UCMR Day 2026

29 January, Rotundan

Programme - Abstracts - Posters - Participants

UCMR
Umeå Centre for Microbial Research

UMEÅ UNIVERSITY

Welcome to UCMR Day 2026!

We are delighted to see such strong interest, with 190 participants registered to the conference. UCMR Day continues to play an important role in strengthening our community and encouraging interdisciplinary collaborations. This year marks the 17th edition of our annual conference.

The event brings together scientists and staff within the Umeå Centre for Microbial Research (UCMR), united by a shared interest in microbial research and infection biology.

The programme includes two keynote lectures by Giuseppe Balistreri (University of Helsinki) and Lea Klingenberg Barfod (University of Copenhagen). Giuseppe specializes in virus entry and antiviral strategies, while Lea's expertise lies in malaria immunology and vaccine development.

A special focus is placed on early-career researchers, with many PhD students and postdocs presenting their work through 10-minute talks or pitches, and a poster session. The awards for best talk and best pitch are sponsored by UBI.

We conclude the event with a panel discussion, where experts from different fields come together to discuss "Where are we heading? Future research directions in infection biology."

We hope you enjoy the programme and take the chance to connect with colleagues – both familiar faces and new ones – who share your passion for microbial research.

Ingrid Söderbergh, Research Coordinator UCMR

On behalf of the scientific organising committee:

Fredrik Almqvist, Director of UCMR, Department of Chemistry

Marta Bally, Deputy Director UCMR, Department of Clinical Microbiology

Laura Carroll, Department of Clinical Microbiology

Verena Kohler, Department of Molecular Biology

Programme

8:30 – 9:00	**REGISTRATION and MOUNTING OF POSTERS**
9:00 – 9:05	**WELCOME!** Katrine Riklund, Dean Faculty of Medicine, Umeå University
9:05 – 9:15	**UCMR UPDATE** Fredrik Almqvist, Director UCMR, Umeå University
9:15 – 10:00	**KEYNOTE LECTURE 1** *Chair: Verena Kohler* **Targeting complement immune evasion mechanism in *P. falciparum* for the development of next generation malaria vaccines** **Lea Klingenberg Barfod, professor at the Centre for Translational Medicine and Parasitology, University of Copenhagen**
10:00 – 10:30	*Coffee break*
10:30 – 11:10	**SHORT TALKS 1** *(10 minutes talks)* *Chair: Laura Carroll*
Poster #34	**Same receptor, different role: How heparan sulfate chemistry shapes adenovirus binding behaviour for cell entry** Fouzia Bano, Department of Clinical Microbiology, Umeå University
	Large-scale insights into the biosynthetic potential of the *Bacillus cereus* group Josefin Blom, Department of Clinical Microbiology, Umeå University
	Identifying DNA methylation patterns in post COVID-19 condition: Insights from a one-year prospective cohort study Christoffer Granvik, Department of Clinical Microbiology, Umeå University
	Long-term evolution of PUUV-specific humoral immune response following primary infection Felicia Hamrin, Department of Clinical Microbiology, Umeå University
11:10 – 11:20	*Short break*

Programme

11:20 – 12:00	**SHORT TALKS 2** *(10 minutes talks)* *Chair: Verena Kohler* **Protein O-glycosylation in the Bacteroidota phylum** Victor Pinedo, Department of Chemistry, Umeå University **Synthesis of ring-fused 2-pyridone derivatives to target FadA protein of *Fusobacterium nucleatum* involved in colorectal cancer** Shalini Shalini, Department of Chemistry, Umeå University **Prokaryotic carbon cycling and metabolic adaptations in Central Arctic Ocean** Ashish Verma, Department of Ecology, Environment and Geoscience, Umeå University
12:00 – 13:00	*LUNCH*
13:00-13:40	**SHORT TALKS 3** *(10 minutes talks)* *Chair: Fredrik Almqvist*
Poster #35	**Immune landscape and microbiome-associated signatures in pancreatic ductal adenocarcinoma: Insights from murine and organoid models** Vasileios Vardas, Department of Molecular Biology, Umeå University **Uncovering a role for hyaluronan in Puumala virus-induced pulmonary disease** Alfred Wennemo, Department of Clinical Microbiology Umeå University
Poster #36	**Antifungal activity of isatin on *Saprolegnia spp.*: *In vitro* and *in vivo* evaluation in salmonid eggs** Tulio Teruo Yoshinaga, Department of Clinical Microbiology, Umeå University
13:40 – 13:50	*Short break*

Programme

13:50 – 14:30 **ELEVATOR PITCHES** *(2-3 minutes talks)*

Chair: Marta Bally

Poster #1 **Human *in vivo* genetic screening for novel CAR T cell design**

Florian Albrecht, Department of Clinical Microbiology, Umeå University

Poster #2 **Medium-dependent modulation of fosfomycin susceptibility by the Cpx envelope stress response in *Yersinia pseudotuberculosis***

Kumar D. Gahlot, Department of Molecular Biology, Umeå University

Poster #3 **Resolving microbial genomes in complex communities at the single-cell level**

Jyoti Verma, Department of Clinical Microbiology, Umeå University

Poster #4 **One-year evaluation of immunogenicity and antibody-dependent enhancement risk of a single-dose KD-382 live-attenuated tetravalent dengue vaccine in flavivirus-naïve adults**

Jean Claude Balingit, Department of Clinical Microbiology, Umeå University

Poster #5 **Tracking antimicrobial resistance in the gut microbiome using single-cell metagenomics**

Chaitra Shanka, Department of Molecular Biology, Umeå University

Poster #6 **Investigation of the potential role of *Parvimonas micra* in colorectal cancer**

Marina Rubio Garcia, Department of Medical Biosciences, Umeå University

Poster #7 **Three-dimensional reconstruction of aquatic microbes using cryogenic electron tomography**

George Westmeijer, Department of Ecology, Environment and Geoscience, Umeå University

Poster #8 **Kidney injury post COVID-19 – a nationwide Swedish register study**

Hanna Jerndal, Department of Clinical Microbiology, Umeå University

Poster #9 **Supported lipid bilayers displaying varied complexity as platforms to study bacteriophage interactions with Salmonella outer membranes**

Hudson Pace, Department of Molecular Biology, Umeå University

Programme

Poster #10	**Targeting the glue: Structural and thermodynamic insights into adhesin-mediated pathogenesis in *enterococci*** Taylor Devlin, Department of Medical Biochemistry and Biophysics, Umeå University
Poster #11	**Serological surveillance of respiratory infections in older adults and young children to characterize age-specific immune response patterns** Preeti Moar, Department of Clinical Microbiology, Umeå University
Poster #12	**Real-time genomic surveillance of *Bacillus cereus sensu lato*, an emerging pathogen group** Vignesh Ramnath, Department of Clinical Microbiology, Umeå University
Poster #13	**Linking high-throughput CRISPR/Cas9 screens with rich phenotyping in malaria parasites** Zhaoshan Chen, Department of Molecular Biology, Umeå University
14:30 – 14:35	**SPONSORS PITCH** Ziming Zhong, BMKGENE – Multi-omics Sequencing Services
14:35 – 14:50	**RESEARCH INFRASTRUCTURE PITCHES** Irene Martinez Carrasco: Biochemical Imaging Centre Umeå. BICU Johan Olofsson Edlund: BioMolecular Characterization Umeå, BMCU Ellen Bushell: Umeå Centre for Comparative Biology, UCCB Sara Henriksson: Umeå Centre for Electron Microscopy, UCEM
14:50 – 16:15	**POSTER SESSION and Coffee break** *Posters are shown in Brashörnan* *Coffee and cake are served in the Lounge*
16:15-17:00	**PANEL DISCUSSION** **Where are we heading? Future research directions in infection biology** *Moderator: Verena Kohler* *Panel:* *Jörgen Johansson, Department of Molecular Biology* *Lea Klingenberg Barfod, keynote speaker* *Johan Normark, Department of Clinical Microbiology* *Martin Rosvall, Department of Physics* *Anna Överby Wernstedt, Department of Clinical Microbiology*
17:00 – 17:10	*Short break*

Programme

17:10 – 17:55	**KEYNOTE LECTURE 2** *Chair: Anna Överby Wernstedt* **A novel technology to follow viral brain invasion in real time and new approaches to block infection** **Giuseppe Balistreri, Group leader Viral Cell Biology Laboratory, University of Helsinki**
17:55 – 18:00	**POSTER REWARDS & CONCLUDING REMARKS** *Pia Keyser, Umeå Biotech Incubator* *Fredrik Almqvist and Marta Bally, Directors UCMR*
18:00 – 18:30	*Reception with mingle* *The reception takes place in the Lounge*
18:30 – 21:00	*DINNER* *The dinner takes place in the Universum restaurant*

Prof. Lea Klingenberg Barfod

Centre for Translational Medicine and Parasitology (CMP), University of Copenhagen, Denmark

Title of the lecture:

"Targeting complement immune evasion mechanism in *P. falciparum* for the development of next generation malaria vaccines"

Abstract: *Plasmodium falciparum* is responsible for the majority of malaria infections and deaths worldwide. Natural immunity to blood stage infection is acquired over several exposures to the parasite and is thought to rely on antibodies. Antibodies can protect from severe disease through different effector functions, with complement activation lately emerging as an important feature of protective humoral responses to malaria infection. Malaria parasites have, however, evolved several mechanisms to evade complement attack, including the recruitment of complement down-regulatory proteins like Factor H (FH) and C1 esterase inhibitor (C1-INH) to the merozoite. Current vaccine development strategies include targeting merozoite antigens, we therefore wanted to investigate the impact of the complement evasion mechanism and the possibilities that it can be counteracted by antibodies or other reagents.
In this talk, I will present data on the role of naturally acquired antibodies towards the malaria antigens involved in the evasion mechanism, as well as our discovery of a third complement regulatory protein recruited to the merozoite surface. Further the development of *de novo* designed minibinders binding both the recruiting antigens as well as the current blood stage vaccine candidate RH5 will be presented.

Summary of research: Monoclonal antibodies (mAbs) and *P. falciparum* immune evasion mechanisms have been the focus of my research throughout my career. I was the first to generate human mAbs specific for malaria variant surface antigens. These mAbs have been excellent tools to identify important protective epitopes and immune evasion mechanisms (Barfod 2007, Barfod 2010, Joergensen 2010, Barfod 2011, Stevenson 2014, Jeppesen 2015, Raghavan 2022). The mAbs have further been used for manipulating the expression of antigens on the surface of the infected red blood cell and identifying an unappreciated link between the antigens expressed and the morphology of the infected cell (Soerli 2009,

Subramani 2015). I have analysed acquisition and kinetics of malaria specific antibodies and memory B cells in Ghanaian women and children with acute malaria (Ampomah 2014A, 2014B, Partey 2018, Walker 2020). At University of Oxford, I was involved in the development of a vaccine based on the *P. falciparum* antigen PfRH5 that has since been examined extensively in human trials (Minassian 2021). I generated more than 50 mAbs against merozoite antigens involved in erythrocyte invasion (Campeotto 2017 and 2020, Payne 2017, Illingworth 2019, William 2024). In 2018 I returned to University of Copenhagen to establish the EPITOPE team with the aim of rationally elucidating the characteristics of highly protective antibody epitopes of complex immune targets. We used *P. falciparum* as a model pathogen and identified synergistic antibody combinations as well as created short peptides harbouring protective antibody epitopes (Walker *2020,* Knudsen *2021,* Knudsen *2022*), transformed the knowledge into novel malaria vaccines (Björnsson 2024) and have now started identifying the impact of complement evasion in naturally immune individuals (Bassi 2025).

