reduce levels of Tmk as expected, but instead yielded reduced pull-down of nucleoside diphosphate kinase (Ndk).

Synergism between betatide and JK274 might be due to a combined action on both translation and respiration that overwhelms the bacterial cell. As a general NDK inhibitor, JK274 will most likely not be suitable as an antimicrobial as it may be toxic to human cells. However, betatide shows promise as novel antimicrobial drug.

<u>Poster Session I - Group I, Tuesday June 6th</u> <u>Meet Amanda Singleton</u>

## Pathways- Enlightening the role of *Pasteurella multocida* in the pathogenesis of bovine respiratory disease (BRD)

Asal Ahmadi<sup>1</sup>, Veslemøy Sunniva Oma<sup>2</sup>, Mette Myrmel<sup>1</sup>, Ann-Katrin Llarena<sup>1</sup>

<sup>1</sup> Department of Paraclinical Sciences, Faculty of Veterinary Medicine, Norwegian University of Life Sciences (NMBU), Ås, Norway

<sup>2</sup> Department of Production Animal Clinical Sciences, Faculty of Veterinary Medicine, NMBU, Ås,

Norway

Bovine respiratory disease (BRD) or pneumonia is a common and costly disease in all cattle production systems worldwide. BRD is a multifactorial disease and develops through interactions between the environment, host, and pathogens. In recent years, an increasing trend of Pasteurella multocida as a major bacterial isolate (instead of Mannheimia hemolytica) from fatal cases of BRD is observed. Although the mechanism of *P. multocida* pathogenicity is not well understood, it is believed that some virulence factors including capsule and lipopolysaccharide (LPS), genes that code iron-acquiring proteins, and surface proteins that bind to hemoglobin play an important role in the BRD pathogenesis. The main aim of this project is to shed light on the role of *P. multocida* in the pathogenesis of BRD in Norwegian herds. In order to achieve this, first we have planned to perform an epidemiological study of isolated P. multocida from the Norwegian cattle population in combination with whole genome sequencing. Methods: we have collected 370 isolates from 16 different Norwegian cattle herds from different anatomical parts of the respiratory tract. Bacterial DNA was extracted and sequenced using Illumina technology. Currently, in silico analyses investigating multilocus sequence typing (MLST), virulence factors, and phylogeny using the assembled genome are going on. We are going to address the genetic variation of *P. multocida* and how it relates to the farms, animal health status, and the anatomic part of the respiratory tract where the sample is collected. Following bioinformatic analysis, we are going to choose some isolates for further investigation (in vitro model and transcriptomic studies).

Poster Session I - Group I, Tuesday June 6th

Meet Asal Ahmadi

## Omicron Variant Generates a Higher and More Sustained Viral Load in Nasopharynx and Saliva than the Delta Variant of SARS-CoV-2

Beathe Kiland Granerud <sup>1,2,</sup>, T Ueland <sup>1,3,4</sup>, A Lind <sup>2</sup>, A Søraas <sup>2</sup>, B Fevang <sup>3,5</sup>, AK Steffensen <sup>1,2</sup>, H Al-Baldawi <sup>1</sup>, F Lund-Johansen <sup>6,7</sup>, P Aukrust <sup>1,3,4,5</sup>, B Halvorsen <sup>1,3</sup>, T B. Dahl <sup>3,8</sup>, S Dudman <sup>1,2</sup>, F Müller <sup>1,2</sup> and JC Holter <sup>1,2</sup>

<sup>1</sup> Institute of Clinical Medicine, University of Oslo, Oslo, Norway, <sup>2</sup>Department of Microbiology, Oslo University Hospital, Oslo, Norway, <sup>3</sup>Research Institute of Internal Medicine, Oslo University Hospital, Oslo, Norway, <sup>4</sup>K.G. Jebsen Thrombosis Research and Expertise Center, Faculty of Health Sciences, University of Tromsø, Tromsø, Norway, <sup>5</sup>Section of Clinical Immunology and Infectious Diseases, Oslo University Hospital, Oslo, Norway, <sup>6</sup>Department of Immunology, Oslo University Hospital, Oslo, Norway, <sup>7</sup>ImmunoLingo Convergence Centre, University of Oslo, Oslo, Norway, <sup>8</sup>Division of Critical Care and Emergencies, Oslo University Hospital,Oslo, Norway

**Introduction:** The SARS-CoV-2 Omicron variant (B1.1.529) was first reported in South Africa on 24 November 2021. Just one week later, on 30 November 2021, a laboratory in Oslo suspected and later confirmed Norway's first case. The Omicron variant spreads more easily than earlier variants, possibly because of a higher viral load in the upper respiratory tract and oral cavity. Other factors that can explain the high attack rate included reduced susceptibility to neutralizing antibodies, environmental factors, such as prolonged indoor exposure, or a high rate of asymptomatic carriage. In this study we have investigated whether the Omicron variant generates a higher viral load than that of the Delta variant in saliva and nasopharynx.

**Methods:** Saliva and nasopharyngeal swabs were collected from 52 confirmed Omicron and 17 confirmed Delta cases at two time points one week apart and analyzed by qRT-PCR, targeting the viral E-gene and the human HPRT1 gene. Viral load was measured as 10 log RNA genome copies per 1000 human cells according to the WHO reference standard. Finally, a linear correlation mixed model was used to assess the association between viral load, symptom days, virus variant and sample material.

**Results:** We found that Omicron cases carried a higher viral load and had more sustained viral shedding compared to the Delta cases, especially in the nasopharynx. This indicates that a higher and more sustained viral load may serve as possible explanation for increased transmissibility of the Omicron variant compared to the Delta variant.

Poster Session I, - Group I Tuesday June 6th

Meet Beathe Kiland Graneurd

# The resistome in general and clinical populations and their associated environment

Bente Sved Skottvoll<sup>1</sup>, M. Khomich<sup>1</sup>, J. Moradi<sup>2</sup>, R. J. Bertelsen<sup>1,2</sup>

<sup>1</sup>Univeristy of Bergen, Bergen, Norway, <sup>2</sup>Oral Health Center of Expertise in Western Norway, Bergen, Norway

Antibiotic resistance encoding genes (ARGs) are of a worldwide health concern and known to occur outside of the clinical setting. But it is still unclear where and how they spread to the clinic. We seek to find hidden reservoirs of known and unknown ARGs by using oral, faecal and wastewater samples as representatives of different ecotopes.

We have whole metagenome sequenced subgingival samples from two general population studies the Hordaland Health Study 3 (HUSK3, n $\approx$ 1800) and the multicentre European Community Respiratory Health Survey 3 (ECRHS3, n=330), as well as faecal samples from The Bergen COPD microbiome study (MicroCOPD, n=145), a clinical study with COPD, asthma, and healthy volunteers. The assembled metagenomes were annotated for ARGs using the CARD (Comprehensive Antibiotic Resistance Database) database to produce resistomes. Wastewater samples from Bergen are yet to be analysed and will reveal information from the associated environment.

