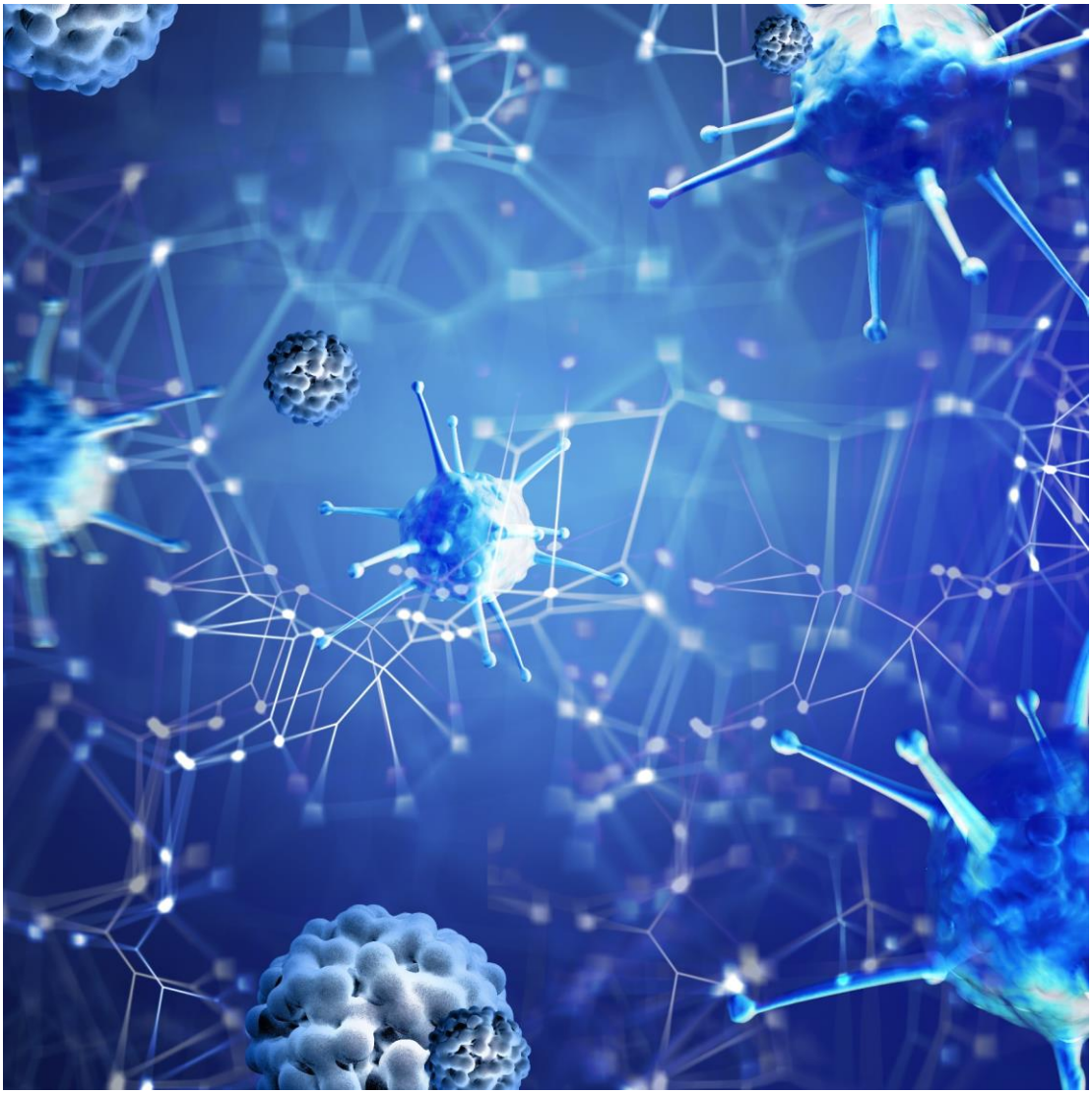

UCMR

Umeå Centre for Microbial Research

UCMR DAY 2023

19 January 2023, Aula Nordica

Programme - Abstracts - Posters - Participants

UMEÅ UNIVERSITY

https://www.umu.se/en/ucmr/

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Welcome to the 14th UCMR Day 2023!

This year marks the first onsite meeting after the COVID-19 pandemic. We all have missed the personal interaction and the discussions during the coffee breaks and the poster sessions.

To embrace this return to “in real life science”, the UCMR Day organizing committee has opted for a different meeting style, with two outstanding keynote speakers (**Tobias Dörr**, Cornell University, and **Maria Grazia Masucci**, Karolinska Institutet) and where all the poster presenters have the possibility to showcase their project in the form of elevator talks. We are confident that this model can fulfill our will to see a lot of good science, learn as much as possible and develop novel ideas.