Area of expertise: *Malaria immunology, monoclonal antibody discovery, epitope characterisation, immune evasion mechanisms, and vaccine development*

More reading: https://researchprofiles.ku.dk/en/persons/lea-klingenberg-barfod/

Dr. Giuseppe Balistreri

Viral Cell Biology Laboratory, Department of Virology, University of Helsinki, Finland

Title of the lecture:

"A novel technology to follow viral brain invasion in real time and new approaches to block infection"

Abstract: Viruses must co-opt host cellular resources to enter and infect cells, yet our ability to study neuroinvasion is limited by the lack of non-invasive tools that reveal when and how viruses first reach the brain.

This lecture will introduce a new, non-invasive eye imaging technology that detects viral penetration into the central nervous system days before clinical symptoms emerge. By enabling early, real-time readouts, this platform opens a window onto the mechanisms of neuroinvasion, the timing and quality of initial immune responses, and the real-time efficacy of antivirals.

The second part will showcase novel host-directed antiviral strategies that disrupt cellular enzymes governing protein lipidation. I will highlight how pharmacologic inhibition of these pathways provides a broad-spectrum approach that impairs essential steps of viral replication across multiple families. Together, these advances pair earlier detection of brain invasion with host-targeted intervention, charting a path toward pre-symptomatic diagnosis, improved therapeutic monitoring, and deeper understanding of the early events in viral neuroinvasion.

Academic background:

Master degree in molecular biology, University of Palermo, Italy

PHD Molecular virology, University of Helsinki, Finland

Postdoctoral fellow, ETH ZURICH, Switzerland. Virus entry and genome wide screening approaches

Since 2018, Group leader, Viral Cell Biology Laboratory, University of Helsinki, Finland

Area of expertise: *Virus entry and antivirals*

More reading: https://researchportal.helsinki.fi/en/persons/giuseppe-balistreri/

Poster List

Poster number	Talk	Presenter's name	Poster title	Research group
#1	Elevator pitch	**Florian Albrecht**	Human *in vivo* genetic screening for novel CAR T cell design	Johan Henriksson
#2	Elevator pitch	**Kumar D. Gahlot**	Medium-dependent modulation of fosfomycin susceptibility by the Cpx envelope stress response in *Yersinia pseudotuberculosis*	Matthew Francis
#3	Elevator pitch	**Jyoti Verma**	Resolving microbial genomes in complex communities at the single-cell level	Johan Henriksson
#4	Elevator pitch	**Jean Claude Balingit**	One-year evaluation of immunogenicity and antibody-dependent enhancement risk of a single-dose KD-382 live-attenuated tetravalent dengue vaccine in flavivirus-naïve adults	Mattias Forsell
#5	Elevator pitch	**Chaitra Shankar**	Tracking antimicrobial resistance in the gut microbiome using single-cell metagenomics	Johan Henriksson
#6	Elevator pitch	**Marina Rubio Garcia**	Investigation of the potential role of *Parvimonas micra* in colorectal cancer	Richard Palmqvist
#7	Elevator pitch	**George Westmeijer**	Three-dimensional reconstruction of aquatic microbes using cryogenic electron tomography	Linda Sandblad
#8	Elevator pitch	**Hanna Jerndal**	Kidney injury post COVID-19 — a nationwide Swedish register study	Anne-Marie Fors Connolly
#9	Elevator pitch	**Hudson Pace**	Supported lipid bilayers displaying varied complexity as platforms to study bacteriophage interactions with *Salmonella* outer membranes	Marta Bally
#10	Elevator pitch	**Taylor Devlin**	Targeting the Glue: Structural and Thermodynamic Insights into Adhesin-Mediated Pathogenesis in Enterococci	Ronnie Berntsson
#11	Elevator pitch	**Preeti Moar**	Serological surveillance of respiratory infections in older adults and young children to characterize age-specific immune response patterns	Mattias Forsell
#12	Elevator pitch	**Vignesh Ramnath**	Real-time genomic surveillance of *Bacillus cereus sensu lato*, an emerging pathogen group	Laura Carroll
#13	Elevator pitch	**Zhaoshan Chen**	Linking high-throughput CRISPR/Cas9 screens with rich phenotyping in malaria parasites	Ellen Bushell
#14		**Daniel Nilsson**	Syringe Holder with Synchronized Push-Pull	Magnus Andersson
#15		**Aicha Kriaa**	Mucus for Dinner: Chasing Commensal Gut Degraders	Björn Schröder

Poster List

Poster number	Talk	Presenter's name	Poster title	Research group
#16		**Supapit Wongkuna**	Milk-derived casein glycomacropeptide improves colonic mucus function under Western-style diet feeding in a sialylation-dependent manner	Björn Schröder
#17		**Linnea Vikström**	A Multicenter Phase IV Study of Long-term COVID-19 Vaccine Immunogenicity Across Four Regions in Sweden	Mattias Forsell
#18		**Olga Panagiotopoulou**	Global genomic surveillance of *Salmonella enterica* serotype Mbandaka sequence type 413	Laura Carroll
#19		**Marie Sorin**	Structural characterization of Human Adenoviruses D56 and D36	Lars-Anders Carlson
#20		**Yong-Dae Gwon**	Dissecting placental host-pathogen interactions: RVFV infection in early human trophoblast stem cells	Magnus Evander
#21		**Zeinab Razooqi**	The Role of a New Oral Bacterial RTX protein in Gum Disease From Early Detection to Future Treatments	Jan Oscarsson
#22		**Koushikul Islam**	Investigating the role of 2-pyridones as antiviral compounds against enterovirus D68 infection	Niklas Arnberg
#23		**Malgorzata Graul**	Molecular mechanisms behind filovirus entry: exploring cell attachment and entry potential of different filovirus-encoded glycoproteins	Marta Bally
#24		**Rebecca Lantto**	Tracing the Genetic Landscape of Puumala Virus in Sweden: Insights from 1990s to Present	Anne Tuiskunen Bäck
#25		**Monique Johnson**	Organ barseq screens in *Plasmodium berghei* : a new tool for identifying critical mediators of malaria parasite sequestration	Ellen Bushell
#26		**Antonio Rodríguez-Blázquez**	Deciphering the molecular mechanism of a novel cryptosporidiosis therapy	Christian Hedberg
#27		**Aakriti Singh**	From Discovery to Validation: Advancing a Selective FabH Inhibitor for Treating *Chlamydia* Infections	Barbara Sixt
#28		**Lonneke Hoffmanns**	Protein O-glycosylation in the Bacteroidota phylum	André Mateus
#29		**Kiran Bala Sharma**	Non-canonical roles of autophagy in enterovirus infection	Lars-Anders Carlson
#30		**Karthikeyan Pandi**	Mapping Determinants of Pathogen Vacuole Stability and Host Defense During Infection with *Chlamydia trachomatis*	Barbara S. Sixt

Poster List

Poster number	Talk	Presenter's name	Poster title	Research group
#31		**Dario Conca**	Direct observation of filovirus interaction with the endothelial glycocalyx	Marta Bally
#32		**Girish Malagi**	Durable immune memory to human hantavirus Infection	Mattias Forsell
#33		**Maud Mutsaers**	The effect of temperature on the vector competence of *Anopheles stephensi* for o'nyong nyong virus	Magnus Evander
#34	Short talk	**Fouzia Bano**	Same Receptor, Different Role: How Heparan Sulfate Chemistry Shapes Adenovirus Binding Behaviour for Cell Entry	Marta Bally
#35	Short talk	**Vasileios Vardas**	Immune Landscape and Microbiome-Associated Signatures in Pancreatic Ductal Adenocarcinoma: Insights from Murine and Organoid Models	Teresa Frisan
#36	Short talk	**Tulio Teruo Yoshinaga**	Antifungal Activity of Isatin on Saprolegnia spp.: *In Vitro* and *In Vivo* Evaluation in Salmonid Eggs	Constantin Urban
Posters by Research Infrastructure				
#37		**Jeanette Tångrot**	National Bioinformatics Infrastructure Sweden (NBIS)	
#38		**Tugrul Doruk**	µNordic Single Cell Hub	
#39	Pitch	**Irene Martinez Carrasco**	BICU. Biochemical Imaging Centre Umeå	
#40	Pitch	**Johan Olofsson Edlund**	BMCU. BioMolecular Characterization Umeå	
#41	Pitch	**Ellen Bushell**	UCCB. Umeå Centre for Comparative Biology	
#42	Pitch	**Sara Henriksson**	UCEM. Umeå Centre for Electron Microscopy	

Human *in vivo* genetic screening for novel CAR T cell design

Florian Albrecht[a,c,d], **Laura M. Carroll**[b,c,d], **Johan Henriksson**[a,c,d]

[a]*Department of Clinical Microbiology, SciLifeLab, Umeå University, Umeå, Sweden*
[b]*Department of Molecular Biology, SciLifeLab, Umeå University, Umeå, Sweden*
[c]*Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden*
[d]*Integrated Science Lab (IceLab), Umeå University, Sweden*

Abstract: Less than 8% of cancer therapies, developed in animal models, are successful in humans. This requires a novel method to replace animal models with more faithful alternatives. Here, we aim to improve CAR T cells by developing the first human *in vivo* faithful genetic screen in cancer patients, to uncover novel genetic programming opportunities. Screening in mice, *in vivo*, has been done using perturb-seq – a CRISPR/Cas9 simultaneous knock-out screen, followed by measuring knock-out impact on the transcriptome. This has been done using single-cell technology and oligo arrays to screen genome-wide in a single experiment. However, using this technology on humans is unethical, as random knockouts may harm the patient. To overcome this problem, we will exploit the natural off-target knockouts during that occur randomly during CAR T-cell production, when the CAR-containing virus inserts into a gene, obviating the use of target-gene knockout using CRISPR/Cas9.

This project is enabled by a new type of microfluidics that generate semi-permeable capsules, providing more flexibility to measure multiple aspects of, and perform complex protocols on, a single cell. Our lab is one of the first in Europe to have access to the required technology. We are have started the process of developing the required wetlab protocol. This includes (1) the inverse PCR to detect lentivirus insertion site and (2) RNA-seq to analyze the associated transcriptome. The data will be analyzed using or novel single-cell computational pipeline, which is the first toolbox tailored for single-cell genome analysis.

Medium-dependent modulation of fosfomycin susceptibility by the Cpx envelope stress response in *Yersinia pseudotuberculosis*

Kumar D. Gahlot[a, b], **Emma Readwin**[a, b], **and Matthew Francis**[a, b]

[a]*Department of Molecular Biology, Umeå University, Umeå, Sweden*
[b]*Umeå Centre for Microbial Research (UCMR), Umeå University, Umeå, Sweden*

Abstract: The rise of antimicrobial resistance necessitates a deeper understanding of bacterial stress responses to existing antibiotics. Fosfomycin, a phosphoenolpyruvate analogue targeting *murA*, exhibits variable efficacy influenced by uptake systems and stress pathways. This study examines the role of the Cpx envelope stress response in regulating fosfomycin susceptibility in *Yersinia pseudotuberculosis*.

Cpx pathway activation was measured using a p*cpxP*::lacZ β-galactosidase translational reporter and immunoblotting. Envelope integrity was evaluated *via* light microscopy to detect cytoplasmic leakage and independently through live/dead cells counting to assess cell viability. Transcriptional profiling by qRT-PCR targeted genes involved in catalysis of the first committed step in peptidoglycan biosynthesis (*murA*), envelope stress (*cpxP, htpG, ldtD*), antibiotic transport and efflux (*glpT, ydhE1, ydhE2*, *acrB*), central metabolism (*ptaA, ackA*), and global regulation (*deoR*).

Cpx activation significantly affected fosfomycin susceptibility depending on the medium. In LB broth, activation increased susceptibility and altered the expression of transporter and efflux genes. In Mueller–Hinton broth (MHB), susceptibility decreased, with distinct transcriptional changes. Microscopy and viability tests confirmed different envelope stress responses across media.