Preliminary results from ECRHS3 subgingival samples suggest that ARGs for 18 classes of antibiotic drugs, following the CARD ontology, are detectable. Remarkably, genes encoding multidrug efflux pumps (median 0.01 relative abundance [range: 0-0.28]), tetracycline resistance (median 0.0 relative abundance [range: 0-0.17]) and  $\beta$ -lactam resistance (median 0.0 relative abundance [range: 0-0.16]) were the most abundant. There were no significant differences between the study centres regarding the total relative abundance of ARGs. The variation in relative abundance of ARGs was not explained by whether the participant had an antibiotic course to help breathing or for nasal/sinus problems last 12 months (24% yes, median courses: 1, range: [1-12]) or visited a hospital since last survey (approx. 10 yrs., 36% yes) or last 12 months (11% yes).

The ECRHS3 study participants are mostly healthy, and this contribute to explain why the abundance of ARGs is low, and why we do not see any clear association to contact with hospital nor antibiotic use for upper respiratory illnesses.

Poster Session I, - Group I Tuesday June 6th

Meet Bente Sved Skottvoll

## Investigation of TisB induced pH depolarization in *Escherichia coli* following DNA damage and the role of ATP synthase as a potential protein partner.

#### Charlotte S. Krog<sup>1</sup>

<sup>1</sup> Department of Microbiology, University of Oslo, and Oslo University Hospital, Rikshospitalet, Oslo, Norway

Antimicrobial resistance (AMR) is a pressing global health crisis. During stressful conditions, bacteria activate stress response systems to enhance survival. However, the underlying mechanisms of bacterial stress responses and their ability to resist unfavorable circumstances remain incompletely understood. Recent research has linked genetic elements called Toxin-Antitoxin (TA) systems to bacterial persistence states that allow them to evade the effects of antibiotic therapy.

TisB peptide, encoded by the *tisB-istR* type 1 TA system and expressed during the bacterial SOS response to DNA damage, affects membrane integrity by modulating membrane functions. TisB overexpression leads to membrane depolarization, ultimately disrupting important cellular processes. Although the damaging effects of TisB overexpression have been studied, little is known about the mechanistic mode of action of this small hydrophobic peptide under membrane depolarization. Our work aims to investigate the molecular mechanisms underlying TisB-mediated proton depolarization in *E. coli*.

At present TisB is believed to interact with and create pores in the membrane, thereby disrupting the membrane potential, resulting in ATP depletion, and growth arrest. Using flow cytometry, we demonstrate TisB's ability to depolarize the proton gradient of the cell membrane upon antibiotic-induced DNA damage. Our results show that proton depolarization upon antibiotic-induced cell death depends on the genetic presence of both *tisB* and *atpA-H* encoding subunits of the FoF1-ATP synthase complex, indicating that TisB-mediated depolarization is dependent on a functional ATP synthase complex. Preliminary results using the bacterial-2-hybrid system suggest a close interaction between TisB and the membrane-embedded part of the FoF1-ATP synthase complex.

Taken together, our results suggest a potential protein-protein interaction between TisB and the FoF1-ATP synthase complex. In our continued work, we will use mass spectrometry (MS) pulldown to further investigate potential partners to TisB. Our study sheds light on how small hydrophobic peptides modulate membrane function by interacting with membrane complexes to resist stress, particularly stresses induced by antibiotics. These findings can be used for further investigations of antibiotic targets, the persister state, and the development of antimicrobial peptides.

Poster Session II, - Group I Wednesday June 7th

<u>Meet Charlotte Kroq</u>

## The Role of Intracellular Complement in *Mycobacterium Tuberculosis* Infection

Charlotte Årseth<sup>1</sup>, N. Niyonzima<sup>1</sup>, M. Haug<sup>1</sup>, T. Espevik<sup>1</sup>, T. H. Flo<sup>1</sup>

<sup>1</sup>Centre of Molecular Inflammation Research, Department of Clinical and Molecular Medicine, Faculty of Medicine and Health Sciences, Norwegian University of Science and Technology, Trondheim, Norway

In 2021, 1.6 million people died from Tuberculosis (TB), and an estimated 10.6 million people fell ill. This makes TB one of our top infectious killers, beaten only by Covid19 the latter years [1]. By 2035, WHO's End TB strategy aims to reduce TB incidence by 90%, and TB deaths by 95%. In 2022, these numbers are however only at 20% and 35%, respectively. TB is treatable, but long treatment duration, complex drug combinations, multidrug-resistant TB, and side effects often yield treatment unsuccessful [2]. Hence, there is an urgent need to understand the pathogenic nature of TB's causative agent, Mycobacterium Tuberculosis (Mtb), to improve treatment strategies and patient outcomes. Mtb mainly infects macrophages, but it activates both innate and adaptive immunity responses. Together, these lead to the formation of granulomas; inflammatory and fibrous cell infiltrates that may cause epithelial cell death and consequently lung destruction. The complement system is a branch of the innate immune system important for curbing bacterial infections. We know that Mtb activates all three pathways of complement, but its role in Mtb pathogenicity remains unclear [3].

The complement system was previously thought to be hepatic and extracellular only, but has been proven to exist and be synthesized intracellularly in a wide range of cells. Where it serves multiple non-canonical functions in basic cellular processes like homeostasis, autophagy, metabolism, and gene expression [4-7]. SARS-CoV-2 infection or inflammatory cytokine stimulation gives excessive intracellular C3 production, and inhibiting this suppressed inflammation and protected epithelial cells from death [8-9]. Simultaneously, the intracellular complement C5/C5aR1 axis alters IL-1 $\beta$  production and controls inflammation in macrophages after cholesterol crystal stimulus. Taken together, these results indicate that intracellular complement may be important in Mtb infection, which also concerns macrophages and epithelial cells. We have performed RNA sequencing on induced macrophages (iMACs) from induced pluripotent stem cells (iPSCs) after stimulation with an attenuated (auxotroph) Mtb strain. The results showed up- and downregulation of different complement components in response to the infection, which were not always in line with the positive LPS control. Intracellular flow cytometry confirmed the presence of intracellular C3a, C3aR, C5a, C5aR1, and C5aR2 in iMACs. Preliminary LDH results indicate that siRNA-knockdown of C5aR1 may be protective in Mtb infection.

We plan to further elucidate the role of extra- and intracellular complement in Mtb pathogenicity by comparing cell permeable and –impermeable inhibitors, investigating protection against cell death, live imaging, and sub-cellular imaging techniques such as electron microscopy.

Poster Session II, - Group I Wednesday June 7th

<u>Meet Charlotte Årseth</u>

## Epidemiology of Rabies in Eastern Rural Uganda: A One Health Approach

**Collins GK Atuheire<sup>1</sup>,** K. Bwambale<sup>1</sup>, M. Taremwa<sup>1</sup>, A. Mirimu<sup>1</sup>, P. Ssajjakambwe<sup>2</sup>, J. Okwee-Acai<sup>2</sup>, M. Munyeme<sup>3</sup>, C. Kankya<sup>1</sup>, O. Terence<sup>1</sup>, S. Ssali<sup>4</sup>, F. Mwiine<sup>5</sup>, M. Tryland<sup>6</sup>

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**Background**: Rabies, a vaccine preventable disease, has been neglected primarily in most rural parts of Africa. It is estimated to claim 16000persons per year in Uganda. Sympatric communities that neighbor national parks and game reserves where rabies reservoirs in wild interact with domestic dogs and livestock and humans have been neglected. The aim of this study was to determine the prevalence and risk factors of rabies among households around Pian Upe game reserve in Bukedea district, Eastern Uganda.