On behalf of the scientific organizing committee,

Barbara Sixt, Department of Molecular Biology

Ignacio Mir-Sanchis, Department of Medical Biochemistry and Biophysics

Felipe Cava, Department of Molecular Biology

Teresa Frisan, Deputy Director of UCMR, Department of Molecular Biology

Yaowen Wu, Director of UCMR, Department of Chemistry

Programme

8.30 – 9.00 **REGISTRATION and MOUNTING OF POSTERS/VISUAL ABSTRACTS**

9.00 - 09.10 **WELCOME**

Mikael Elofsson, Dean at the Faculty of Science and Technology

9.10 – 9.30 **UCMR UPDATE**

Yaowen Wu, Director of UCMR

9.30 – 10.15 **ELEVATOR TALKS I** *(3 minutes talks)*

Chair: Barbara Sixt

#P1 **Insights into the genetic contexts of sulfonamide resistance among early clinical isolates of *Acinetobacter baumannii***
Anju Bala, Department of Molecular Biology, Umeå Centre for Microbial Research (UCMR), Umeå University

#P2 **Metabolic and Morphotypic Trade-offs within the Eco-Evolutionary Dynamics of *Escherichia coli***
Nikola Zlatkov, Department of Molecular Biology, Umeå University, Sweden

#P3 **Siderophore transporter-based vaccines promote protection in sole fish against *Aeromonas salmonicida* infection**
Diego Rey-Varela, Departamento de Microbioloxía e Parasitoloxía, Instituto de Acuicultura, Universidade de Santiago de Compostela, Spain

#P5 **Characterization of Fluorescent Filovirus Pseudotypes**
Kerstin Seier, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine, Umeå University, Sweden

#P6 **Sulfation patterns of heparan sulfate modulate the interactions between human papillomavirus and cell surface glycosaminoglycans at the single particle level**
Fouzia Bano, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine, Umeå University, Sweden

#P11 ***Streptococcus pyogenes* FASII regulator FabT avoids unnecessary energy expenditure and is required for human tissue infection.**
Clara Lambert, Université Paris Cité, Institut Cochin, INSERM, U1016, CNRS, UMR8104, Paris, France

#P12 **Towards structural characterization of diacylglyceryl transferase (Lgt) from *P. gingivalis***
Sampath Kumar Yalamanchili, Department of Chemistry, Umeå University, Sweden

#P13 ***In vivo*** **transcriptomics of** ***Salmonella*** **Typhimurium persistence**
Ummehan Avican, Department of Microbiology, Tumor and Cell Biology, Karolinska Institutet, Stockholm, Sweden, and Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden

#P16 **Recruitment of apolipoprotein E facilitates Herpes simplex virus 1 release from infected cells**
Lifeng Liu, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine, Umeå University, Sweden

#P19 **Temperature and nutrient control of aquatic prokaryotic maintenance respiration, growth efficiency, and community composition**
Ashish Verma, Department of Ecology and Environmental Science and Umeå Marine Sciences Center, Umeå University, Sweden

#P22 **Tissue-specific effects of genotoxigenic** ***Salmonella*** **during** ***in vivo*** **infection**
María López Chiloeches, Department of Molecular Biology and Umeå Centre for Molecular Research (UCMR), Umeå University, Umeå, Sweden

#P24 **Study of the SaPI2 De-repression Complex**
Gianluca Debiasi-Anders, Department of Medical Biochemistry and Biophysics and Wallenberg Centre for Molecular Medicine, Umeå University, Sweden

10.15-10.45 **COFFEE and SANDWICH**

Served in the Lounge

10.45-11.15 **ELEVATOR TALKS II** *(3 minutes talks)*

Chair: Felipe Cava

#P25 **Enterovirus 2C protein and cellular membrane: what could go wrong??**
Kasturika Shankar, Department of Medical Biochemistry and Biophysics, Wallenberg Centre for Molecular Medicine, Molecular Infection Medicine Sweden, Umeå University, Sweden

#P26 **The effect of the lipid membrane environment on the binding kinetics of norovirus to glycolipids**
Konrad Thorsteinsson, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine, Umeå University, Sweden

#P27 **Evaluating the role of NUPs in TBEV infection**
Marie Berit Akpiroro Peters, Department of Clinical Microbiology and Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå University, Sweden

#P28 **Transient Glycolytic Complexation of Arsenate Enhances Resistance in the Enteropathogen *Vibrio cholerae***
Emilio Bueno, Laboratory for Molecular Infection Medicine Sweden, Department of Molecular Biology, Umeå Centre for Microbial Research, Umeå University, Sweden

#P29 **Characterization of a primase-polymerase encoded by Phage-inducible chromosomal islands**
Cuncun Qiao, Department of Medical Biochemistry and Biophysics and Wallenberg Centre for Molecular Medicine, Umeå University Sweden

#P30 **Undecaprenyl phosphate translocases confer conditional microbial fitness**
Emilio Bueno, Laboratory for Molecular Infection Medicine Sweden (MIMS), Department of Molecular Biology, Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden.