These findings identify the Cpx system as a key regulator of fosfomycin effectiveness in *Y. pseudotuberculosis*, with medium composition critically influencing stress response dynamics. This highlights the importance of environmental context in antimicrobial susceptibility testing and treatment development.

Resolving microbial genomes in complex communities at the single-cell level

Jyoti Verma[a,b,c,e*], **Hadrien Gourlé**[a,b,c*], **Iryna Yakovenko**[a,b,c,d*], **Julian Dicken**[a,d], **Florian Albrecht**[b,c,d,e], **Linas Mažutis**[d,g], **Johan Normark**[a,b,c], **Nongfei Sheng**[f], **Nicklas Strömberg**[f], **Tommy Löfstedt**[h], **Laura M. Carroll**[a,b,c,e#], **Johan Henriksson**[c,d,e#]

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[b]*Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå University, Umeå, Sweden*
[c]*Umeå Centre for Microbial Research (UCMR), Umeå University, Umeå, Sweden*
[d]*Department of Molecular Biology, SciLifeLab, Umeå University, Umeå, Sweden*
[e]*Integrated Science Lab (IceLab), Umeå University, Umeå, Sweden*
[f]*Department of Odontology, Umeå University, Umeå, Sweden*
[g]*Life Sciences Center, Vilnius University, Vilnius, Lithuania*
[h]*Department of Computing Science, Umeå University, Umeå, Sweden*

Abstract: Understanding microbial communities at the strain level remains a major challenge in microbiome research. Traditional bulk metagenomics struggles to assemble complete genomes, link plasmids to their hosts, and distinguish closely related strains. Single-cell genome sequencing offers a solution but has been limited by low throughput, high costs, and a focus on eukaryotic single-cell RNA analyses. We present a new single-cell metagenomics method (scMetaG) that uses semi-permeable capsules to process thousands of individual microbial genomes in a single reaction. Our scMetaG protocol utilizes a novel microfluidics approach of the Atrandi Onyx instrument to encapsulate bacterial cells in semi-permeable capsules (SPCs), lyse, and further sequence them. The cell's genomic DNA stays inside the SPC, but due to the semi-permeability of the SPC, enzymes and buffers can be easily exchanged. A mock bacterial community with an even mix of 10 bacterial species was used for the optimization. Lysis being the primary source of bias, combinations of different lysis methods were tried, and species-specific primers were used to assay lysis efficiency. Furthermore, libraries were prepared following Atrandi's instructions and sequenced using Illumina Novaseq X. A complete R-based pipeline (Zorn) was designed for end-to-end analysis of scMetaG data. To manage the large amount of data, a custom file format with an integrated workflow manager (Bascet) was designed. Our method provides an affordable and scalable solution for studying microbiomes at unprecedented resolution, opening new possibilities for microbial ecology, evolution, and functional genomics.

One-year evaluation of immunogenicity and antibody-dependent enhancement risk of a single-dose KD-382 live-attenuated tetravalent dengue vaccine in flavivirus-naïve adults

Jean Claude Balingit[a], **Motoharu Abe**[a,b], **Ryosuke Suzuki**[c], **Dalouny Xayavong**[d], **Mya Myat Ngwe Tun**[a,e], **Yuki Takamatsu**[a,d], **Kengo Sonoda**[b], **Koucihi Morita**[a,d,f]

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[d]*Department of Virology, Institute of Tropical Medicine, Nagasaki University, Japan*
[e]*Center for Vaccines and Therapeutic Antibodies for Emerging Infectious Diseases, Shimane University, Japan*
[f]*DEJIMA Infectious Disease Research Alliance, Nagasaki University, Japan*

Abstract: A robust neutralizing antibody (nAb) response is essential for dengue vaccines to protect against all four dengue virus (DENV) serotypes while minimizing antibody-dependent enhancement (ADE) risk. This study evaluated the immunogenicity and ADE potential of a single-dose KD-382 tetravalent vaccine in 15 healthy, flavivirus-naïve adults from a Phase I clinical trial. Serum samples were collected at 1, 2, 3, 6 and 12 months post-vaccination. To assess cross-neutralizing and cross-enhancing antibody responses, single-round infectious particles (SRIPs) representing 17 DENV genotypes were employed. While some SRIPs shared genotypes with vaccine strains, they were derived from different viral strains, enabling a broader evaluation of genotype-specific responses. Neutralization assays were performed using BHK-21 cells, while ADE assays used BHK-21 cells stably expressing human FcγRIIA to evaluate enhancement across a range of serum dilutions. Peak enhancement titers (PET) were calculated to assess ADE risk. KD-382 induced durable nAb responses to DENV-1, DENV-2, and DENV-4 (GMTs: 151, 179, and 92, respectively), and lower but protective titers to DENV-3 (GMT: 27). Genotype-specific responses were strongest against DENV-1 Genotype II, DENV-2 Asian II and American genotypes. Vaccine-homologous genotypes also elicited high nAb titers. All 17 genotypes demonstrated enhancement *in vitro*; however, PET values remained above the proposed ADE risk threshold (PET >80) and declined over time, with stable enhancement profiles observed from 2 to 12 months, suggesting KD-382-induced antibodies may mitigate ADE risk. The findings support KD-382 as a safe, broadly protective dengue vaccine and highlight the critical need for thorough ADE risk evaluation.

Tracking antimicrobial resistance in the gut microbiome using single-cell metagenomics

Chaitra Shankar[2,3,4], **Iryna Yakovenko**[1,2,3,4], **Jyoti Verma**[1-6], **Julian Dicken**[1,2,3], **Johan Normark**[1,2,3], **Laura Carroll**[1,2,3,5], **Johan Henriksson**[3,4,5]

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[2]*Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå University, Umeå, Sweden*
[3]*Umeå Centre for Microbial Research (UCMR), Umeå University, Umeå, Sweden*
[4]*Department of Molecular Biology, SciLifeLab, Umeå University, Umeå, Sweden*
[5]*Integrated Science Lab (IceLab), Umeå University, Umeå, Sweden*
[6]*Department of Odontology, Umeå University, Umeå, Sweden*

Abstract: The gut microbiome is closely linked to human health and several diseases. It is altered by various factors such as use of antibiotics, infections such as COVID-19 and other conditions including inflammatory bowel disease and Crohn's disease. While existing bulk sequencing technologies can estimate changes in gut microbiome diversity, it is challenging to identify subpopulations within species, hetero-resistance to antimicrobials or horizontal gene transfer (HGT) that spreads antimicrobial resistance. Our study introduces a prototype application of single-cell metagenomics (scMetaG) using the Atrandi Onyx microfluidics system, to analyse individual bacterial cells in faecal specimens from patients before and after antibiotic treatment at different time points. Faecal samples were collected longitudinally from a patient cohort at Norrlunds Universitetssjukhus (NUS) at 5 time points after treatment with one of three antibiotics- penicillin, ciprofloxacin, doxycycline. For each antibiotic, three samples (before, during, and after treatment) collected from three patients will undergo scMetaG (27 total). For the analysis of scMetaG data, we will use our software Bascet/Zorn (http://zorn.henlab.org/), which is the first software package for this purpose. Our study aims to use scMetaG to identify bacterial strains harbouring antimicrobial resistance plasmids within the gut, determine plasmid transfer events between bacterial species (particularly in response to antibiotic exposure) and analyse pre- and post-treatment shifts in the gut plasmidome. Hence, using scMetaG, we will be able to examine changes in bacterial diversity, strain variations, and resistance patterns by sequencing representatives of all bacterial cells in the faecal samples.

Investigation of the potential role of *Parvimonas micra* in colorectal cancer

Marina Rubio Garcia[a], **William Rosenbaum**[a], **Anna Löfgren Burström**[a], **Sofia Edin**[a], **Sun Nyunt Wai**[b], **Richard Palmqvist**[a], **Vicky Bronnec**[a]

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Abstract: Colorectal cancer (CRC) is the second-leading cause of cancer related deaths. Several risk factors contribute to CRC development, some of which can be non-modifiable (*e.g.* age, genetic predisposition), while others can be modifiable (*e.g*. smoking, western-style diet). Recent studies have shown an association between gut microbiota and CRC progression. Risk factors can disrupt the bacterial symbiotic relationship in the human gut, leading to imbalances in microbial composition called dysbiosis, which has been linked to CRC development. Recent studies have highlighted some onco-pathogens, such as *Fusobacterium nucleatum, Parvimonas micra,* and *Peptostreptococcus stomatis,* as being associated with CRC, but their exact role in tumour progression remains unclear.

Full-length 16S sequencing alongside qPCR analysis was used to quantify the relative abundance of *P. micra* in clinical samples. To further investigate microbial interactions, multiple clinical strains of *P. micra* were evaluated for their ability to form biofilms, as well as their interactions with other CRC-related bacteria in biofilm formation. To assess the impact of *P. micra* on CRC progression, cell proliferation and migration assays were conducted. Western blot analysis was performed on protein extracts from CRC cell lines infected with *P. micra* to evaluate the activated pathway.

Results from qPCR analyses and 16S rRNA sequencing confirmed the association of *P. micra* with CRC, in line with our previous findings. Biofilm analysis showed an increase in biomass when *P. micra, F. nucleatum* and *P. stomatis* were co-cultured, suggesting a possible synergistic microbial interaction. Preliminary protein expression analysis showed upregulation of autophagy-related markers, suggesting activation of autophagy pathways.

Our findings support a role for *P. micra* in CRC progression and suggest a possible link to other onco-pathogens through the biofilm synergy. Moreover, *P. micra* might induce autophagy as a survival mechanism. These findings highlight a potential crosstalk between microbial biofilm formation, autophagy induction, and CRC progression.

Three-dimensional reconstruction of aquatic microbes using cryogenic electron tomography

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Abstract: Marine microorganisms play a fundamental role in the global carbon cycle, yet we know little about their morphology. Although representative genomes are available for most marine microbial lineages, we lack information on their cell shape, cell size, and structural adaptation to their environment. This is a big gap, as morphology is strongly tied to microbial ecology as it influences processes such as cell-cell interactions, surface attachment, and nutrient uptake efficiency. Here, we use cryogenic electron tomography to characterize marine microorganisms in high resolution and in 3d. Currently, we are finalizing a protocol that includes water sampling, concentration of cells, plunge freezing, imaging and data processing. In the near future, we will complement the microscopy with genomics to screen the genomes for morphological features (e.g. flagella, pili, or extracellular substances) to try and identify the cells in the micrographs. This identification would allow us to construct an image library of (marine) microorganisms, visualize cell-cell interactions, and do biomass estimates. Overall, these analyses will deliver insights that are impossible to obtain without any direct visualization and will improve our understanding of the ecology of these marine microorganisms.

Kidney injury post COVID-19 – a nationwide Swedish register study

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Background/Aims: This study aimed to determine the **risk of kidney injury following COVID-19** in a Swedish nationwide register-based cohort.

Method: The total Swedish adult population was included in this nationwide multi-registry study from January 1, 2020, to July 26, 2022. COVID-19 date (exposure) was defined as the first SARS-CoV-2 related date registered in SmiNet. Outcome was defined as all first-time events of kidney injury diagnosis (acute, non-specified and chronic) reported with ICD10 codes after COVID-19 date. The Self-Controlled Case Series (SCCS) method, adjusted for deaths, was used to determine the incidence rate ratio (IRR) of kidney injury up to 6 months post-infection.

Results: Among the total 2 426 423 COVID-19 cases, **10 351 first kidney injury events were diagnosed**. The risk of developing kidney injury was increased up to 90 days post-infection. The highest IRR was during the first week of COVID-19 onset, being 56.1 (95% CI 52.5–60). The IRR was elevated longer for men (up to 90 days) than for women (up to 60 days). The highest IRRs were seen in the age group **51–74 years**. **Individuals with higher COVID-19 disease severity** were also at higher risk.