**Methods**: We employed a questionnaire based cross-sectional study among households in purposively selected six sub-counties of Bukedea district located at >14kms (far), 4-14kms (near) and <4kms (close) to the game reserve. Informed consent was sought prior to data collection and the kobo collect tool was used by trained research assistants. Socio-demographic and prevalence data were summarized as means, SD, percent and frequencies. Bivariable and multivariable Logistic regression was carried out to assess predictors of rabies prevalence and mortality among humans, livestock, wildlife and dogs.

**Results**: A total sample of 302 were studied, the mean age of participants was 44 years with SD=16yrs, with about 34% of the households being headed by females. It was found out that 47% of owned dogs had no history of vaccination over the previous one year and 39% of the households practiced hunting.

Over all, the prevalence of rabies increased as one approached the game reserve. It increased from 7.5% to 15.7% in humans and dogs equivalently, and then from 5% to 9.8% among livestock. Mortality of rabies follows similar trend like that of rabies prevalence in Bukedea. Mortality increased from 49.7-98.0 per 1000 animals for livestock, from 87.0-196.1 per 1000 animals for wildlife and from 43.5 -55.6 per 1000 persons in humans.

The predisposing factors at close and near distances respectively included: primary level of education aOR[95%CI] = 10.0[1.0-154.4] and grazing in game reserve aOR[95%CI] = 7.2[1.0-79.8]. Conversely, far distance from the game reserve conferred protection from rabies among humans as shown by: Primary aOR[95%CI] = 0.06[0.01-0.43] and secondary aOR[95%CI] = 0.04[0.00-0.46] levels of education.

Fetching fire wood versus not from the game reserve had 7.5 odds more for mortality (aOR=7.5, 95%CI 1.5-37.0). There was interaction between firewood source and distance of house hold to the game reserve, the probability of mortality was 18% for those coming from distances over 15kms while those coming from near (4-14km) had a 7% probability of mortality.

**Conclusions**: Rabies risk increased as house hold proximity to the game reserve increased. The potential risk factors associated with rabies included fetching fire wood from the game reserve, grazing in game reserve and primary level of education. Community sensitization and targeted dog rabies vaccination among communities surrounding game reserves is highly warranted.

Key words: One health, Rabies burden, Wildlife-Animal Human Interface, Sympatry

Poster Session II, - Group I Wednesday June 7th

Meet Collins Atuheire

# Association between indoor bacterial communities, lung function and airway inflammation

<u>Hesham Amin<sup>1</sup></u>, Cramer C<sup>2</sup>, Drengenes C<sup>1</sup>, Finster K<sup>3</sup>, F Gómez<sup>1</sup>, Gislason T<sup>4</sup>, Holm M<sup>5</sup>,
 Janson C<sup>6</sup>, Jogi NO<sup>1</sup>, Jogi R<sup>7</sup>, Malinovschi A<sup>6</sup>, Marshall I. P<sup>3</sup>, Modig L<sup>8</sup>, Norbäck D<sup>9</sup>, Shigdel R<sup>1</sup>, Sigsgaard T<sup>2</sup>, Svanes C<sup>10</sup>, Thorarinsdottir H<sup>11</sup>, Wouters I. M<sup>12</sup>, Šantl-Temkiv T<sup>3</sup>, Schlunssen V<sup>2</sup>, Bertelsen RJ<sup>1</sup>

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Objective: We studied the association between indoor bacterial exposure and lung function and airway inflammation measured as Fractional exhaled Nitric Oxide (FeNO) in adults in Northern Europe.

Methods: The bacterial communities of settled airborne dust samples from the bedrooms of 1038 participants in the European Community Respiratory Health Survey (ECRHS) III from five study centres were characterised by 16S rRNA amplicon sequencing, and bacterial load by qPCR. The samples were collected concurrently with spirometry and FeNO measurements (outcomes). Adjusted linear regression stratified by sex were used to model the association between bacterial profiles and outcomes.

Results: Higher bacterial diversity and richness were associated with an increase in FVC and FEV1 Z scores in males (P < 0.05), and with elevated FeNO in females only (P < 0.05). Most bacterial genera associated with higher lung function were from the Actinobacteriota phylum. Higher relative abundance of Bacteroidia, Myxococcota, and Clostridia was associated with lower lung function, as was true also for several bacterial genera from the core oral microbiome, including Streptococcus and Veillonella. Higher FeNO levels were positively associated with the presence of Campylobacter and negatively with the presence of Cellulomonas.

Conclusion: We conclude that a higher microbial diversity is associated to higher lung function in males and increased inflammation and lower lung function in females. Further studies are needed to understand the relation between exposure to specific types of bacteria and lung conditions.

Poster Session II, - Group I Wednesday June 7th

Meet Hesham Amin Mohamed Abouelhana

## CHARACTERISATION OF PUTATIVE VIRULENCE FACTORS IN ENTEROCOCCUS FAECIUM

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Increasing antibiotic resistance hampers treatment options and thus new ways of treatment are needed. Therefore, it is important to understand how resistant bacteria cause disease and use this knowledge to find new targets for intervention. Multi-resistant *Enterococcus faecium* is posing a great threat to public health and needs to be studied closer. Clinical strains of *E. faecium* are increasingly invasive, and their ability to acquire new resistance and virulence genes stands out. Discovery of new virulence factors and novel knowledge of bacterial expression upon meeting with host, will provide future targets for infection prevention and/or treatment.

In this project, bioinformatic methods comparing whole genome sequencing data of nosocomial and commensal *E. faecium* isolates were utilized to point out putative new virulence factors. Potential candidate genes will be characterized regarding their prevalence and genetic context. Their expression profile has been studied through RT-qPCR in standard bacterial growth medium showing that the genes of interest are present in three different nosocomial strains of clinically relevant STs. Furthermore, the genes will be studied in conditions representing an infection context, including different components mimicking infection sites. Further functional assays will be designed depending on potential functional domains of the putative virulence factor predicted based on the genomic sequence of the candidate gene. Transposon mutants will be isolated from a transposon mutant library and used for the functional assays.

Characterization of novel virulence factors will help the understanding of the invasive properties of *E. faecium* and may reveal interesting targets for therapeutics discovery using anti-virulence strategies.

Poster Session III - Group I, Thursday June 8th

Meet Ingeborg Mathiesen

## Respiratory virus surveillance during an effectiveness trial to evaluate the protection of children and pregnant women by influenza vaccine in rural Bangladesh

<u>Ingeborg Yddal<sup>1</sup></u>, Khalequzzaman Zaman<sup>2</sup>, Warda Haque<sup>2</sup>, Amit Bansal<sup>1</sup>, Doli Goswami<sup>2</sup>, John Clemens<sup>2</sup>, Sarah Lartey<sup>1</sup>, Rebecca J. Cox<sup>1</sup>

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Influenza is one of the leading causes of severe disease and mortality in young children under five years and pregnant women. Maternal influenza infection during pregnancy is associated with increased risk of hospitalization and fetal malformation. In Bangladesh, influenza is responsible for 10% of all childhood pneumonias. Influenza is vaccine-preventable and young children and pregnant women are prioritized for vaccination. However, in most low-to-middle-income countries influenza vaccination is not part of the vaccination program.