#P31 **Investigating Key Microbiota Molecules to Rescue Western-style Diet-induced Mucus Defects in Mice**
Sandra Holmberg, Department of Molecular Biology and Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå University Sweden

#P32 **Chemo-optogenetic systems for reversible control of protein function in live cells**
Jun Zhang, Department of Chemistry and Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden.

11.15-12:00 **KEY NOTE LECTURE I**

Chair: Felipe Cava

Stress signaling promotes antibiotic resistance and tolerance in Gram-negative pathogens

Dr. Tobias Dörr, Cornell University, USA

12.00-13.00 **LUNCH**

Buffet, Universum restaurant

Programme

13.00-14.00 **ELEVATOR TALKS III** *(3 minutes talks)*

Chair: Ignacio Mir-Sanchis

#P33 **Combination of gallium citrate with linezolid or levofloxacin potentiate growth inhibition of drug resistant *Mycobacterium tuberculosis* and results in differential metabolome changes**
Oleksandr Ilchenko, Umeå University, Sweden and Odessa National University, Ukraine

#P34 **The importance of dietary fibre for gut health**
Rachel Feeney, The laboratory for Molecular Infection Medicine Sweden (MIMS) and Department of Molecular Biology, Umeå University, Sweden

#P35 ***In-situ* structural analysis of the chikungunya virus double-stranded RNA**
Timothée Laurent, Department of Medical Biochemistry and Biophysics, Wallenberg Center for Molecular Medicine (WCMM) and Molecular Infection Medicine Sweden (MIMS), Umeå University, Sweden

#P36 **Metabolic cooperation in dual-species biofilms related to catheter associated urinary tract infections**
Dmytro Sokol, Umeå University, Sweden and Odesa I. I. Mechnikov National University, Ukraine

#P37 **Insights into alphaviral genome capping - *A mechanistic study***
Karim Rafie, Department of Medical Biochemistry and Biophysics, Wallenberg Centre for Molecular Medicine, and Molecular Infection Medicine Sweden, Umeå University, Sweden

#P38 **An alternative ATG12-ATG5-TECPR1 E3-like conjugation complex regulates unconventional LC3 lipidation at damaged membranes**
Dale Corkery, Department of Chemistry and Umeå Centre for Microbial Research, Umeå University, Sweden

#P39 **Molecular mechanisms of *Salmonella* invasion and persistence via *in vivo* transcriptomics**
Barbara Forró, Department of Molecular Biology, Laboratory for Molecular Infection Medicine Sweden (MIMS) and Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden

#P43 **Tantalosin impaires the assembly of ESCRT complex with a normal topology**
Shuang Li, Department of Chemistry and Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden

#P44 **Role of binding avidity and membrane complexity in the attachment of SARS-CoV-2 variants**
Dario Conca, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine, Umeå University, Sweden

Programme

#P45 **Discovery of novel anti-chlamydial compounds through a multi-strategy screening approach**
Magnus Ölander, Department of Molecular Biology, The Laboratory for Molecular Infection Medicine Sweden (MIMS) and Umeå Centre for Microbial Research (UCMR), Umeå University, Sweden

#P46 **Characterization of a novel penicillin-binding protein in *Vibrio cholera***
Víctor Pinedo, Department of Molecular Biology, Laboratory for Molecular Infection Medicine Sweden and Umeå Centre for Microbial Research, Umeå University, Sweden

#P18 **Structural insights of Mak proteins from *Vibrio cholera***
Nandita Bodra, Department of Chemistry, Umeå University, Sweden

#P21 **Association between Legionella species and humic substances during early summer in the northern Baltic Sea**
Karolina Eriksson, Department of Ecology and Environmental Sciences and Umeå Marine Sciences Centre, Umeå University, Sweden

#P48 **Umeå Postdoc Society: By postdocs, for postdocs**
Anaïs Lamy, Umeå Postdoc Society (UPS), Umeå University and Swedish University of Agricultural Sciences, SLU

14.00-14.10 **GROUP PHOTO in Aula Nordica *please remain seated***

14.10-15.30 **POSTER WALKS and COFFEE and CAKE**

Six groups led by PIs for organised walks (40 min)

Posters are shown in Brashörnan

Coffee and cake are served in the Lounge

15.30-17.00 **GROUP DISCUSSIONS**

Please find your group number on a table in the Universum restaurant

17.00-17.45 **KEY NOTE LECTURE II**

Chair: Teresa Frisan

Host cell remodeling by herpes virus encoded deconjugases

Professor Maria Grazia Masucci, Karolinska Institutet

17.45-18.00 **CONCLUDING REMARKS AND POSTER AWARDS**

Chair: Teresa Frisan

18.00-18.30 **RECEPTION AND MINGLE**

The reception takes place in the Lounge

18.30-22.00 **THREE COURSE DINNER**

The dinner takes place in the Universum restaurant

Tobias Dörr

PhD, Assistant professor, Department of Microbiology, Weill Institute for Cell & Molecular Biology, Cornell University, USA

Title of the lecture:

"Stress signaling promotes antibiotic resistance and tolerance in Gram-negative pathogens"

Abstract: The bacterial cell wall, made primarily from peptidoglycan (PG) ensures bacterial structural integrity and thus survival. Consequently, our most powerful antibiotics are those that target PG synthesis, typically resulting in rapid lysis and cell death. However, many bacteria are "tolerant" against cell wall-active antibiotics, i.e. they survive destruction of their cell wall and recover to growing cells upon dissipation of the drug. In this talk, I will delineate molecular mechanisms by which Gram-negative bacteria maintain structural integrity during normal growth and when exposed to cell wall-acting antibiotics.