Conclusion: COVID-19 is associated with an increased risk of diagnosed kidney injury, which remained elevated up to **90 days** following infection. The risk was higher for **men, patients with severe COVID-19** and the age group **51–74 years**. These results highlight the importance of **vaccination** to prevent severe COVID-19, and the need for **follow-up** to detect kidney injury following COVID-19.

Supported lipid bilayers displaying varied complexity as platforms to study bacteriophage interactions with Salmonella outer membranes

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Abstract: With the rise of antibiotic resistance within pathogenic bacteria, bacteriophages, the viruses that kill bacteria, have become promising tools for alternative antimicrobial strategies. For this, a thorough understanding of their mechanism of action is required. Lipopolysaccharides (LPS) are a key component in the defensive barrier of the outer membrane of Gram-negative bacteria. The ability to create biomimetic platforms that capture such aspects of the Gram-negative bacteria's surface are key to the systematic study of how bacteriophages overcome such defensive barriers. Herein we demonstrate the ability to readily (< 1hr) form supported lipid bilayers (SLBs) containing varying amounts of LPS from *Salmonella enterica* serovar *Typhimurium* (*S. Typhimurium*). Using quartz crystal microbalance with dissipation (QCM-D) monitoring we demonstrate that SLBs with varying LPS content can be systematically produced. Fluorescent recovery after photobleaching indicates that our protocol forms high quality SLBs. Additionally, native SLBs were created from outer membrane vesicles (OMVs) of *S. Typhimurium* and characterized using QCM-D and fluorescence microscopy. We demonstrate how these systems can be used to probe interactions with O-antigen specific *Salmonella* phages and their tailspike receptor binding proteins. Finally, we show how 2D tracking analysis of fluorescent phages can characterize the behavior of individual phages as they arrive on these biomimetic platforms. Altogether, this work provides a roadmap to create versatile and robust LPS-based platforms that facilitate studies of interactions between phages and Gram-negative bacterial membranes.

Targeting the Glue: Structural and Thermodynamic Insights into Adhesin-Mediated Pathogenesis in *Enterococci*

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Abstract: Gram-positive Enterococci account for a significant fraction of hospital-acquired infections but are often difficult to treat due to their proficiency in colonizing damaged host tissues, forming biofilms, and acquiring and spreading antimicrobial resistance (AMR). These AMR genes and additional virulence factors are often disseminated among the bacterial population via conjugation, the horizontal transfer of mobile genetic elements between cells in physical contact. Type 4 Secretion Systems (T4SSs) facilitate conjugation, and a key feature of many Gram-positive T4SSs are cell surface-anchored adhesion proteins. Adhesins not only stabilize mating pair junctions during conjugation but also promote biofilm formation and adherence to host cells during infection, making them a major factor in why hospital-acquired Gram-positive infections are so difficult to treat. One such adhesin is PrgB, which is encoded on the conjugative pCF10 plasmid from *Enterococci faecalis*. The Berntsson lab has previously characterized a subset of PrgB binding partners involved in biofilm formation, but the features of PrgB-host interactions required for infiltration and colonization remain unknown. Now, we aim to identify host-derived receptors that interact with PrgB, quantify the thermodynamic parameters of binding, and structurally resolve important binding interfaces. The resulting molecular details will explain how these adhesins confer virulence by promoting pathogenic colonization of host tissues, stabilizing mating pairs during conjugation, and initiating biofilm formation.

Serological surveillance of respiratory infections in older adults and young children to characterize age-specific immune response patterns

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Abstract: Older adults in long-term care facilities (LTCFs) and young children are highly vulnerable to severe outcomes from respiratory viral infections. Our study aimed to assess respiratory infection burden in these populations and to characterize age-specific immune response patterns. We analyzed capillary blood samples collected longitudinally from 1,622 LTCF residents (median-age 87 years) across five regions of Sweden over 3.5 years (2021–2024) and serum samples from 188 children (aged 18 months) enrolled in the NorthPop cohort. A multiplex platform was used to quantify antigen-specific IgG/IgM responses to RSV pre-F/post-F/G-protein, influenza-A H1N1/H3N2 HA, influenza-B HA and SARS-CoV-2 spike. Linear mixed models were used to assess the dynamics of antibody levels over time, adjusted for demographics and comorbidities. We observed marked recurrent RSV circulation post relaxation of COVID-19-related visiting restrictions in Swedish LTCFs, indicating substantial under-recognized RSV exposure. Pre-F/post-F IgG ratios increased with age and were significantly higher in older adults compared with young children ($p<0.01$), consistent with repeated boosting of pre-F-focused neutralizing immunity. For influenza, elderly residents exhibited broader cross-reactive antibody profiles across strains, whereas children displayed, strain-specific responses. Moreover, higher RSV pre-F IgG levels during periods of low population immunity in LTCFs predicted increased one-year mortality (OR=1.43, $p=0.024$), underscoring the potential life-saving value of timely RSV vaccination in this population. Future work will integrate data from the NorthPop cohort to define primary RSV and influenza immune signatures and compare these with repeated-exposure responses in older adults. Together, these findings support implementing sustained, age-tailored vaccination strategies and demonstrate the utility of serological surveillance for detecting respiratory virus circulation in vulnerable populations.

Real-time genomic surveillance of *Bacillus cereus sensu lato*, an emerging pathogen group

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Abstract: The ability to cause anthrax, foodborne illness and other non-gastrointestinal infections have been attributed to numerous lineages within the *Bacillus cereus sensu lato (s.l.)*., an emerging pathogen group. Existing public data portals linking whole-genome sequencing (WGS) data to epidemiologically relevant metadata can lead to dangerous pathogen misidentifications when applied to this group. Current taxonomic assignments are inadequate for evaluating *B. cereus s.l.* pathogenic potential at the strain level, as many important virulence factors can be gained, lost, and variably present within and across species boundaries. Moreover, incomplete metadata in general-purpose pathogen databases also makes epidemiological surveillance challenging.

To combat these issues, we developed BTyperDB, an atlas of *B. cereus s.l.* genomes with standardized metadata. The BTyperDB pipeline regularly scans public databases, aggregating all available *B. cereus s.l.* samples, which undergo genome assembly, taxonomic assignment, virulence factor detection and in-silico typing using a variety of methods, along with standardized metadata curation and phylogeny construction. To facilitate real-time phylogenomic surveillance of *B. cereus s.l.* strains in BTyperDB, we provide BTracker, which is an Auspice powered interactive phylogenomic visualization tool. To help users rapidly explore and download the latest genomic (meta)data associated with *B. cereus s.l.* strains, genomes within BTyperDB and BTracker can be queried via an interactive web application (www.btyper.app).

In summary, BTyperDB and BTracker represent high-quality, curated genomic resources, which can help improve real-time epidemiological surveillance and risk evaluation of *B. cereus s.l.* strains.

Linking high-throughput CRISPR/Cas9 screens with rich phenotyping in malaria parasites

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Abstract: Malaria remains one of the most devastating infectious diseases, driven by the parasite *Plasmodium falciparum*. Despite recent advances in antimalarial drugs and vaccines, resistance and limited efficacy continue to pose urgent challenges. This project aims to develop novel strategies to dissect host–parasite interactions and to identify mechanisms underlying parasite survival, immune evasion, and drug resistance. By integrating high-resolution genetic perturbation tools with advanced cell imaging and systems biology approaches, we investigate the functional role of essential parasite genes and host pathways during infection. A key focus is on validating candidate targets for therapeutic intervention and exploring the evolutionary dynamics of resistance under selective pressure. The interdisciplinary framework combines molecular parasitology, computational modeling, and translational research, thereby bridging basic discovery with potential clinical application. Ultimately, the project aspires to contribute to the development of innovative interventions that can strengthen malaria control and accelerate progress towards eradication.

Syringe Holder with Synchronized Push-Pull

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Abstract: When performing experiments in open containers, e.g. culture plates or test tubes, steps like washing, staining, or replenishing the sample medium often require a controlled exchange of liquids. This poses a challenge when the sample volume must be kept constant. One way to mitigate this is by operating two syringes in a push-pull configuration, extracting the old liquid while adding the same amount of new liquid. Since this is hard to do by hand, electronic syringe pumps are often used instead. However, these are expensive to buy and time-consuming to set up. Typically requiring a computer and power supply to run, as well as hoses to reach the sample. So, to combat these challenges, we invented a simple syringe holder that can mechanically synchronize two syringes in a push-pull configuration. This device can also be used on closed systems, like micro-channels, phantoms models, organ-on-a-chip and fuel cells, to prevent a build-up of pressure and allowing for fluidic multiplexing. The *Push2Pull* syringe holders accepts standard disposable syringes of sizes 1-60 mL and can be used out in the field, since it requires no electricity or extra components. The addition/extraction accuracy was measured to more than 98% v/v during the whole travel range. These syringe holders can be 3D printed and assembled in the lab by anyone, while costing less than $10 in materials. With this, we hope to increase the accuracy of fluid exchanges and reduce the risk of mistakes, while providing a cheap and simple option to electric syringe pumps.

Poster #15

Mucus for Dinner: Chasing Commensal Gut Degraders

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Abstract: Deep inside the hidden corridors of our intestines, an invisible army of good bacteria and a powerful mucus barrier work tirelessly to keep us healthy. The mucus, a sticky shield lining the gut, is constantly built and rebuilt to protect the delicate intestinal walls. Beyond its protective function, mucus supplies substrates that sustain beneficial microbes involved in digestion, immunity, and homeostasis. Maintaining this balance is critical: when dietary fibers are lacking, microbial metabolism shifts toward mucus glycoproteins, compromising barrier integrity and exposing the epithelium.

Most research to date on mucus breakdown and metabolism has focused on Carbohydrate-Active Enzymes (CAZymes) which primarily cleave terminal monosaccharides from mucin O-glycans, while the role of O-glycopeptidases (i.e. mucinases) remains largely unexplored.

This project aims to uncover the hidden enzymatic arsenal behind mucin glycan degradation and its link to inflammation. We focus on mapping the repertoire of commensal bacterial enzymes involved in mucus breakdown and understanding their relevance to the disease.

Poster #16

Milk-derived casein glycomacropeptide improves colonic mucus function under Western-style diet feeding in a sialylation-dependent manner

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Abstract: The colonic mucus layer is the primary interface between the host and the gut microbiota. It serves both as an ecological niche for bacteria and a barrier protecting the host from microbial exposure. Disruption of the mucus layer, particularly under Western-style diet (WSD) feeding, increases the risk of infection and inflammation. Here, we identify casein glycomacropeptide (CGMP), a milk-derived glycopeptide, as a novel dietary supplement capable of preserving mucus function under WSD consumption. Notably, we demonstrate that the sialylation level of CGMP is a key determinant of its protective effects. Supplementation of highly sialylated CGMP not only prevented WSD-induced mucus defects but also altered the gut microbiota composition, enhancing beneficial bacterial genera, particularly *Bifidobacterium*. Mechanistically, bacterial shifts were associated with increased production of the short-chain fatty acid propionate, which can induce mucus growth. Our findings thus reveal sialylated CGMP as a promising prebiotic supplement to counteract diet-induced mucus dysfunction, highlighting the importance of protein-bound glycan structures in modulating host-microbiota interaction.

A Multicenter Phase IV Study of Long-term COVID-19 Vaccine Immunogenicity Across Four Regions in Sweden

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Abstract: The CoVacc clinical trial was established to investigate how different COVID-19 vaccination regimens shape long-term immune responses. In Sweden, the initial two-dose program included the adenoviral vector-based Vaxzevria (AstraZeneca) and the mRNA-based Comirnaty (Pfizer–BioNTech) or Spikevax (Moderna), followed by mRNA-only boosters.