We have conducted a randomized trial in pregnant women and young children to assess the impact of inactivated influenza in preventing influenza in the community. Pregnant women in the third trimester and children under 5 years in 10 villages were randomized to receive the influenza vaccine and 10 villages received the control polio vaccine. We followed the subjects postvaccination and collected respiratory swabs if they exhibited influenza-like illness (ILI) to confirm viral aetiology. We also collected saliva and serum samples from all children over 2 years to investigate influenza-specific antibody responses.

We have conducted virological surveillance to investigate which respiratory viruses are circulating in Bangladesh. From April 2021 to August 2022 samples were collected from 997 participants from the 20 villages and 226 samples from villagers who had ILI symptoms. Overall rhinovirus was the most dominant virus followed by adenovirus, influenza virus, SARS-CoV-2, respiratory syncytial virus (RSV), human metapnuemonia virus, and human parainfluenza virus (HPIV). RSV started appearing in July and peaked in September before decreasing in November. Rhinovirus was present all year with the highest peak in March 2022. SARS-CoV-2 showed highest prevalence in February 2022.

The Influenza positive samples were characterized for typing and subtyping. All samples were identified as Influenza B virus Victoria lineage until August 2021. In September there was a spike in influenza cases all characterized as Influenza A (H1N1). In November, influenza cases decreased. One Influenza A (H3N2) virus was identified, while the rest were positive for H1N1. Between December and May 2022, no influenza virus was detected. In the last week of June, H3N2 started appearing and reached its peak in July 2022. This is an ongoing study and the serological response to the infecting influenza viruses is currently being analyzed at the University of Bergen.

Poster Session III - Group I, Thursday June 8th

Meet Ingeborg Yddal

## Tracking Antibiotic Resistance Genes in rivers polluted by Sewage Water in La Paz Bolivia

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The global impact of antibiotic resistance poses a significant threat to human health. Antibiotic resistance genes (ARGs) are now recognized as emerging contaminants, particularly affecting aquatic environments. This is concerning because ARGs have the potential to be acquired by pathogens, leading to serious public health concerns. The discharge of wastewater is a key factor in the co-occurrence of mobile genetic elements (MGEs) and various ARGs in water and sediments. Analyzing wastewater samples looking for pathogenic and antibiotic resistant bacteria as well as ARGs is a good way to survey for strains and ARGs circulating in the community. Previous studies in La Paz Bolivia analyzed the presence and abundance of ARGs and antibiotic resistant bacteria in water samples, by culture dependent methods, or by qPCR approaches. As there are thousands of ARGs and qPCR approaches are limited to the surveillance of few of them, metagenomic approaches have the advantage to look for all the known ARGs types and variants in a single experiment together with information about mobile genetic elements and pathogenic bacteria in the samples. Thus, metagenomic analysis of wastewater in La Paz could help us to have a more comprehensive perspective of the circulation of diarrheic bacterial pathogens and antibiotic resistance genes in the community and could contribute to better understand the evolution of antibiotic resistance in the environment. The present study has the aim to contribute to the understanding of the contribution of untreated wastewater discharges into the La Paz River in Bolivia and survey the presence of circulating ARGs and pathogen in the population using a metagenomic approach for the sewage/river interfaces.

Poster Session III - Group I, Thursday June 8th

Meet Jorge Agramont

## Structure and Diversity of Environmental Resistomes in aquatic environments of the Seke and Seco Rivers in El Alto-Bolivia

Josue Mamani Jarro<sup>1</sup>, Johan Bengtsson Palme<sup>2</sup>

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Antimicrobial resistance (AMR) is an escalating concern due to the indiscriminate use of antibiotics and environmental pollution. This has led to the emergence of resistant bacteria, severely limiting our treatment options and amplifying the risk of severe and potentially fatal infections.

Research has demonstrated that water basins, such as the Rio Seke and Rio Seco, prominent rivers in El Alto city, are contaminated with sewage, heavy metals, industrial waste, and agrochemical waste. Consequently, there is a high probability of direct or indirect transmission of pathogens or antibiotic resistance genes from these rivers to the human population residing in the vicinity. This is particularly worrisome as these waters are used for irrigating vegetables and agricultural soils in communities surrounding the Katari basin.

By utilizing metagenomics approaches, we can analyze the microbiome and resistome derived from environmental sources, facilitating a deeper understanding of the role of pathogens and the distribution and transmission of AMR. Examining the bacterial resistome in the aquatic environment of Rio Seke and Rio Seco we can help identify alternative sources of resistance gene evolution beyond antibiotic consumption, such as aquatic environments contaminated with heavy metals or pesticides. Furthermore, it allows for modeling the mechanisms of resistance gene transmission and discovering potential new resistance genes related to heavy metals and pesticides (co-selectivity), as well as genes associated with xenobiotic degradation. These findings contribute to the implementation of preventive measures aimed at mitigating the problem of antibiotic resistance in Bolivia.

Poster Session III - Group I, Thursday June 8th

<u>Josue Mamani Jarro</u>

### **CRISPR-based antivirals against emerging viruses**

<u>Klara Andersson<sup>1,2</sup></u>, Vanessa Monteil<sup>2</sup>, Gözde Güclüer Akpinar<sup>1</sup>, Ani Azatyan<sup>1</sup>, Laura Sardon Puig<sup>1</sup>, Martin Ekenberg<sup>1</sup>, Theodor Pramer<sup>1</sup>, Ali Mirazimi<sup>2</sup>, Marjo Puumalainen<sup>1</sup>

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The increasing globalisation provides an opportunity for viral spread with the inevitable consequence of new emerging, infectious viruses. We need effective antivirals for novel viruses, recently demonstrated by the COVID-19 pandemic caused by SARS-CoV-2. Unfortunately, viruses have clever ways of escaping our immune system, promoting frequently mutated viral strains, thus increasing the risk of high resistance to antiviral treatments.

In this project we suggest targeting viruses with CRISPR-Cas technology, based on microbial immune systems that recognize and destroy foreign RNA/DNA. The system can be utilized for many purposes due to a large selection of effector molecules. Cas13 is one effector molecule that can cut viral RNA by building a complex together with CRISPR RNA (guide RNA), that will guide the Cas13 protein to the target sequence. Together they compose a CRISPR-Cas system with potential to work as an antiviral agent. By designing 30 nucleotides long guide RNAs that target essential viral genes, ideally at multiple sites at once, the hope is to find a new treatment for diseases caused by RNA viruses.

The project is carried out as a collaboration between Karolinska Institutet and a start-up company called Biomedrex, who are specializing in CRISPR-based antivirals. The project will shed light on CRISPR-Cas efficacy, toxicity, and potential as a combination treatment with crRNAs targeting multiple sites of a viral genome. Ultimately, the project aims to contribute to development of novel antivirals against emerging viruses.