Maria G. Masucci

MD PhD, Professor of Virology, CMB, Karolinska Institutet, Stockholm, Sweden

Title of the lecture:

"Host cell remodeling by herpes virus encoded deconjugases"

Abstract: Post-translational modification of proteins by covalent conjugation of ubiquitin or ubiquitin-like (UBL) polypeptides regulates numerous cellular processes. The effect of the modification is reversed by deconjugases that hydrolyze the covalent bond and recycle ubiquitin and the UBLs. The importance of protein ubiquitination to for the control of viral infections is underscored by the finding that many DNA and RNA viruses, including human pathogenic coronaviruses, encode Ub and UbL deconjugases that interfere with cellular processes captured by viruses to promote infection and suppress antiviral responses.

Epstein-Barr virus (EBV) is a human lymphotropic herpesvirus that is implicated in the pathogenesis of lymphoid and epithelial cell malignancies. The N-terminal domains of the herpesvirus large tegument proteins encode a conserve cysteine protease with ubiquitin and NEDD8 specific deconjugase activity. The protein is expressed during productive infection and is incorporated into virus particles suggesting possible roles during both the early and late phases of infection. We found that the EBV encoded member of this vial protein family interacts with many cellular proteins and protein complexes, which correlates with regulation of viral genome replication and the release of virus particles as well as inhibition of cellular antiviral responses including the type I IFN production and autophagy. The conserved nature of the viral enzymes and their double role in the regulation of the virus life cycle and the host antiviral response suggest that the development of specific inhibitors may be a promising new avenue of the development of effective antiviral drugs.

Poster walks

The poster session takes place **14:10-15:30 in Brashörnan**. It starts with poster walks that are organised to enhance interactions between the attendees and the poster presenters. The posters have been dived into six groups, A-F. Below, you can find a list of the groups and freely choose which group you would like to join.

Each group are led by two PIs and one group spend 5 minutes by each poster, in total 8 posters. The two PIs task is to make sure that the discussion is active and to keep track of the time. The whole walk takes 40 minutes.

After 40 minutes, all attendants can move freely in Brashörnan and view/discuss all the posters before the start of the next session.

The PIs that are leading all groups are also part of the evaluation committee of the best poster award. The three best posters will be given awards sponsored by UCMR and Agrisera.

	Poster walk group A	**Poster walk group B**	**Poster walk group C**
Panel members	**Felipe Cava** **Vicky Shingler**	**Yaowen Wu** **Göran Wadell**	**Barbara Sixt** **Lars-Anders Carlson**
Poster #, Poster presenter	#P1, Bala Anju #P2, Zlatkov Nikola #P3, Rey Varela Diego #P4, Mushtaq Fizza #P5, Seier Kerstin #P6, Bano Fouzia #P7, Toh Eric #P8, Lee Natuschka	#P9, Lee Natuschka #P11, Lambert Clara #P12, Yalamanchili Sampath Kumar #P13, Avican Ummehan #P14, Jaiman Deepika #P15, Nadeem Aftab #P16, Liu Lifeng	#P17, Henriksson Johan #P18, Bodra Nandita #P19, Verma Ashish #P20, Guest Thomas #P21, Eriksson Karolina #P22, Lopez Chiloeches Maria #P23, Avican Kemal #P24, Debiasi-Anders Gianluca

	Poster walk group D	**Poster walk group E**	**Poster walk group F**
Panel members	**Natuschka Lee** **Andre Mateus**	**Ignacio Mir-Sanchis** **Karina Persson**	**Teresa Frisan** **Johan Henriksson**
Poster #, Poster presenter	#P25, Shankar Kasturika #P26, Thorsteinsson Konrad #P27,Peters Marie Berit Akpiroro #P28, Bueno Emilio #P29, Qiao Cuncun #P30, Bueno Emilio #P31, Holmberg Sandra #P32, Zhang Jun	#P33, Ilchenko Oleksandr #P34, Feeney Rachel #P35, Laurent Timothée #P36, Sokol Dmytro #P37, Rafie Karim #P38, Corkery Dale #P39, Forró Barbara	#P40, Puertolas Balint Fabiola #P41, Gilmore Michael #P42, Sorin Marie #P43, Li Shuang #P44, Conca Dario #P45, Ölander Magnus #P46, Pinedo Victor #P47, Berg Alexandra