We conducted a longitudinal cohort study within CoVacc, including 578 individuals who received varying vaccine regimens. SARS-CoV-2-specific IgG responses against spike (S), nucleocapsid (N), and receptor-binding domain (RBD) were measured after the second and third vaccine doses.

S-specific IgG levels were significantly higher in individuals primed with mRNA vaccines compared to those primed with Vaxzevria, both after the second and third doses. Despite these initial differences, all groups responded robustly to the third mRNA dose, and by day 30 post-dose 3, antibody levels were comparable between homologous mRNA and Vaxzevria-primed groups.

Serological analysis revealed that 48.7% of all samples were positive for N-specific IgG, indicating widespread prior exposure. Individuals with evidence of prior infection exhibited markedly higher S-specific IgG responses following the first vaccine dose compared to naïve individuals.

Detailed analyses in a subset of 39 participants showed that mRNA-primed individuals developed higher frequencies of spike-specific memory B cells and greater IgG avidity after two doses, with differences diminishing after the third dose.

Multivariable analysis confirmed that antibody levels increased significantly with each vaccine dose, and prior infection was associated with stronger responses. Age showed a weak negative association with antibody levels, while sex, BMI, and comorbidity burden had no significant impact.

The CoVacc trial confirms the superior immunogenicity of mRNA vaccines and their strong boosting capacity, even after heterologous priming. Age may modestly influence antibody responses, whereas other demographic factors appear to play a limited role in shaping vaccine-induced immunity in a generally healthy population.

Poster #18

Global genomic surveillance of *Salmonella enterica* serotype Mbandaka sequence type 413

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Abstract: Zoonotic foodborne pathogen *Salmonella enterica* serotype Mbandaka, sequence type 413, (ST413) is a globally widespread Salmonella lineage, which has been isolated from a wide range of sources (e.g., animals and foods, particularly poultry). ST413 has been responsible for human outbreaks around the world and is known for its ability to persist in the environment and form biofilms, suggesting adaptation for survival outside the host. However, despite its relevance to animal and human health, ST413 remains poorly studied.
In this study, we analyzed 2,592 publicly available ST413 genomes from multiple continents and diverse source types. Our aim was to characterize the global distribution, antimicrobial resistance (AMR) determinants, plasmid content and evolutionary dynamics of ST413. Using genomic prediction of AMR genes and plasmid replicons, we identified AMR profiles associated primarily with resistance to tetracyclines (tetA and tetB; 11.36% of genomes), sulfonamides (sul1) and aminoglycosides (aadA1, aadA2, aph(3'')-Ib, aph(6)-Id; 7.65% and 11.68% of genomes, respectively). These AMR determinants were consistently linked to IncHI2, IncHI2A, and RepA_1_pKPC-CAV1321 plasmids (Fisher's exact test, odds ratios 11-Infinity, raw p-values < 10^{-35}), suggesting that these plasmid groups may act as major vehicles for the dissemination of aminoglycoside, sulfonamide and tetracycline resistance determinants.

Phylogenetic and evolutionary analyses revealed geographically structured AMR lineages and indicated multiple emergence events associated with horizontal gene transfer via plasmids. Our findings provide new insights into the worldwide distribution of ST413, the plasmid-mediated dissemination of AMR, and the ecological factors that may contribute to the persistence of AMR determinants in animal production environments.

Structural characterization of Human Adenoviruses D56 and D36

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Abstract. Human adenoviruses (HAdVs) cause a broad range of diseases affecting the respiratory, gastrointestinal, ocular, and lymphoid tissues. Nearly 100 types have been identified and classified into seven species (A–G). Species D adenoviruses represent approximately two-thirds of all known HAdVs and are widely used as vaccine vectors, including in candidate vaccines against SARS-CoV-2, HIV, Ebola virus, RSV, and Zika virus. The efficacy of such vectors strongly depends on how the viral capsid interacts with host cells.

The adenovirus capsid is built from three major proteins: hexon, fiber, and penton base. HAdV-D56 and HAdV-D36 are closely related but exhibit distinct biological properties: HAdV-D56 is associated with rare cases of conjunctivitis, whereas HAdV-D36 has been linked to an increased risk of obesity. HAdV-D56 engages CD46 via its hexon protein, while HAdV-D36 binds specifically to the glycan 4-O-acetyl-Neu5Ac through its fiber knob. Although their hexon proteins are highly conserved, predictions suggest notable differences in their exposed loop regions.

We collected cryo-EM datasets for both viruses, achieving maps at 3.8 Å resolution for HAdV-D56 and 2.8 Å for HAdV-D36. Our goal is to build atomic models and perform a detailed structural comparison, with particular focus on the loop regions that may underlie differences in receptor usage. Interestingly, the HAdV-D36 map shows a well-resolved fiber, unlike HAdV-D56. Thus, through localized reconstruction approaches, we aim to obtain a high-quality fiber structure, which is typically challenging due to its flexibility.

Dissecting placental host-pathogen interactions: RVFV infection in early human trophoblast stem cells

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Abstract. Rift Valley fever virus (RVFV) is a mosquito-borne Phlebovirus and zoonotic pathogen affecting maternal–fetal health. Vertical transmission is linked to miscarriage and severe fetal outcomes, but mechanisms of placental pathogenesis remain unclear. We used first-trimester human trophoblast stem cells (hTSCs) to model infection at the maternal–fetal interface. Immunofluorescence, qRT-PCR, Western blotting, and single-cell transcriptomics showed that hTSCs are highly susceptible to RVFV. Strand-specific viral transcriptomics confirmed the ambisense S segment and revealed preferential transcription of the M and S segments over L. RVFV induced G1 arrest, impairing trophoblast proliferation and differentiation, and drove widespread transcriptional reprogramming, including strong interferon lambda 1 (IFN-λ1) but modest type I interferon responses, and dysregulation of inflammatory and preeclampsia-associated genes such as RUNX1 and TGFBRAP1. Recombinant IFN-λ pretreatment reduced RVFV protein expression, highlighting hTSCs as a robust model and IFN-λ as a promising antiviral strategy.

The Role of a New Oral Bacterial RTX protein in Gum Disease From Early Detection to Future Treatments

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Abstract: Gum disease (periodontitis) is one of the most common chronic infections worldwide. It destroys the tissues that support our teeth and can lead to tooth loss. But its impact reaches far beyond the mouth. People with severe gum disease show a higher risk of heart attacks and stroke, highlighting the need for new diagnostic tools and treatment strategies. Research has focused on bacteria such as *Aggregatibacter actinomycetemcomitans* (A.a), and *Porphyromonas gingivalis*, however, recent studies showed that another bacterium, *Filifactor alocis* (F.a), may be just as important in driving the gum disease. Unlike A.a that appears to disappear as periodontal pockets deepen, *F. alocis* thrives in this environment, suggesting that it might have an important role in sustaining chronic inflammation.

Findings

Our earlier research has uncovered a previously unknown oral bacterial repeats-in-toxin (RTX) protein, FtxA, produced by about 50% of all known *F. alocis* strains. FtxA hence belongs to the same toxin family as the well-known leukotoxin of A.a., but it appears to behave differently. Instead of killing immune cells, our novel data using THP1 cells and RNA-Seq and cytokine antibody arrays, show that FtxA seemingly suppresses the host defense system by dampening inflammatory signaling, preventing programmed cell death, and prolonging the survival of immune cells in the diseased tissue. This would generate a long-lasting environment where selected bacteria can proliferate while the body fails to clear the infection. Through studies in this laboratory, assessing both adolescents in Ghana and adult patients in Australia, we found that individuals carrying *ftxA*-positive *F. alocis* were at significantly higher risk of gum disease progression, especially when combined with A.a. Our laboratory experiments confirmed that FtxA can act through extracellular vesicles released by the *F. alocis*, thereby delivering signals that reprogram human immune cells into a state of reduced activity.

Implications

By identifying the *ftxA* gene in saliva and/or dental plaque samples, dentists may one day be able to recognize individuals at highest risk before the disease causes irreversible damage, ideally as a rapid test. This could guide preventive care and reduce the need for costly surgical treatments. Beyond diagnostics, FtxA also represents a promising target for future precision therapies.

Investigating the role of 2-pyridones as antiviral compounds against enterovirus D68 infection

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Abstract: Enterovirus D68 (EV-D68) is a non-polio enterovirus associated primarily with respiratory illness, ranging from mild cold-like symptoms to severe respiratory distress, particularly in children and individuals with asthma. EV-D68 gained attention during outbreaks in recent years, notably in 2014, due to its potential link to acute flaccid myelitis (AFM), a rare but serious neurological condition. According to the CDC over 500 cases of AFM has been reported since and currently there is no effective treatment or vaccine against EV-D68. 2-pyridones are a versatile group of chemical compounds extensively used in medical drug development due to their broad range of applications. Compounds based on a thiazolo ring-fused 2-pyridone central scaffold with a peptidomimetic backbone, have been developed and extensively studied in the Almqvist lab in Department of Chemistry, Umeå. There are several reports from the group that highlight investigation into the multiplicity of biological functions associated with different functionalization patterns of this core scaffold. With this in mind, a small representative in-house library of (dihydro)thiazolo ring-fused bicyclic 2-pyridones were screened against EV-D68 using the human carcinoma epithelial cell line A549 as the host cell. This phenotypic screen led to the identification of compound SN95 as an anti-EV-D68 with no sign of cytotoxicity at high concentration. Preliminary investigations using flurescence and qRT-PCR assay suggests that SN95 target early steps of EV-D68 life cycle. Further investigation is under way to understand the mode of action of SN95 against EV-D68. Our study suggests that compounds with 2-pyridone scaffold is a promising class of chemicals with the potential to develop as antiviral drugs.

Poster #23

Molecular mechanisms behind filovirus entry: exploring cell attachment and entry potential of different filovirus-encoded glycoproteins

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Abstract: Filoviruses are amongst the most dangerous human pathogens. They pose a great health concern, with the emergence of a growing number of species that vary in tropism and pathogenicity. The glycoprotein (GP), the sole protein exposed on the virion surface, is crucial for entry. While the main filovirus entry receptor has been identified as the endosome-located NPC1 protein, the role of the many cell-surface attachment factors remains more elusive. In this work, we aim at establishing a difference in preferences of attachment factor usage by filoviruses in correlation to their pathogenicity.

To study these BSL4 viruses, we use a transcription and replication competent virus-like particles (trVLP) and produce particles decorated with the GP from the desired filovirus species. A total of five filovirus GPs are studied; three GPs encoded by pathogenic filoviruses: MARV (genus *Marburgvirus*), EBOV and SUDV (genus *Ebolavirus*), and GPs from two filoviruses that never caused lethal infection in humans, RESTV and LLOV (genus *Cueravirus)*.

Infection assays suggest differences in preferred attachment factors use among filoviruses, as we detected variations in the transduction efficiencies of trVLPs presenting different GPs across various cell types. To explain these observations, we analysed the relative importance of attachment factors, focusing on heparan sulfate (HS) and TIM1 receptor. While GPs of Ebolaviruses seemed to be more dependent on TIM1 receptor than MARV GP, LLOV GP decorated trVLPs were the most dependent on HS.

Understanding the differences between determinants of host cell membrane and virus interaction of pathogenic and apathogenic viruses may help to predict the pathogenicity of newly emerging filoviruses.

Tracing the Genetic Landscape of Puumala Virus in Sweden: Insights from the 1990s to Present

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Abstract: Orthohantaviruses are globally distributed RNA viruses primarily transmitted by rodents, and pose significant public health risks due to their high morbidity, mortality, airborne transmission, and lack of effective antiviral treatments. In northern Sweden, *Orthohantavirus puumalaense* (PUUV) is highly endemic and is the causative agent of a mild form of hemorrhagic fever with renal syndrome. The incidence follows cyclic fluctuations in bank vole populations, with the largest Swedish outbreak recorded in 2006–2007. The genetic variability of PUUV in outbreaks remains poorly explored, as sequencing of the viral genome primarily has been conducted on cell-cultured virus strains, not reflecting real world diversity. Consequently, there has been a lack of data derived from wild-type PUUV genomes.