Poster Session I – Group II, Tuesday June 6th

<u>Meet Klara Andersson</u>

# Bovine Intestinal Organoids as an *in vitro* infection model for *Cryptosporidium Parvum*

<u>Lise Benette Hovd<sup>1</sup></u>, Mathilde Varegg<sup>2</sup>, Lucy Robertson<sup>2</sup>, Alejandro Jimènez Melèndez<sup>2</sup>

<sup>1</sup> Faculty of Biosciences, Norwegian University of Lifesciences, Aas, Norway <sup>2</sup>Faculty of Veterinary Medicine, Norwegian University of Lifesciences, Aas, Norway

Having good and reliable infection models is crucial in order to conduct research on host-pathogen interactions. *In vivo* experiments can be costly, time-consuming, hard to replicate and have ethical implications. The novel field of utilizing organoids in host-pathogen research offers a new outlook for improving our understanding and abilities to create new medicines and precautionary actions for pathogens that pose a risk for both human and animal health. Organoids offer an approach that is closer to *in vivo* than current cell-culture models do, as they differentiate and organize themselves in a 3D system.

*Cryptosporidia* are protozoan parasites with a broad range of both specific hosts and a liberal approach to accidental hosts. *Cryptosporidium parvum is* one of the main intestinal pathogens for young calves in Norway and has an impressive zoonotic potential. This combined with the resilient oocyst stage of the parasite, making it difficult to get rid of in the environment, nominates this parasite as a great contestant as a research subject in this novel infection model.

To our knowledge, no one has fully triumphed in replicating this parasite's lifecycle *in vitro*, and hence there are still riddles to solve for us to be able to understand this parasite fully. In our research we're utilizing intestinal bovine organoids, a 3D growing cell-culture, in attempts to replicate the lifecycle of *cryptosporidium* under controlled conditions.

So far, the research has been unyielding in regards fulfilling the lifecycle of the parasite but thrilling in the hunt for new approaches for this novel infection model.

<u> Poster Session I – Group II, Tuesday June 6th</u>

Meet Lise Benette Hovd

## Comparing the Humoral Immune Responses Induced Following Live Attenuated Influenza Vaccine Immunization in Children & Adults

Lukas Hoen<sup>1</sup>, Sarah Lartey<sup>1</sup>, Fan Zhou<sup>1</sup>, K.G.I Mohn<sup>1</sup>, K.A Brokstad<sup>1</sup>, R.J. Cox<sup>1</sup>

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A variety of influenza vaccine types are available, with differing capacities to elicit humoral and cellular immunological responses with desirable phenotypes depending on the age of the recipient. Live attenuated influenza vaccines (LAIVs) are a formula of three or four attenuated influenza vaccine strains containing season specific surface glycoproteins, Haemagglutinin (HA) and Neuraminidase (NA) proteins, on a well-established backbone of cold adapted and temperature sensitive genome segments. LAIV induces milder infection localized to the upper respiratory tract. In contrast to licensed inactivated, recombinant, and experimental mRNA influenza vaccines, LAIV elicited immune responses more closely mirror a natural local infection and is considered the optimal priming vaccine for young children with limited prior exposure to influenza. However, adults benefit more from inactivated influenza vaccines, due to pre-existing immunity capable of limiting LAIV replication before any sufficient antigen can be presented to the adaptive immune system.

In this study we aimed to understand the quantitative, qualitative and durability of the humoral immune responses after LAIV vaccination in children and adults. We specifically assessed neutralizing antibodies by micro-neutralization assay (MN), antibodies capable of inhibiting the enzymatic activity of the neuraminidase by enzyme-linked lectin assay (ELLA), and the affinity maturation of antibodies (by avidity ELISA) targeting NA and HA up to one year after vaccination. We found a higher fold induction of neutralizing antibodies in children following LAIV vaccination than in adults. Interestingly we observe, exceptionally high NA-specific antibodies against influenza B in adults, which is 10-fold higher than that observed in vaccinated children, this is perhaps because the influenza B vaccine strain replicates well in adults. Overall, our findings shed light on the differences in immune responses by age group allowing targeting of the most appropriate licensed influenza vaccine to provide optimal protection.

Poster Session I – Group II, Tuesday June 6th

<u>Meet Lukas Hoen</u>

## The Composition of the Ocular Surface in Dry Eye Disease Patients

Maria Naqvi<sup>1</sup>, Fredrik Fineide<sup>2</sup>, Tor Utheim<sup>2,3</sup> and Colin Charnock<sup>1</sup>

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#### **Background/aims:**

The ocular microbiome and its impact on the development of dry eye disease is not well established. Previous work suggests that the ocular core microbiome consists of *Proteobacteria*, *Firmicutes* and *Actinobacteria*. However, results in previous studies vary and depend on the identification method. In this study we investigate the ocular microbiome of healthy individuals and dry eye disease patients. Our aim is to determine what the ocular microbiome consists of in a healthy population and dry eye disease patients.

#### Methods:

Dry eye patients were recruited from the Norwegian Dry Eye Clinic and healthy volunteers were recruited from Oslo Metropolitan University (OsloMet). Participants filled out the dry eye questionnaire-5 (DEQ-5) and the ocular surface disease index questionnaire (OSDI) prior to conjunctival sampling. Flocked swabs were used to collect tear samples from the inferior conjunctival fornix from both eyes in duplicate. One of the swabs was used for bacterial cultures, and the other swab was used for 16S rRNA sequencing. Swabs collected for bacterial cultures were inoculated in MSwab medium and plated on MacConkey agar, Brain heart infusion agar, R2A agar with Vancomycin, Schaedler anaerobe agar, Sabouraud glucose agar with chloramphenicol and Columbia agar with chocolate horse blood. All agar plates were incubated at 37°C for 1 week except for Schaedler and Columbia agar. Schaedler agar was incubated in anaerobic conditions whereas Columbia agar was incubated in aerobic, anaerobic, and microaerophilic conditions.

#### **Results:**

In total 79 participants were enrolled of which 61 were dry eye patients and 18 were healthy individuals without symptoms. Fifty-five of the swabs obtained from the dry eye group were cultured of which 49 swabs were positive for bacteria (89%). Seventeen of the 18 swabs obtained from the healthy group were positive (94%). Of the positive swabs, 41 were positive for more than one bacterial species (74%) in the dry eye group. In the healthy group 14 swabs were positive for more than one species (78%). No growth was observed on Sabourad glucose agar.

#### **Conclusion:**

Overrepresentation of a certain species of bacteria can be used as a biomarker for dry eye disease and contribute to improved diagnostic methods. The bacteria present on the ocular surface of dry eye patients can be further investigated to determine if they interact with mucin secretion which is an important component of the tear film.

#### <u> Poster Session I – Group II, Tuesday June 6th</u>

#### <u>Meet Maria Naqvi</u>

## Comparing the metabolomic profile of clinical resistant and sensitive *E. coli* strains through non-targeted HR-MS analysis

Pérez Syltern, Marita<sup>1</sup>, Fredheim, Elizabeth G.A<sup>1</sup>., Vasskog, Terje<sup>1</sup>, Hansen,

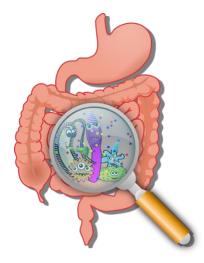
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Common human pathogens, such as *Escherichia coli*, show an increasing trend of acquiring antimicrobial resistance (AMR) to antibiotics. Infections caused by resistant bacteria have socioeconomic consequences, such as extended hospitalisation stays, costly treatments and simple medical procedure can lead to a deadly outcome.