Poster List

Poster walk groups	Poster number	Eleva -tor talk	Last name	First name	Poster title	Research group
A	**#P1**	Yes	**Bala**	**Anju**	Insights into the genetic contexts of sulfonamide resistance among early clinical isolates of *Acinetobacter baumannii*	Bernt Eric Uhlin
A	**#P2**	Yes	**Zlatkov**	**Nikola**	Metabolic and Morphotypic Trade-Offs within the Eco-Evolutionary Dynamics of *Escherichia coli*	Bernt Eric Uhlin
A	**#P3**	Yes	**Rey Varela**	**Diego**	Siderophore transporter-based vaccines promote protection in sole fish against *Aeromonas salmonicida* infection	
A	**#P4**		**Mushtaq**	**Fizza**	Emergence of high colistin resistance in carbapenem resistant *Acinetobacter baumannii* in Pakistan and its potential management through immunomodulatory effect of an extract from *Saussurea lappa*	Irfan Ahmad
A	**#P5**	Yes	**Seier**	**Kerstin**	Characterization of Fluorescent Filovirus Pseudotypes	Marta Bally
A	**#P6**	Yes	**Bano**	**Fouzia**	Sulphation patterns of heparan sulfate modulate the interactions between human papillomavirus and cell surface glycosaminoglycans at the single particle level	Marta Bally
A	**#P7**		**Toh**	**Eric**	Bacterial protein MakA causes suppression of tumour cell proliferation via inhibition of PIP5K1A/Akt signalling	Sun Nyunt Wai
A	**#P8**		**Lee**	**Natuschka**	Antimicrobial properties and antibiotic resistance in honey	Natuschka Lee
B	**#P9**		**Lee**	**Natuschka**	Applications of One Health Principles to Spaceflight	Natuschka Lee
B	**#P10 Cancelled**		**Dahmane**	**Selma**	Membrane-assisted assembly and selective secretory autophagy of enteroviruses	Lars-Anders Carlson
B	**#P11**	Yes	**Lambert**	**Clara**	*Streptococcus pyogenes* FASII regulator FabT avoids unnecessary energy expenditure and is required for human tissue infection.	Felipe Cava
B	**#P12**	Yes	**Yalamanchili**	**Sampath Kumar**	Towards structural characterization of diacylglyceryl transferase	Karina Persson

Poster List

Poster walk groups	Poster number	Eleva -tor talk	Last name	First name	Poster title	Research group
					(Lgt) from *P. gingivalis*	
B	**#P13**	Yes	**Avican**	**Ummehan**	*In vivo* transcriptomics of *Salmonella* Typhimurium persistence	Maria Fällman, Mikael Rhen
B	**#P14**		**Jaiman**	**Deepika**	Structural and antibiotic binding comparison of LolA and LolB from Gram-negative pathogens	Karina Persson
B	**#P15**		**Nadeem**	**Aftab**	Protein-lipid interaction at low pH induces oligomerization of the MakA cytotoxin from *Vibrio cholerae*	Sun Nyunt Wai
B	**#P16**	Yes	**Liu**	**Lifeng**	Recruitment of apolipoprotein E facilitates Herpes simplex virus 1 release from infected cells	Marta Bally
C	**#P17**		**Henriksson**	**Johan**	HybridVI: Variational Autoencoder with a von Mises-Fisher and Gaussian latent space	Johan Henriksson
C	**#P18**	Yes	**Bodra**	**Nandita**	Structural insights of Mak proteins from *Vibrio cholerae*	Karina Persson
C	**#P19**	Yes	**Verma**	**Ashish**	Temperature and nutrient control of aquatic prokaryotic maintenance respiration, growth efficiency, and community composition	Johan Wikner
C	**#P20**		**Guest**	**Thomas**	Genetic dissection of LD-transpeptidation in *Agrobacterium tumefaciens*	Felipe Cava
C	**#P21**	Yes	**Eriksson**	**Karolina**	Association between Legionella species and humic substances during early summer in the northern Baltic Sea	Agneta Andersson
C	**#P22**	Yes	**Lopez Chiloeches**	**Maria**	Tissue-specific effects of genotoxigenic *Salmonella* during in vivo infection	Teresa Frisan
C	**#P23**		**Avican**	**Kemal**	A systems biology approach to uncover bacterial stress responses linked to infections	Kemal Avican
C	**#P24**	Yes	**Debiasi-Anders**	**Gianluca**	Study of the SaPI2 De-repression Complex	Ignacio Mir-Sanchis
D	**#P25**	Yes	**Shankar**	**Kasturika**	Enterovirus 2C protein and cellular membrane: what could go wrong??	Lars-Anders Carlson