Utilizing a targeted hybrid-capture protocol, we've sequenced complete wild-type PUUV genomes from both human and bank vole samples, some dating back to the 1990s. All belonged to the northern Scandinavian lineage and showed strong regional and temporal conservation. Human isolates were phylogenetically interspersed among bank vole sequences, indicating no clade-specific association with human infection. Segment-specific phylogenies revealed incongruent topologies, suggesting historical reassortment. We also observed substantial and uneven homoplasy, potentially indicating past recombination events. No variant was linked to specific outbreak periods.

Our findings suggest that PUUV evolution in Sweden is shaped by long-term geographic stability and segment reassortment, rather than the emergence and spread of new variants during outbreaks.

Organ barseq screens in *Plasmodium berghei*: a new tool for identifying critical mediators of malaria parasite sequestration

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Abstract. Malaria is a life-threatening infectious disease, and despite significant global efforts it remains a major public health threat, with 263 million cases and 597,000 deaths annually. *Plasmodium berghei*, a rodent-infecting species of *Plasmodium*, serves as an important model organism for studying malaria due to its shared biological and genetic features with human-infecting *Plasmodium* species. Importantly, it provides access to whole organism studies not possible with human malaria parasites, which lack nonprimate *in vivo* models.

Barcode sequencing (Barseq) knockout screens have previously identified those genes that are essential or dispensable to *P. berghei* blood-stage growth, as well as those genes that give rise to slow growth upon knockout. A key feature of the disease-causing blood-stages of *Plasmodium* parasites is organ sequestration, the cytoadhesion of infected red blood cells to the inside of blood vessels in organs. This drives severe disease and cause restriction of blood flow, hypoxia and inflammation in infected organs. Mutants unable to sequester efficiently are cleared by the spleen and typically have a slow-growth phenotype.

We here apply Barseq to slow-growth *P. berghei* knockout mutants to identify mutants with sequestration deficiencies. We present proof-of-principle data demonstrating that organ barseq identifies *Plasmodium* genes that are critical for organ sequestration and virulence. This methodology promises to identify novel disease-reducing drug and vaccine targets, enhancing our efforts to combat malaria and address the challenges to effective control posed by malaria drug resistance and immune evasion.

Poster #26

Deciphering the molecular mechanism of a novel cryptosporidiosis therapy

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Abstract. Cryptosporidiosis is a diarrheal disease affecting humans and mammals, caused by microscopic protozoan parasites of the genus Cryptosporidium, within the phylum Apicomplexa. Among the several species infecting humans, *Cryptosporidium hominis* is human-specific, whereas *Cryptosporidium parvum* is the most common zoonotic species. Recent global health reports have recognised Cryptosporidium species as one of the most lethal pathogens affecting malnourished infants and young children. The disease ranks among the top three diarrheal pathogens in terms of disability-adjusted life years (DALYs) lost. In Sweden, cryptosporidiosis has been a notifiable human disease since 2004. There is an urgent need in developing novel compounds against cryptosporidiosis to expand the unique FDA-approved therapeutic. We have developed experimental drug candidates targeting essential, non-redundant metabolic pathways in the parasite. After years of optimising compounds, the lead compounds have demonstrated an extraordinary preventive and curative efficacy against infection in both in vitro and in vivo infection models. However, their molecular targets remain unidentified. To address this, we employ an affinity-based protein labelling and structural homology analysis and molecular docking. These approaches aim to elucidate the identification of target and the molecular mechanisms of action of the lead compounds against cryptosporidiosis.

From Discovery to Validation: Advancing a Selective FabH Inhibitor for Treating *Chlamydia* Infections

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Abstract: *Chlamydia trachomatis* is the most common bacterial cause of sexually transmitted infections and the agent of blinding trachoma. Current clinical management relies on broad-spectrum antibiotics, which not always eradicate the infection fully, disrupt the commensal microbiota, and contribute to the development of antimicrobial resistance. To address these challenges, we recently conducted a comprehensive antichlamydial discovery campaign, which identified over 60 potent antichlamydial compounds that are chemically distinct from existing antibiotics. Extensive mechanistic and cellular analyses demonstrated that our most potent candidate, $c1_e$, selectively inhibits *C. trachomatis* fatty acid biosynthesis by targeting FabH, as confirmed by resistance mutation mapping, thermal proteome profiling, and *in vitro* enzyme inhibition assays. To enable translational progression, *in vitro* pharmacokinetic profiling revealed favorable drug-like properties, including high solubility, low plasma protein binding, and strong cellular permeability. To further optimize these characteristics, we developed solid lipid nanoparticle formulations of $c1_e$ with uniform particle size in the 200–250 nm range, 38% drug loading, and 99% encapsulation efficiency. The formulations display sustained release properties while retaining high antimicrobial efficacy, physicochemical stability, and low cytotoxicity in cell culture infection models. We are currently initiating *in vivo* pharmacokinetic analyses, followed by efficacy and selectivity studies in an established murine model of genital *C. trachomatis* infection. These experiments will provide the first *in vivo* proof-of-concept for selective FabH inhibition as a therapeutic strategy against *Chlamydia*. Altogether, this work demonstrates the feasibility of developing pathogen-selective therapies as a sustainable alternative to broad-spectrum antibiotics in the fight against antimicrobial resistance.

Poster #28

Protein O-glycosylation in the Bacteroidota phylum

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Abstract: The human gut microbiome is a complex and variable community of microorganisms, with Bacteroidota being one of the most abundant phyla. These bacteria are known to heavily decorate their surface proteins with glycans, primarily on serine and threonine, though the composition and function of these glycans remain largely unknown for most species. It is hypothesized that this modification helps the bacteria evade the human immune system or aids in self-recognition. To map the diversity of O-glycan structures in Bacteroidota species, we performed bottom-up global proteomics across a range of species of this phylum using nanoflow liquid chromatography coupled to a Thermo Fisher Orbitrap Exploris 480 mass spectrometer using a stepped HCD method. We identified the biosynthetic pathways of these complex glycans through sequence- and structure-based protein alignment tools. For each species analyzed we identified a small fraction of peptides (<0.5%) with a mass shift >1000 Da. These peptides originated from proteins containing a predicted signal peptide, suggesting that they leave the cytoplasm and agreeing with the current understanding that this glycosylation happens in the periplasm. The exact mass of this modification varied between species and using open search and the diagnostic ion mapping feature of MSFragger, combined with a graph-based approach, we reconstructed the general species-specific glycan structures. We are currently exploring NMR to characterize the detailed structure including stereoisomers and monosaccharide linkages. Most species share a common inner part but differ in the monosaccharide composition and branching of the outer part of their glycan.

Poster #29

Non-canonical roles of autophagy in enterovirus infection

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Abstract: Enteroviruses are a diverse group of RNA viruses with over 100 human-infecting members, causing diseases ranging from myocarditis and pancreatitis (e.g., Coxsackievirus B3, CVB3) to poliomyelitis (poliovirus) and the common cold (rhinoviruses). These viruses extensively remodel intracellular membranes to form replication organelles that support viral genome replication and assembly. Intriguingly, these membranes are decorated with the autophagy protein LC3.

Autophagy, or "self-eating," is a degradative cellular process that maintains homeostasis by recycling cellular components. A key step in autophagy is LC3 lipidation—the covalent attachment of LC3 to cellular membranes. While LC3 lipidation is critical for canonical autophagy, it also functions in damage-responsive membrane remodeling, termed as non-canonical autophagy (NCA). This damage responsive NCA is mediated by two distinct LC3 lipidation complexes. However, it remains unknown whether and how enteroviruses exploit NCA to support their replication.

In this study, we employed a combination of advanced cell biology and molecular virology techniques to elucidate the role of NCA in enterovirus replication. We first assessed viral infection across a panel of NCA-related knockout cell lines to determine the contribution of specific NCA factors. To dissect the specific role of these proteins in replication independent of viral entry, we employed a CVB3-GFP replicon system. Furthermore, cryo-electron tomography will be used to visualize how NCA factors contribute to cytoplasmic remodeling during infection. This work aims to uncover detailed mechanistic insights into how enteroviruses hijack specific NCA functions, possibly unveiling new targets for antiviral treatments.

Poster #30

Mapping Determinants of Pathogen Vacuole Stability and Host Defense During Infection with *Chlamydia trachomatis*

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Abstract: The success of intracellular pathogens often depends on their ability to reside in a pathogen-containing vacuole (PCV), which protects them from immune detection while providing a permissive niche for replication. Despite the central importance of PCVs in infection biology, their native molecular composition and the dynamic host–pathogen interactions occurring at their interface with the host cell remain poorly defined. Here, we strive to investigate the PCV (inclusion) of *Chlamydia trachomatis* (*Ct*), a clinically significant bacterium causing ocular and sexually transmitted infections, to identify molecular features that maintain inclusion stability. Using proximity-based protein labelling combined with mass spectrometry (MS), we aim to compare the proteomic landscape of stable versus unstable inclusions generated by wild-type (WT) *Ct* and a mutant deficient for the effector CpoS, previously shown to have a role in inclusion stability. To achieve this, we engineered WT and *CpoS*-deficient *Ct* strains to express TurboID biotin ligase as a fusion protein displayed on the inclusion membrane, enabling selective biotinylation of proteins in proximity to the inclusion surface. Expression and proper membrane localization of the fusion construct were confirmed by western blotting and fluorescence microscopy. Infected cells were incubated with biotin, and labelled proteins were enriched using streptavidin-conjugated magnetic beads and will now be subjected to MS analysis. We anticipate that the comparative proteomic profiles will reveal distinct molecular signatures, uncovering previously unrecognized host pathways that contribute to inclusion integrity and facilitate immune evasion by *Ct*. This work has the potential to transform our understanding of PCV biology and identify novel targets for therapeutic intervention in chlamydial disease.

Poster #31

Direct observation of filovirus interaction with the endothelial glycocalyx

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Abstract: The endothelial glycocalyx, a matrix of membrane-bound proteoglycans and glycoproteins extending hundreds of nanometres into the vascular lumen, constitutes a major physical barrier to pathogen invasion. How viruses penetrate this barrier remains unclear. Filoviruses (Ebola, Marburg) cause severe haemorrhagic fevers and bear a highly glycosylated mucin-like domain (MLD) on their glycoprotein, which is known to influence interactions with cell-surface glycans in several viruses.

In our study, in collaboration with the Welsher Lab (Duke University, USA), we employ three-dimensional tracking and imaging (3D-TrIm) microscopy, a technique combining active single-particle tracking with volumetric cell imaging, to directly observe lentiviral pseudotypes bearing filovirus glycoproteins approaching and diffusing in the glycocalyx. With nanometric resolution and millisecond frame rates, we show that the thick glycocalyx of human endothelial cells (CiGEnC) prolongs virion contact with the cell surface, potentially facilitating receptor engagement, whereas removal of the MLD increases particle mobility. This interplay between the glycocalyx and MLD is reflected in infection assays, in which deleting the MLD significantly enhances infection in CiGEnC compared to cell lines lacking a thick glycocalyx. Furthermore, using a single-particle kinetic assay, we show that filoviruses directly interact with heparan sulfate – a major component of the glycocalyx – through the MLD, and that enzymatic removal of heparan sulfate causes a ~60% reduction in CiGEnC infection.

Together, these results provide the first direct measurements of viral 3D dynamics within the endothelial glycocalyx and reveal how the MLD and glycocalyx influence the earliest steps of filovirus entry.