Metabolites, which are small organic molecules, are the final downstream product. This implies that metabolomics is closest to the phenotype with respect to other omics fields. Hence, any variation in the environment or interaction between protein expression and/or gene expression will be reflected in the metabolome. For that reason, metabolomics provides an unique description of drug action and elucidates fluctuations in the abundance of metabolites when conducting drug treatment. We therefor believe that AMR can benefit from a new and growing field of omics, **non-targeted metabolomics**.

We have initially focused on the endometabolome, which comprises metabolites present in the bacterial cell. Information acquired through the visualization of the endometabolome from sensitive and resistant clinical *E. coli* strains will be further assessed with the prospect of conducting studies including data from other omics fields. The overall objective is to provide comprehensive and crucial insight into the acquisition of resistance mechanisms of both in-use and potential novel antimicrobials through a multi-omics approach.



Our work this far consists of extracting endometabolomic samples from liquid cultures. *E. coli* samples were obtained by fast filtration and liquid nitrogen was used to quench the metabolome. Efficient sampling and quenching are the pivotal steps in any metabolomics experiment due to the rapid turn-over rates of metabolites. The collected samples were analysed with state-of-the-art HR-LCMS, Thermo Scientific Orbitrap Id-X. The analysis was performed in reverse phase in positive mode and hydrophilic interaction chromatography (HILIC) in negative mode to acquire as much coverage of the metabolome as possible with reasonable instrument time.

<u> Poster Session II – Group II, Wednesday June 7th</u>

Meet Marita Pérez Syltern

## Tales of a Mighty Conqueror: Insights into *Blastocystis* Metabolism

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*Blastocystis* are among the most successful parasites to have colonized the human gut. It has been estimated that 1 to 2 persons out of seven may be colonized by *Blastocystis* (Scanlan and Stensvold, 2013). Our work focuses on a unique biochemistry exhibited by this parasite: mitochondrial glycolysis and bioenergetics. The project can be envisaged as divided into three overlapping parts: (i) development of molecular tools for the genetic manipulation of Blastocystis; (ii) use of these tools to delve into the molecular/biochemical details of the mitochondrial glycolysis as it relates to mitochondrial physiology and energy metabolism; and (iii) generate and integrate multi-omics datasets to better understand this parasite. Here, we report a modification of the transformation protocol originally developed by our collaborators at the National University of Singapore and a fluorescent protein reporter system compatible with *Blastocystis* and potentially other anaerobic biological systems.

Poster Session II – Group II, Wednesday June 7th

Meet Mitchellrey Magbanua Toleco

### BAT ECOLOGY AND SOCIAL EPIDEMIOLOGY OF *FILOVIRIDAE* HAEMORRAGIC FEVERS IN UGANDA

#### Ninsiima Lesley Rose, Kampala, Uganda

Department of Biosecurity, Ecosystems and Veterinary Public Health, Makerere University

**Background**: Viral Hemorrhagic Fevers are caused by single-stranded RNA viruses with a lipid envelope which vary in size and morphology. Filoviruses include but are not limited to Ebola and Marburg viruses, which are recognized as a significant threat to public health as they cause periodic human and non-human primate outbreaks with high mortality rates in humans ranging from 25% to up to 90%. Filoviruses can be transmitted by direct contact with blood, bodily fluids, or skin of patients or individuals who have died of the disease. Indirect contacts to humans can be through fecal material, urine, or saliva left on fruits or branches of trees.

**Methodology**: A cross sectional study design using field-based and questionnaires data collection will be employed to study the bat ecology and social epidemiology of *filoviridae* haemorrhagic fevers in Uganda in the Rwenzori areas with Bundibugyo, Mubende and Kassanda district inclusive in Uganda. The main objective is to assess the relationship aspects of bat ecology and social epidemiology of *filoviridae* hemorrhagic fevers in Uganda.

**Objective 1**: To evaluate the information on the relationship between bat behavior and distribution leading to spillover events of filoviruses. This is a systematic review and meta-analysis and will be done through data search for literature from different data bases. **Objective 2**: To assess the knowledge perception and practices towards bats by people leaving near bat roosting sites that leads to spillover events. This will use a cross sectional study design using questionnaires and key informant interviews to assess knowledge, perceptions and practices at household level. **Objective 3**: To explore anthropogenic drivers of spillover events from Ebola virus disease in Mubende and Kassanda districts. This study will use a cross sectional study design with qualitative and participatory methods. **Objective 4**: To predict the probability of spillover of filoviruses using a new time nonhomogeneous stochastic process at different seasons of the year in Albertine region. **Objective 5**: To determine the presence of filoviruses among selected bat roosting sites in the Rwenzori region. This will use a serial cross sectional study design-field using sample collection checklist and laboratory-based methods to extract RNA for Ebola and Marburg virus detection. Fecal samples will be collected using convenience sampling method.

**Anticipated findings and Conclusion**: Results from this study will inform about previous spillover events of filoviruses in the Rwenzori areas including Bundibugyo, Mubende and Kassanda districts to raise awareness on environmental factors affecting bats ecology, their environmental reservoirs and social epidemiological dynamics by providing scientific evidence for policy and public health strategy development against bat-borne zoonotic illness outbreaks in Uganda.

Poster Session II – Group II, Wednesday June 7th

Meet Ninsiima Lesley Rose

## Genetic Circuitry's Response to Energy Availability in Decision-Making Processes

Rajneesh Kumar<sup>1</sup>, Iain G. Johnston<sup>2</sup>

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Energy is necessary for all life, at all scales, from the cellular to the evolutionary, as it is a driven, non-equilibrium system. Like human society, a consistent energy supply is essential for cells to process information and take actions, and when cellular energy supplies are challenged, disorders can result. Here we use a mathematical model of gene expression in a bistable decision-making regulatory network to explore cellular bifurcation behaviour when we vary the energy availability. We impose that the rates of the associated gene expression processes in our model are reliant on an ATP concentration parameter since we know that each step-in transcription and translation requires energy in the form of ATP. We discuss both a deterministic model, to explore the emergence of different attractors under different ATP concentration parameters, and a stochastic case to explore how ATP influences the noisy dynamics and magnitude of protein expression and importantly whether switching between two protein concentration become more or less common.

Poster Session II – Group II, Wednesday June 7th

Meet Rajneesh Kumar

# Investigating the mechanism of action of a new novel dual $\beta$ -lactamase inhibitor

<u>Rebekka Rekkedal Rolfsnes</u><sup>1,2</sup>, P. Rongved<sup>1</sup>, B. Klem<sup>1</sup>, R. Hovland<sup>1</sup>, O.A. Økstad<sup>2</sup>, H.C. Winther-Larsen<sup>2</sup>

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**Background**:  $\beta$ -lactamases are enzymes which destroy  $\beta$ -lactam antibiotics and can roughly be divided into two categories: serine  $\beta$ -lactamases (SBLs) and metallo- $\beta$ -lactamases (MBLs). Carbapenem-resistant strains of *Klebsiella pneumoniae*, *Escherichia coli*, *Pseudomonas aeruginosa* etc. are all on the Priority 1 (critical) part of the WHO priority pathogens list for R&D of new antibiotics. Although there are a few compounds on the market that inhibit the SBLs, no compounds are currently available that targets the MBLs, or both. APC247 is a novel  $\beta$ -lactamase inhibitor, where initial testing for antimicrobial activity has shown the contours of the first dual  $\beta$ -lactamase inhibitor.