Poster List

Poster walk groups	Poster number	Elevator talk	Last name	First name	Poster title	Research group
D	**#P26**	Yes	**Thorsteinsson**	**Konrad**	The effect of the lipid membrane environment on the binding kinetics of norovirus to glycolipids.	Marta Bally
D	**#P27**	Yes	**Peters**	**Marie Berit Akpiroro**	Evaluating the role of NUPs in TBEV infection	Anna Överby
D	**#P28**	Yes	**Bueno**	**Emilio**	Transient Glycolytic Complexation of Arsenate Enhances Resistance in the Enteropathogen *Vibrio cholerae*	Felipe Cava
D	**#P29**	Yes	**Qiao**	**Cuncun**	Characterization of a primase-polymerase encoded by Phage-inducible chromosomal islands	Ignacio Mir-Sanchis
D	**#P30**	Yes	**Bueno**	**Emilio**	Undecaprenyl phosphate translocases confer conditional microbial fitness	Felipe Cava
D	**#P31**	Yes	**Holmberg**	**Sandra**	Investigating Key Microbiota Molecules to Rescue Western-style Diet-induced Mucus Defects in Mice	Björn Schröder
D	**#P32**	Yes	**Zhang**	**Jun**	Chemo-optogenetic systems for reversible control of protein function in live cells	Yaowen Wu
E	**#P33**	Yes	**Ilchenko**	**Oleksandr**	Combination of gallium citrate with linezolid or levofloxacin potentiate growth inhibition of drug resistant *Mycobacterium tuberculosis* and results in differential metabolome changes	Madeleine Ramstedt
E	**#P34**	Yes	**Feeney**	**Rachel**	The importance of dietary fibre for gut health	Björn Schröder
E	**#P35**	Yes	**Laurent**	**Timothée**	In-situ structural analysis of the chikungunya virus double-stranded RNA	Lars-Anders Carlson
E	**#P36**	Yes	**Sokol**	**Dmytro**	Metabolic cooperation in dual-species biofilms related to catheter associated urinary tract infections	Madeleine Ramstedt
E	**#P37**	Yes	**Rafie**	**Karim**	Insights into alphaviral genome capping - A mechanistic study	Lars-Anders Carlson
E	**#P38**	Yes	**Corkery**	**Dale**	An alternative ATG12-ATG5-TECPR1 E3-like conjugation complex regulates unconventional LC3 lipidation at damaged membranes	Yaowen Wu

Poster List

Poster walk groups	Poster number	Elevator talk	Last name	First name	Poster title	Research group
E	**#P39**	Yes	**Forró**	**Barbara**	Molecular mechanisms of *Salmonella* invasion and persistence via *in vivo* transcriptomics	Maria Fällman
F	**#P40**		**Puertolas Balint**	**Fabiola**	Western-style diet effect on host antimicrobial peptide expression and microbiota composition along the small intestine	Björn Schröder
F	**#P41**		**Gilmore**	**Michael**	Peptidoglycan recycling mediated by an ABC transporter in the plant pathogen *Agrobacterium tumefaciens*	Felipe Cava
F	**#P42**		**Sorin**	**Marie**	Structural insight into filament proteins found in vesicles of enterovirus-infected cells and their involvement in enterovirus egress	Lars-Anders Carlson
F	**#P43**	Yes	**Li**	**Shuang**	Tantalosin impaires the assembly of ESCRT complex with a normal topology	Yaowen Wu
F	**#P44**	Yes	**Conca**	**Dario**	Role of binding avidity and membrane complexity in the attachment of SARS-CoV-2 variants	Marta Bally
F	**#P45**	Yes	**Ölander**	**Magnus**	Discovery of novel anti-chlamydial compounds through a multi-strategy screening approach	Barbara Sixt
F	**#P46**	Yes	**Pinedo**	**Victor**	Characterization of a novel penicillin binding protein in *Vibrio cholerae*	Felipe Cava
F	**#P47**		**Berg**	**Alexandra**	Nematocida displodere mechanosensitive ion channel of small conductance 2 assembles into a unique six-channel super-structure *in vitro*	Jonas Barandun
	#P48	Yes	**Lamy**	**Anaïs**	Umeå Postdoc Society: By postdocs, for postdocs	
	#P49		**Tångrot**	**Jeanette**	National Bioinformatics Infrastructure Sweden (NBIS)	
	#P50		**Bueno**	**Emilio**	Umeå Hypoxia Research Facility (UHRF)	

Poster #1 (group A)

Insights into the genetic contexts of sulfonamide resistance among early clinical isolates of *Acinetobacter baumannii*

Anju Bala[a]*, Bernt Eric Uhlin[a], Nabil Karah[a]

[a]Department of Molecular Biology, Umeå Centre for Microbial Research (UCMR), Umeå University, SE-90187 Umeå, Sweden

Since late 1930s, resistance to sulfonamides has rapidly emerged across different bacterial species including *Acinetobacter baumannii*, an opportunistic pathogen recognized as a key contributor to mounting burden of antimicrobial resistance worldwide.