Poster #32

Durable immune memory to human hantavirus Infection

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Abstract: Zoonotic hantavirus infections may cause mild to severe disease in humans. The virus is cleared from circulation within 1-2 weeks after disease onset. Currently, there are no records of disease-causing re-infections from hantavirus. This suggests that the initial infection generates potent immune memory against the virus. Here, we set out to determine the longevity and quality of humoral immunity after Puumala hantavirus (PUUV) infection.
By assessing longitudinal samples from 40 PUUV infected individuals, we could determine that serum IgG-binding and avidity to the Spike protein (GnGc) and the Nucleocapsid protein (N) increased over the course of more than 11 years. In contrast, levels of GnGc- and N-IgA and IgM initially increased but declined over the first 3-6 months after disease onset. By assessing longitudinal samples from 210 individuals in the Northern Sweden Health and Disease biobank, we could determine that the seroprevalence was 12% over the course of 20 years, and that once GnGc-IgG was detected, subsequent samples remained positive over a timeperiod of up to 20 years. Even though the avidity circulating GnGc-specfic IgG and IgA antibodies increased over the course of the study, this was not associated with increased neutralization potential. To investigate the cause for this, we leveraged advanced proteomics methodology, Ig-Seq, to understand the dynamics of PUUV specific antibody clonotypes and epitope-specificity over the years. Taken together, our data provides a detailed kinetics of durable antibody response to Puumala hantavirus infection and point towards B cell dependent long-term antibody production over the course of decades after primary infection.

The effect of temperature on the vector competence of *Anopheles stephensi* for o'nyong nyong virus

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Abstract: O' nyong nyong virus (ONNV) is an arthropod-borne virus transmitted by *Anopheles (An.) gambiae* and *An. funestus* in Africa. The first epidemic lasted three years and affected more than 2 million people in sub-Saharan Africa, since then there have been sporadic outbreaks. Since 2012 *An. stephensi* species are confirmed to have invaded the horn of Africa and several sub-Saharan ONNV endemic, the introduction of *An. stephensi* can result in large spread ONNV outbreaks. Furthermore, to *An. stephensi* preference for urban areas over rural and its tolerance for a wider temperature range than the primary vectors, it is possible that it will introduce ONNV to new areas affecting more people. As most studies on *Anopheles* species vector competence for ONNV are done at the optimal temperature of 28 ± 1°C and for only one strain, it is unknown how temperature and strain effects vector competence. To investigate the influence of temperature influences and strain on the vector competence of *An. stephensi* for ONNV, mosquitoes were infected at 3 different temperatures with ONNV and after dissection, the amount of viral RNA in the mosquito was determined using RTq-PCR. The infection – and transmission rate was assessed at 7 days post infection (dpi) and 14 dpi for three different strains. At 28°C for all strains the infection and transmission rate was 100% at both time points. The infection and transmission rate reduced significantly at 23.5°C for DaKAr and SG650, IBH10964 did not significantly decrease and at 32°C there was infection but barely any transmission.

Same Receptor, Different Role: How Heparan Sulfate Chemistry Shapes Adenovirus Binding Behaviour for Cell Entry

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Abstract: Heparan sulfate (HS) is a common cell-surface glycan exploited by many viruses, including adenovirus to promote entry. Interestingly, human adenoviruses make different uses of HS, depending on species/type: enteric adenovirus F40 employs HS as an entry receptor, while ocular adenovirus D37 interacts with HS as a decoy receptor. Yet, the mechanism by which HS can play a dual role in shaping adenoviruses binding behavior for entry remains unclear. To address this, we applied atomic force microscopy-based single-molecule force spectroscopy together with cell-surface mimic platform to quantify three key parameters of adenovirus–HS interactions: (i) binding affinity, (ii) bond strength (force required to break a single bond), and (iii) bond lifetime under tension -catch bond. Our results reveal striking differences between these clinically relevant strains. F40–HS interactions exhibit 58-fold higher binding affinity than D37–HS under physiological conditions, and affinity increases significantly for F40–HS after exposure to synthetic gastric juice, mimicking the gastrointestinal environment of the gut. Moreover, while bond strength is similar for both strains, it rises 1.2 times for F40–HS in acidic conditions. Finally, we also reveal catch-bonds for F40–HS interactions in both physiological and acidic conditions, but for D37–HS, it is present only after exposure to synthetic gastric juice. These findings demonstrate that HS-mediated interactions are dynamic and environment-sensitive and contribute to explaining the dual role of HS. By uncovering how HS chemistry modulates adenovirus binding behavior, this work provides fresh insights into adenovirus-host interactions and suggests new strategies for antiviral design.

Immune Landscape and Microbiome-Associated Signatures in Pancreatic Ductal Adenocarcinoma: Insights From Murine and Organoid Models

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Abstract: Pancreatic ductal adenocarcinoma (PDAC) remains one of the most lethal malignancies, driven by profound immune suppression and stromal heterogeneity. Increasing evidence suggests that both age at disease onset and microbial exposures influence tumor immunogenicity and treatment response. This study investigates (i) age-dependent immune and spatial biomarker signatures within the PDAC microenvironment and (ii) the impact of genotoxin-producing *Salmonella* on pancreatic tumor development using murine and organoid models. Tumors were generated in young (8-week) and old (30-week) mice through orthotopic implantation of pancreatic cancer cells and the inducible KPC model of spontaneous PDAC. Multiplex *in situ* immunofluorescence was optimized on FFPE and fresh-frozen tissues using LabSat, applying an immune panel including MHC-II, MPO, B220, CD3e, Ly6G, CD45, and F4/80. For microbiome-related analyses, normal (mN1), preneoplastic (mp4), and cancerous (mt4) pancreatic organoids were infected with genotoxin-producing ($CdtB^+$) *Salmonella* or an isogenic ΔcdtB mutant and evaluated for morphological changes, DNA damage (γH2AX), and proliferation (Ki67) in 2D and 3D cultures. Initial multiplex immunofluorescence has been successfully established, with preliminary results indicating age-related differences in immune cell infiltration and spatial organization. In organoids, $CdtB^+$ infection induced measurable DNA damage and altered proliferation. Normal organoids exhibited increased γH2AX in both ΔcdtB and $CdtB^+$ conditions, accompanied by reduced Ki67 in ΔcdtB and elevated Ki67 in $CdtB^+$ infections, suggesting the emergence of damage-tolerant proliferative subpopulations. These early findings indicate that host age and microbial genotoxins may shape PDAC immune landscapes, influencing tumor evolution and therapeutic vulnerability.

Antifungal Activity of Isatin on *Saprolegnia* spp.: *In Vitro* and *In Vivo* Evaluation in Salmonid Eggs

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Abstract: *Saprolegnia spp*. are a major pathogen in the aquaculture industry, causing economic losses especially during salmonid egg incubation. Currently treatments with formalin and hydrogen peroxide are neither highly effective nor safe. In this regard, isatin (1H-indole-2,3-dione), a naturally compound that prevents the infection fungal infection in shrimp eggs, shows potential as a prophylactic agent in aquatic systems. This study evaluated the inhibitory effect of isatin on Saprolegnia and its application in salmonid egg infection models in vitro. Three Saprolegnia species were expose to different isatin concentrations. IC_{50} was determined using Celltiter-Glo and disk diffusion assays. Gene expression analyses identify affected molecular pathways and infected eggs were incubated in isatin solution for *in vivo* testing. Isatin showed a hormesis effect in lower concentrations, with IC_{50} values established between 25-60 µg/ml, while clear inhibitory zones were observed in concentration below 100 µg/ml in disk diffusion assay. Lower concentrations of isatin induces expression of cell wall synthesis enzymes and reduces oxidative stress makers. At 25ug/ml, isatin induces expression of apoptosis and metabolic makers without affecting oxidative stress or cell wall enzymes. Nevertheless, concentration above 50 µg/ml suppressed global RNA expression. Salmonid eggs incubated in 100 µg/ml of isatin solution showed lower viability of saprolegnia infected eggs compared to control group. These findings demonstrate the isatin antifungal effect and its potential as a prophylactic treatment in salmonid egg incubation offering a safer alternative to harmful chemicals while improving fish welfare, reproduction success, and alignment with ecological and food safety standards.

National Bioinformatics Infrastructure Sweden (NBIS)

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Abstract: NBIS, National Bioinformatics Infrastructure Sweden, is a distributed national research infrastructure. We are the SciLifeLab bioinformatics platform and the Swedish node in Elixir, a European intergovernmental organisation bringing together life science resources from across Europe. With over a hundred staff members, we work with bioinformatics support, infrastructure and training.

NBIS has staff at six sites: Umeå, Uppsala, Stockholm, Linköping, Göteborg, and Lund. We provide expertise in most areas of bioinformatics, including omics analysis, genome assembly/annotation, image analysis and biostatistics. We also offer support in systems development, such as interactive websites and data processing pipelines.

NBIS is mainly funded by the Swedish Research Council, SciLifeLab, the Knut and Alice Wallenberg Foundation, and Swedish universities.

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- The Swedish Bioinformatics Advisory program - A mentorship program for PhD students interested in guidance from a bioinformatics expert.

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µNordic Single Cell Hub

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Abstract: µNiSCH (µNordic Single Cell Hub) is a cutting-edge infrastructure dedicated to advancing single cell biology at Umeå University. Our facility focuses on consolidating and expanding research in this dynamic field. We are also at the forefront of developing innovative molecular tools for single cell transcriptomics and genomics, with a special emphasis on microbial studies. The facility is located within the Department of Molecular Biology.

Both the advanced equipment and specialized expertise are open to all research groups interested in single cell biology applications on eukaryotic and prokaryotic cells, including:

- Single cell transcriptomics
- Single cell genomics
- Cell-based phenotypic screening assays
- Multi-step biological workflows in single cells
- Spatial distribution of gene expression
- Characterization of tissue microenvironment

Currently, we can offer expertise and access to the following equipment:

***Onyx Microfluidics Platform*,** a microfluidic platform designed to generate and manipulate water-in-oil droplets or semi-permeable capsules for high-throughput single-cell and single-molecule analysis.

Chromium Single Cell Gene Expression system, a cutting-edge platform designed to analyze gene expression at the single-cell level.

BD Rhapsody HT Xpress System, a high-throughput microwell-based technology for advanced single-cell analysis.

LabSat Multiplex immunostaining system, an advanced automated platform designed for rapid and precise immunohistochemistry and immunofluorescence staining, particularly in research and clinical settings.

The Pannoramic Midi II, an automatic digital slide scanner for high-throughput microscopy imaging, offering precision and efficiency in pathology and research applications.

The OT-2 multi-well pipetting robot, an automated liquid-handling system designed to streamline and enhance laboratory workflows.

Mantis Rapid Dispenser System, liquid handling platform designed to streamline and enhance precision in laboratory workflows.

For requests or questions, please contact:
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Large-scale insights into the biosynthetic potential of the *Bacillus cereus* group

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Abstract: Bacterial secondary metabolites (SMs) are a critical source of natural product-derived drugs. However, SM discovery efforts have focused overwhelmingly on *Actinomycetes*, potentially overlooking other key producers. Here, we explore the biosynthetic potential of the *Bacillus cereus* group, an underexplored complex of SM producers. Using a combined rule- and machine learning-based approach, we mine an unprecedented number of *B. cereus* group genomes (n = 9,744) for SM-producing biosynthetic gene clusters (BGCs; n = 200,196). Notably, 158,678 *B. cereus* group BGCs (78.2%) did not cluster with previously described BGCs, suggesting new chemical scaffolds to be explored. *B. pseudomycoides* was particularly prolific in terms of its SM production potential (30.8 BGC families/genome, Kruskal-Wallis $p < 0.0001$), and we identify a previously uncharacterized, *B. pseudomycoides*-unique peptide. Overall, our study represents the largest survey of *B. cereus* group biosynthetic potential to date and posits the complex as an under-queried SM resource.