**Method**: Susceptibility testing was performed according to CLSI M07-A9 guidelines for broth microdilution method (19th edition). The effect of APC247, at concentrations of 16 and 32 mg/L, on the minimum inhibitory concentration (MIC) of a partner  $\beta$ -lactam (amoxicillin, aztreonam, cefepime, ceftazidime and meropenem) was investigated. A total of 17 isolates harbouring combinations of SBLs and/or MBLs with extended spectrum  $\beta$ -lactamases (ESBLs) and/or carbapenemase activity were included. Isolates included *K. pneumoniae* (n=8) and *E. coli* (n=9). Isolates harbouring ESBLs and MBLs only, were not tested for meropenem and aztreonam respectively, as these are intrinsically susceptible. To investigate if MIC was brought to a therapeutically relevant level, the EUCAST clinical breakpoint table (version 12.0) was consulted.

**Results**: The combination of  $\beta$ -lactams and APC247 at 32 mg/L reduced the MIC for 0/17 (amoxicillin), 9/16 (aztreonam), 12/17 (cefepime), 4/17 (ceftazidime) and 12/15 (meropenem) of the tested isolates to therapeutically relevant levels. At 16 mg/L the same was true for 0/17 (amoxicillin), 5/16 (aztreonam), 8/17 (cefepime), 4/17 (ceftazidime) and 11/15 (meropenem). All strains investigated was brought to a therapeutically relevant level with one/several combinations of APC247 and  $\beta$ -lactam, except for *K. pneumonia* harbouring both bla<sub>OXA-48</sub> (carbapenemase) and bla<sub>CTX-M-15</sub> (ESBL) enzymes.

**Conclusion**: The novel combined MBL/SBL-inhibitor APC247 successfully reduced the MIC of several partner  $\beta$ -lactam antibiotics towards Priority 1 pathogens and achieved therapeutically relevant levels in 16/17 cases. Ongoing studies investigating the effect on the individual  $\beta$ -lactamases will extend our knowledge about the mechanism of action of APC247.

<u>Poster Session III – Group II, Thursday June 8th</u> Meet Rebekka Rolfnes

## Structural elucidation of the interaction between RseP and the LsbB family of bacteriocins

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The extracytoplasmic function (ECF)  $\sigma$ -factors are important for bacterial cells to adapt to environmental stress. In many bacteria, ECF  $\sigma$ -factors are kept inactive by membrane-bound anti  $\sigma$ -factors. During stress,  $\sigma$ -factors are released from the membrane by the proteolytic action of the intramembrane zinc-metalloprotease RseP in a process called regulated intramembrane proteolysis. The genes activated by the  $\sigma$ -factor promote stress tolerance. Examples include the essential ECF  $\sigma^{E}$  in *Escherichia coli* and the lysozyme-activated  $\sigma^{V}$  in *Enterococcus faecalis*, both of which are controlled by RseP. RseP is also reported to be involved in the removal of signal peptides from the membrane and the release of pheromones in *Staphylococcus aureus* and other Gram-positive bacteria.

RseP has also been shown to act as receptor for the LsbB family of bacteriocins, a family of short α-helical peptides that bind specifically to RseP to quickly cause cell death by an unknown mechanism. RseP is ubiquitous in bacteria and shows great promise as a therapeutic target for the treatment of multi-resistant pathogens, including vancomycin-resistant *Enterococcus faecium*. The LsbB family bacteriocins could potentially be engineered with great specificity and potency for the treatment of any pathogen that employs RseP. LsbB family bacteriocins selects for the growth of mutants with mutations in RseP *in vitro*, however, these mutants exhibit reduced stress tolerance and can therefore effectively be eliminated by combinatorial treatment with other bacteriocins and/or antibiotics.

In this PhD-project we will further unravel the functional roles of RseP in Gram-positive bacterial pathogens and better understand the killing mechanism of the LsbB family of bacteriocins. We wish to employ single particle Cryo EM of purified RseP bound to bacteriocin. Our structural studies will be complemented with structural predictions using AlphaFold to characterize mutations in *rseP* that render cells insensitive to the bacteriocins. We hope the insight we gain into the interaction between the bacteriocin and RseP can be used to engineer novel LsbB-like bacteriocins for treatment of bacterial infections.

<u>Poster Session III – Group II, Thursday June 8th</u> Meet Simen Hermansen

# Development of a bead-based multiplex immunoassay for diagnosis of canine and human leptospirosis

Vilde Irene Nilsson

Phd NMBU, Oslo, Norway

Leptospirosis is one of the most widespread zoonotic diseases. The clinical presentation of leptospirosis is unspecific, misdiagnosis is frequent, and diagnosis is based upon laboratory results. Current laboratory methods are outdated, time consuming and expensive. They rely on a continuous supply of animal products (rabbit anti-sera) and require specialist expertise and equipment. The current gold standard diagnostic assay for leptospirosis (MAT) cannot determine IgG from IgM antibodies and relies on live cultures, which presents problems in the way of maintenance and attenuation. The aim of this study is to develop a new diagnostic assay for serological diagnosis of leptospirosis that is more specific, sensitive, and able to discriminate between IgG and IgM classes of antibodies—as well as being more cost effective. We will be using a bead-based multiplex immunoassay for the serological detection of Leptospira serovars in dogs and humans in Norway and compare the new method with the current gold standard (MAT) and IgM snap test.

<u>Poster Session III – Group II, Thursday June 8th</u> <u>Meet Vilde Irene Nilsson</u>

### Isolation of Atlantic Salmon skin cells: A preliminary study

Vismai Naik Thuppe, Jaya Kumari Swain

Fish Immunology and Vaccinology group, Norwegian College of Fishery Science, UiT-The Arctic University of Norway, Tromsø, Norway

Salmon lice (*Lepeophtheirus salmonis*) is a crustacean ectoparasite causing large scale losses in the Salmon aquaculture industry globally due to slower growth of fish, mortality or the need for chemical delousing. Previous studies have shown differential susceptibility exists between Salmonid species with Atlantic salmon being most susceptible. This difference in susceptibility can even be seen within a population. The infected fish show delayed or reduced inflammation and wound healing at the site of parasite attachment, so it is important to understand the interaction between the parasite and Salmon during infestation. Local immune response is crucial during early infestation but as the parasite metamorphoses, it gains the ability to move over the host making it difficult to mount a strong local immune response. Skin is one of the first line of defense against infections in fish and an important mucosal tissue. Understanding the immune factors in skin and the scale of their response during infection is essential to determine the role played by mucosal immune system in fish skin. Along with the various immune factors we are also trying to identify the cells in Atlantic salmon skin that are important during Salmon lice infestation.

Poster Session III – Group II, Thursday June 8th

Meet Vismai Naik Thuppe

### **Poster instructions**

Each student has been assigned a poster group and a poster number (see below). Poster group 1 mounts and presents their posters in St.Petri while poster group 2 mounts and presents their poster in Gregerstua

The posters are numbered from 1-24, make sure to hang your poster on the board marked with your poster number.