Our study aimed to detect the occurrence and to explore events involved in acquisition of *sul* genes, particularly *sul2*, among the earliest available isolates of *A. baumannii*. The study utilized genomic data of 19 strains of *A. baumannii* isolated before 1985. Included were the whole genomes of 5 clinical isolates obtained from the CCUG, Sweden, sequenced in the present work using the Illumina MiSeq system. Acquired resistance genes, insertion sequence elements and plasmids were detected using ResFinder, ISfinder and Plasmidseeker, respectively, while sequence types were assigned using the PubMLST. BLASTn was used to verify the occurrence of *sul* genes and to map their genetic surroundings. The *sul1* and *sul2* genes were detected in 4 and 9 isolates, respectively. Interestingly, *sul2* appeared in isolates thirty years earlier than *sul1*. We identified different genetic contexts of *sul2*, first appearing in genomic island GI*sul2* located on a plasmid, and later evolving toward transposon Tn6172 carried on plasmid pA297-3. Sulfonamide resistance in *A. baumannii* was efficiently acquired and transferred vertically e.g., among ST52 and ST1 isolates, as well as horizontally among non-related strains by means of few efficient plasmids and transposons. Timely acquisition of *sul* genes, adding evidence on remarkable genomic plasticity of *A. baumannii*, has probably contributed to the survival skill of this nosocomial pathogen under high antimicrobial stress of hospital settings.

Metabolic and Morphotypic Trade-offs within the Eco-Evolutionary Dynamics of *Escherichia coli*

Nikola Zlatkov[1], Moa Elsa Cecilia Näsman[1], Filip Gòmez Sawicki[1], and Bernt Eric Uhlin[1]

[1]Department of Molecular Biology, Umeå University, Sweden

The species designation *Escherichia coli* encompasses an extremely broad and diverse group of bacteria that display significant differences in genome content as well as morphological and metabolic traits. In general, the species is defined as comprising enterobacteria that are rod-shaped, facultative anaerobes capable of both aerobic/anaerobic respiration and a variety of fermentative capacities, but which are also incapable of aerobic growth on citrate. There are strains designated as belonging to *E. coli* that are filamentous and capable of aerobic growth on citrate, calling into question what the real definitional boundaries of the species might be.

Here, we approach this question by examining two closely related isolates that have been identified to belong to E. coli, but which are boundary cases - one displaying a filamentous morphology (RS218) and the other capable of aerobic growth on citrate (IHE3034). The strains are two morphologically distinct "atypical" isolates of extraintestinal pathogenic *E. coli* O18:K1 that can frequently cause sepsis or neonatal meningitis. We use proteomic and metabolic approaches to examine these aberrant phenotypes and identify their bases. We found that growth on citrate by IHE3034 is improved by reduced expression of the c-di-GMP phosphodiesterase SfaY, which causes it to have higher intracellular levels of c-di-GMP. By contrast, overexpression of SfaC, which positively regulates the expression of the sfa operons, in IHE3034 caused increased filamentation while also greatly reducing the capacity to grow on citrate, whereas ceased expression of the sfa operons had the opposite effect, suggesting a trade-off between the two phenotypes that is controlled by phase variation. Finally, we also found that RS218 preferentially grows by oxidative metabolism, while IHE3034 tends toward anaerobic respiration.

The findings together show how major changes in phenotype can be controlled in a phase-dependent manner that may provide a means with which to adapt to prevailing environmental conditions.

Siderophore transporter-based vaccines promote protection in sole fish against *Aeromonas salmonicida* infection

Diego Rey-Varela[1], Miguel Balado[1], Diana Martínez-Matamoros[2], Jaime Rodríguez[2], Carlos Jiménez[2] and Manuel L. Lemos[1]

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Aeromonas salmonicida is a gamma-proteobacterium with great importance in worldwide aquaculture since it is the causal agent of furunculosis in fish, a disease which causes great losses in the aquaculture sector. One of the main virulence factors of *A. salmonicida* is the production of siderophores to obtain iron from its host. Our group has shown that this bacterium produces two different catechol-type siderophores: acinetobactin and amonabactins. In addition, we have also identified the proteins that internalize those siderophores, FstB for acinetobactin and FstC for amonabactin, being only *fstC* highly induced *ex vivo*. In this work, we propose the use of these receptors as vaccines to immune protect sole fish against *A. salmonicida*. Groups of 50 fishes were administered with purified FstB and FstC by intraperitoneal injection (30 µg of protein per fish) in 2 doses 30 days apart, at day 60 immune response of fish and protection against *A. salmonicida* was evaluated.

The results indicate that both proteins generate a good immune response, being FstB more immunogenic than FstC. Conversely, only FstC protected fish against *A. salmonicida* infection. These findings suggest that amonabactins transporter FstC can be employed for vaccine formulations against furunculosis produced by *A. salmonicida*.

Poster #4 (Group A)

Emergence of high colistin resistance in carbapenem resistant *Acinetobacter baumannii* in Pakistan and its potential management through immunomodulatory effect of an extract from *Saussurea lappa*

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Carbapenem resistant *Acinetobacter baumannii* has emerged as one of the most difficult to treat nosocomial bacterial infections in recent years. It was one of the major causes of secondary infections in Covid-19 patients in developing countries. The polycationic polypeptide antibiotic colistin is used as a last resort drug to treat carbapenem resistant *A. baumannii* infections. Therefore, resistance to colistin is considered as a serious medical threat.