Identifying DNA Methylation Patterns in Post COVID-19 Condition: Insights from a One-Year Prospective Cohort Study

Christoffer Granvik[a*], **Jelena Smiljanic**[b,c*], **Alicia Lind**[d], **David Martinez-Enguita**[e], **Shumaila Sayyab**[f], **Clas Ahlm**[a], **Frida Nikesjö**[g,h], **Mattias N. E. Forsell**[a], **Sara Cajander**[i], **Mika Gustafsson**[e], **Martin Rosvall**[b], **Johan Normark**[a*], **Maria Lerm**[f*]

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Abstract: Post COVID-19 condition (PCC) presents with a wide range of persistent symptoms, yet its underlying biological mechanisms remain poorly understood. This project aimed to identify epigenetic signatures associated with long-term symptom persistence by tracking longitudinal DNA methylation changes in individuals recovering from SARS-CoV-2 infection. We analyzed genome-wide methylation profiles over one year in 22 non-hospitalized individuals with sustained symptoms and reduced quality of life (PCC+) and 22 matched convalescent controls without persistent symptoms (PCC−). Differential methylation analysis was performed at multiple timepoints to identify group-specific changes and their associations with clinical manifestations. Our results reveal distinct methylation differences between PCC+ and PCC− individuals that gradually diminish over time, suggesting dynamic epigenetic remodeling during recovery. Notably, methylation changes within *TXNRD1* were strongly associated with cognitive complaints and fatigue, implicating redox imbalance as a contributor to PCC pathology. Pathway enrichment analysis highlighted alterations in PI3K-Akt and AMPK signaling pathways, offering a potential mechanistic explanation for emerging evidence supporting metformin's efficacy in reducing PCC incidence. Although epigenetic age acceleration did not differ between groups, longitudinal shifts were detected in methylation patterns linked to RAS and RAP1 signaling, pointing to broader regulatory disruptions during prolonged recovery. These findings provide valuable insight into the molecular mechanisms underlying PCC, identify oxidative stress–related pathways as promising therapeutic targets, and support the use of epigenetic profiling to track and understand post-infection sequelae. This work underscores the importance of continued investigation into post-viral biology to guide effective treatment and prevention strategies.

Long-term evolution of PUUV-specific humoral immune response following primary infection

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Abstract: Puumala hantavirus (PUUV) causes hemorrhagic fever with renal syndrome, and the virus is cleared within 1-4 weeks after disease onset. To investigate long-term humoral immunity, we analysed a clinically verified cohort containing samples collected over a decade post-infection. Multiplex immunoassay revealed that all individuals were IgM, IgA and IgG positive for spike (GnGc)- and nucleocapsid (N) proteins within a week after disease onset. However, while the GnGc- and N-IgA and IgM declined over the first three months, the levels of IgG to both viral proteins increased over the course of three years. Screening 210 individuals from the Northern Sweden Health and Disease Cohort revealed that IgG persists for at least 20 years. Avidity of GnGc-IgG increased over three years, with immunofocusing toward the Gn subunit. Subtype analysis indicated a dominant GnGc-IgG1 response and isotype switching to IgG4 at 3-6 months. N-IgG1 behaved similarly but N-IgG4 was absent. These findings suggest that PUUV antibody responses are driven by GnGc proteins and mediated by IgG+ B cells and plasma cells. Preliminary data shows increasing FcγIIIa binding of GnGc-IgG during the acute phase, indicating enhanced capacity for inducing ADCC, while FcγIIIa/IIa binding remained stable after 2-8 weeks. This suggests that effector function capacity did not change significantly at later time points. We are currently investigating the neutralization capacity to understand the biological significance of the observed immunofocusing of IgG. Collectively, PUUV-IgG shows long-term maintenance and evolution, implying prolonged immune exposure to GnGc and N proteins for up to 10-20 years after viral clearance.

Synthesis of ring-fused 2-pyridone derivatives to target FadA protein of *Fusobacterium nucleatum* involved in Colorectal Cancer

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Objectives: Colorectal cancer (CRC) remains a leading cause of death, and growing evidence shows that the gut bacterium *Fusobacterium nucleatum* (Fn) can worsen disease by helping tumors grow, spread and resist therapy. A key factor in this process is amyloid-like protein FadA. By facilitating the adhesion of Fn to host cells, FadA promotes CRC progression, suggesting this protein as a promising therapeutic target. Previous research has shown that ring-fused 2-pyridone compounds can target amyloid-like structures, including FadA. These compounds are characterized by a peptidomimetic backbone, which allows them to interact with amyloid fibrils and β-sheet structures, common features in many amyloid proteins. Building on these features, tricyclic pyridine-fused thiazolo-2-pyridone, and bicyclic thiazine-2-pyridone scaffolds were designed as ring-expanded analogs of the parent 2-pyridones.

Methods: The general sequence of multi-step synthesis has been standardized to prepare designed analogs to get expanded structure-activity-relationship with improved potency, selectivity, and non-cytotoxicity.

Results: Initial studies identified one compound from both bicyclic (MN241) and tricyclic (PS598) series as hit compounds that significantly inhibited Fn attachment to HCT116 cells in a dose-dependent manner. The further designed analogs will be synthesized and tested against the tested cancer cell lines. We further aim to investigate the molecular mechanisms by which FadA contributes to colorectal cancer progression and how its inhibition modulates tumor development and response to immunotherapy *in vivo*.

Conclusion: The available findings and the proposed work suggest that the tricyclic pyridine-fused thiazolo-2-pyridone, and bicyclic thiazine-2-pyridone scaffolds hold significant potential developing FadA-targeted anti-virulence compounds and may provide a useful new approach for treating Fn-associated CRC.

Figure 1: *General structures of the designed tricyclic and bicyclic 2-pyridone derivatives*

Prokaryotic carbon cycling and metabolic adaptations in Central Arctic Ocean

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Abstract: The Central Arctic Ocean (CAO) is one of the most extreme marine environments, shaped by severe cold, persistent ice cover and seasonal darkness. With the Arctic Ocean warming four times faster than the global average, the ecological functioning of its microbial food web is expected to shift, yet the mechanisms underlying prokaryotic carbon cycling and its adaptations in this region remain poorly understood. marine food web. Prokaryotes (Bacterial and Archaea) play a central role in regulating the balance between carbon released as CO_2 and carbon sequestered in the ocean. We combined prokaryotic rate measurements including respiration quantified using high precision optode sensors and growth assessed through ^{3}H-thymidine incorporation. We also used high resolution scanning electron microscopy (SEM) and metatranscriptomics to examine morphological features and the functional metabolism. The prokaryotic growth efficiency (PGE) was 2%, the lowest reported to date, indicating that significantly low fraction of carbon is assimilated as biomass, resulting in fourfold higher carbon demand than previously assumed. The low PGE suggested elevated respiration supporting cellular maintenance, consistent with transcript profiles associated with osmoregulation, defense and DNA repair. SEM revealed distinct morphological features including presence of extracellular polymeric substances, pili- and tubule-like structures, consistent with transcript abundance patterns. Together, these results show that CAO prokaryotes thrive at sub-zero temperatures through high carbon demand, structural modifications and increased maintenance costs. These findings refine our understanding of carbon cycling in a rapidly warming Arctic and highlight the potential role of prokaryotes in shaping the Arctic ecosystem.

Uncovering a role for hyaluronan in Puumala virus-induced pulmonary disease

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Abstract: Hantaviruses are globally distributed zoonotic viruses that cause severe human disease with high case-fatality rates. In Europe, Puumala orthohantavirus (PUUV) is the most prevalent hantavirus with over 2,000 reported cases annually. Infections cause a mild form of haemorrhagic fever with renal syndrome, characterized by increased vascular permeability, acute kidney injury and thrombocytopenia. While these symptoms are well characterized, reports highlight pulmonary symptoms in PUUV patients, revealing significant gaps in our understanding of the disease. A potential contributor to these symptoms is hyaluronan, a highly hydrophilic glycosaminoglycans implicated as a driver of the pulmonary manifestations in other respiratory infections. Here, we investigate the role of hyaluronan in PUUV pathogenesis, examining its accumulation in the lungs of deceased patients and analysing changes in hyaluronan concentration in bronchoalveolar lavage fluid. Additionally, we characterize the hyaluronan accumulation and regulation of lung fibroblast and epithelial cells following PUUV infection.

As an initial observation, immunohistochemical staining of lung tissue from deceased PUUV patients revealed extensive accumulation of hyaluronan in the alveolar space, with regions showing complete loss of alveolar morphology. Supporting this, patients infected with PUUV exhibited significantly elevated levels of hyaluronan in bronchoalveolar lavage fluid compared to healthy controls, with hyaluronan levels positively correlating with disease severity. *In vitro*, lung fibroblasts and epithelial cells accumulated hyaluronan following PUUV infection, with peak levels observed at 72- and 96-hours post-infection, respectively. This accumulation was a result of the increased expression of hyaluronan synthase 2 and 3, with fibroblasts and epithelial cells exhibiting distinct, cell-specific regulation of hyaluronan synthases and hyaluronidases.

In summary, we demonstrate extensive accumulation of hyaluronan in the lungs of deceased PUUV patients and identify a correlation between hyaluronan and disease severity. Furthermore, we show that lung fibroblasts and epithelial cells accumulate hyaluronan following PUUV infection as result of increased expression of hyaluronan synthase. Together, these results provide the first insight into the role of hyaluronan as a potential driver of PUUV lung pathogenesis, highlighting an understudied aspect and possible novel target for symptomatic treatment.

Booths

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https://umeapostdocs.com/

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IDS creates opportunities for collaboration between Umeå University researchers, business and the public sector through jointly funded research projects. Together with an external part, the doctoral student and supervising researcher work for four years with a common problem that needs to be explored. We have trained almost 100 researchers together with more than 70 external partners.

https://www.umu.se/en/research/research-with-us/doctoral-studies/research-schools/industrial-doctoral-school-for-research-and-innovation/

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Research Infrastructure Presentations

NBIS. National Bioinformatics Infrastructure Sweden (poster #37)
Jeanette Tångrot
https://nbis.se

µNordic Single Cell Hub (poster #38)
Tugrul Doruk
https://www.umu.se/en/research/infrastructure/micro-nordic-single-cell-hub/

BICU. Biochemical Imaging Centre Umeå (pitch and poster #39)
Irene Martinez Carrasco
BICU is a national infrastructure that provides state-of-the-art microscopy technology, including advanced light microscopy and atomic force microscopy. Together with UCEM, BICU forms a node in the National Microscopy Infrastructure (NMI), Sweden for Correlative imaging.
https://www.umu.se/en/research/infrastructure/biochemical-imaging-centre-umea-bicu/

BMCU. BioMolecular Characterization Umeå (pitch and poster #40)
Johan Olofsson Edlund
BMCU is an interdisciplinary facility that provides state-of-the-art technology to characterize biomolecules. The facility allows measurements of affinity using different technologies, together with molecular weight analysis, folding and overall structure.
https://www.umu.se/en/research/infrastructure/biomolecular-characterization-umea-/

UCCB. Umeå Centre for Comparative Biology (pitch and poster #41)
Ellen Bushell
Experimental animal research at Umeå University is organized within UCCB. The main task is to organize and administrate with a focus on use of laboratory animals, which in plain terms means housing, husbandry and help breeding of the experimental animals demanded by researchers.
https://www.umu.se/en/umea-centre-for-comparative-biology/

UCEM. Umeå Centre for Electron Microscopy (pitch and poster #42)
Sara Henriksson
UCEM is a university and national resource for research and higher education in electron microscopy techniques.
https://www.umu.se/en/research/infrastructure/umea-centre-for-electron-microscopy-ucem/

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