Poster Nr	Poster group	Presentation date	Name
1	1	June 6	Amanda H. Singleton
2	1	June 6	Asal Ahmadi
3	1	June 6	Beathe Kiland Granerud
4	1	June 6	Bente Sved Skottvoll
5	1	June 7	Charlotte S. Krog
6	1	June 7	Charlotte Årseth
7	1	June 7	Collins GK Atuheire
8	1	June 7	Hesham Amin
9	1	June 8	Ingeborg Mathiesen
10	1	June 8	Ingeborg Yddal
11	1	June 8	Jorge Agramont
12	1	June 8	Josue Mamani Jarro
13	2	June 6	Klara Andersson
14	2	June 6	Lise Benette Hovd
15	2	June 6	Lukas Hoen
16	2	June 6	Maria Naqvi
17	2	June 7	Pérez Syltern, Marita
18	2	June 7	MitchellRey Toleco
19	2	June 7	Ninsiima Lesley Rose
20	2	June 7	Rajneesh Kumar
21	2	June 8	Rebekka Rekkedal Rolfsnes
22	2	June 8	Simen Hermansen
23	2	June 8	Vilde Irene Nilsson
24	2	June 8	Vismai Naik Thuppe

Three **poster sessions** are organised on Tuesday, Wednesday and Thursday. During the poster sessions one person in each group will present at a time (10 min for presentation and 5 min for questions). When the first person finishes his/her presentation, the next person in the group starts, everyone else in the group listens.

Local IBA members and the lecturers will also be invited to listen to the poster presentations. Tentatively you can expect 10-15 people listening to your presentation

In the session called **"free poster viewing of posters of the day"** all the presenters of the given day will be present at their poster for discussions and questions. This is the time where group I can listen to presentations in group II and vice versa.

## Group 1 – Session I – Tuesday June 6th

- Amanda Singleton Meet <u>Amanda</u> Signalomic and metabolomic analysis of a beta-clamp inhibitor and putative thymidylate monophosphate kinase inhibitor. Abstract <u>poster 1</u>
- Asal Ahmadi Meet <u>Asal</u> Pathways- Enlightening the role of *Pasteurella multocida* in the pathogenesis of bovine respiratory disease (BRD) Abstract <u>poster 2</u>
- **3.** Beathe Kiland Granerud Meet <u>Beathe</u> Omicron Variant Generates a Higher and More Sustained Viral Load in Nasopharynx and Saliva than the Delta Variant of SARS-CoV-2. Abstract <u>poster 3</u>
- 4. Bente Skved Skottvoll Meet <u>Bente</u> The resistome in general and clinical populations and their associated environment. Abstract <u>poster 4</u>

## Group 2 – Session I – Tuesday June 6th

- Klara Andersson- Meet <u>Klara</u> CRISPR-based antivirals against emerging viruses. Abstract <u>poster 13</u>
- Lise Benette Hovd Meet Lise Bovine Intestinal Organoids as an *in vitro* infection model for *Cryptosporidium Parvum*. Abstract <u>poster 14</u>
- 15. Lukas Hoen -Meet Lukas Comparing the Humoral Immune Responses Induced Following Live Attenuated Influenza Vaccine Immunization in Children & Adults. Abstract poster 15
- **16.** Maria Naqvi Meet Maria The Composition of the Ocular Surface in Dry Eye Disease Patients. Abstract poster 16

## Group 1 – Session II - Wednesday June 7th

- 5. Charlotte Krog– Meet <u>Charlotte</u> Investigation of TisB induced pH depolarization in *Escherichia coli* following DNA damage and the role of ATP synthase as a potential protein partner. Abstract <u>poster 5</u>
- 6. Charlotte Årseth Meet <u>Charlotte</u> The Role of Intracellular Complement in *Mycobacterium Tuberculosis* Infection. Abstract <u>poster 6</u>
- 7. Collins GK Atuheire Meet <u>Collins</u> Epidemiology of Rabies in Eastern Rural Uganda: A One Health Approach. Abstract <u>poster 7</u>
- 8. Hesham Amin Mohamed Abouelhana Meet <u>Hesham</u> Association between indoor bacterial communities, lung function and airway inflammation. *Abstract* <u>poster 8</u>

### Group 2 – Session II - Wednesday June 7th

- Marita Pérez Syltern Meet Marita Comparing the metabolomic profile of clinical resistant and sensitive *E. coli* strains through non-targeted HR-MS analysis. Abstract poster 17
- Mitchellrey Magbanua Toleco Meet <u>Mitchellrey</u> Tales of a Mighty Conqueror: Insights into *Blastocystis* Metabolism. Abstract <u>poster 18</u>
- Ninsiima Lesley Rose- Meet <u>Ninsiima</u>
  BAT ECOLOGY AND SOCIAL EPIDEMIOLOGY OF *FILOVIRIDAE* HAEMORRAGIC FEVERS IN UGANDA. Abstract <u>poster 19</u>
- 20. Rajneesh Kumar <u>Rajneesh</u> Genetic Circuitry's Response to Energy Availability in Decision-Making Processes. Abstract <u>poster 20</u>

## Group 1 – Session III - Thursday June 8th

- Ingeborg Mathiesen Meet Ingeborg CHARACTERISATION OF PUTATIVE VIRULENCE FACTORS IN ENTEROCOCCUS FAECIUM Abstract poster 9
- 10. Ingeborg Yddal Meet <u>Ingeborg</u> Respiratory virus surveillance during an effectiveness trial to evaluate the protection of children and pregnant women by influenza vaccine in rural Bangladesh. Abstract <u>poster 10</u>
- Jorge Agramont Meet Jorge Tracking Antibiotic Resistance Genes in rivers polluted by Sewage Water in La Paz Bolivia. Abstract poster 11
- Josue Mamani Jarro- Meet Josue Structure and Diversity of Environmental Resistomes in aquatic environments of the Seke and Seco Rivers in El Alto-Bolivia. *Abstract* poster 12

## Group 2 – Session III - Thursday June 8th

- 21. Rebekka Rolfnes Meet <u>Rebekka</u> Investigating the mechanism of action of a new novel dual β-lactamase inhibitor Abstract <u>poster 21</u>
- 22. Simen Hermansen Meet <u>Simen</u> Structural elucidation of the interaction between RseP and the LsbB family of bacteriocins. Abstract <u>poster 22</u>
- 23. Vilde Irene Nilsson, Meet <u>Vilde Irene</u> Development of a bead-based multiplex immunoassay for diagnosis of canine and human leptospirosis. Abstract<u>poster 23</u>
- Vismai Naik Thuppe Meet <u>Vismai</u>.
  Isolation of Atlantic Salmon skin cells: A preliminary study. Abstract <u>poster 24</u>

## The Norwegian PhD School in Infection Biology and Antimicrobials (IBA)

IBA is a national training network for PhD students and postdocs active in infection biology and antibiotics research in Norway. The programme is anchored at the University of Oslo and includes the partnering institutions UIT - The Arctic University of Norway, Norwegian University of Science and Technology, Norwegian University of Life Sciences, University of Bergen and Norwegian Institute of Public Health.

IBA aims to connect these research environments by offering advanced training courses, workshops, and conferences that promote research and recruitment of young scientists. IBA supports participation in these activities and relevant international courses via co-funding of travel and accommodation costs. We also offer co-funding for research visits in Norway and abroad.

IBA is funded through the Research Council of Norway (RCN/NFR).

For additional information please visit: www.ibaschool.no.