The purpose of this study was to assess the current status of colistin resistance in Pakistan, a country where carbapenem resistant *A. bumannii* infections are endemic, to understand the impact of colistin resistance on virulence in mice and to assess alternative strategies to treat such infections. Out of 150 isolates collected from five hospitals in Pakistan during 2019-20, 84 percent were carbapenem resistant and 7.3 percent were additionally resistant to colistin. There were two isolates resistant to all tested antibiotics and 83 percent of colistin resistant isolates were susceptible to only tetracycline family drugs doxycycline and minocycline. Doxycycline exhibited a synergetic bactericidal effect with colistin even in colistin resistant isolates. Exposure of *A. baumannii* 17978 to sub inhibitory concentrations of colistin identified novel point mutations associated with colistin resistance. Colistin tolerance acquired independent of mutations in lpxA, lpxB, lpxC, lpxD, and pmrAB supressed the proinflammatory immune response in epithelial cells and the virulence in a mouse infection model. Moreover, the oral administration of water extract of Saussuria lappa, although not showing antimicrobial activity against *A. baumannii* in vitro, lowered the number of colonizing bacteria in liver, spleen and lung of the mouse model and also lowered the levels of neutrophils and interleukin 8 in mice.

Our findings suggest that the *S. lappa* extract exhibits an immunomodulatory effect with potential to reduce and cure systemic infections by both opaque and translucent colony variants of *A. baumannii*.

Characterization of Fluorescent Filovirus Pseudotypes

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Single particle tracking (SPT) which allows for the visualization of key steps of the virus life cycle on a single virus particle level, promises to expand the knowledge in the field of virology drastically: it offers the possibility to reveal transient and dynamic processes that are otherwise masked in static or ensemble-averaged measurements. In the context of applying SPT to studying virus attachment and entry into host cells, virus pseudotypes, i.e., particles displaying the structural core of one virus and the functional envelope glycoprotein (GP) of a heterologous virus of interest, are promising candidates: they are compatible with BSL-2 conditions and can be easily labelled. However, heterogeneities in the GP distributions represent a significant hurdle for single particle applications, as they may affect the particle behaviour. Accordingly, it is important to characterize pseudotypes on an individual particle level, to optimize production, labeling and data acquisition strategies.

In this work, we produce fluorescent pseudotypes of the deadly filoviruses Ebola and Marburg, using a lentiviral pseudotyping system with a mCherry-tagged viral core. We verify the presence of GPs on individual particles via immunostaining and fluorescence colocalization analysis. Our investigations reveal that standard particle production protocols based on transient transfection and sucrose-gradient-purification result in samples containing significant fractions of particles lacking either GP or the viral core, with large batch-to-batch variability. We show that these aspects can be significantly improved through the creation of stable cell lines for pseudotyping. To further characterize the distribution of GPs across individual particles, we use electron microscopy. The resulting images will allow us to count the number of GPs on each particle. Furthermore, we are testing if the GP density affects virus binding using biomimetic model surfaces. This enables us to directly compare the association and dissociation rates between different pseudotype variants.

A thorough characterization of pseudotype properties and the production of more homogenous samples will strengthen the interpretations of experimental results, as exemplified by our own data. In addition, understanding the viral landing and dissociation in more detail can help developing therapeutics for future outbreaks.

Poster #6 (Group A)

Sulfation patterns of heparan sulfate modulate the interactions between human papillomavirus and cell surface glycosaminoglycans at the single particle level

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Glycosaminoglycans (GAGs), negatively charged oligosaccharides and part of a soft and thick layer of sugar molecules around cells known as glycocalyx, play a vital role in initially recruiting and accumulating viruses at the cell surface. In this context, the type of GAG sulfation shown to modulate the kinetics behaviour of enveloped viruses [M. Bally *et al*., *Biophys. J*. (2017)]. However, our understanding about the interactions between non-enveloped viruses and GAGs is still very limited, specifically when it comes to their dynamics.

Here, we show how the kinetics and diffusion of a non-enveloped virus, human papillomavirus type 16 (HPV16), is regulated on the cell surface. HPV16 is the leading cause for cervical cancer [M. Schelhaas *et al*., *Cellular microbiol*., (2013)] and of great medical significance. Using a biophysical toolbox [F. Bano et., *Sci. Rep*., (2016) and M. Bally *et. al*., *ACS Chem. Biol*, (2019)], we demonstrate that 6-O sulfation patterns of heparin play a major role in mechanically strengthening the HPV16 and GAG interactions. Whereas N-sulfation patterns is critical for further stabling the binding of HPV16 virions to heparin and 2-O sulfation is dispensable. Further result indicates that HPV16 virions diffuse very slowly on heparin surfaces irrespective of N- and O- sulfation patterns.

Altogether, these results highlight the direct involvement of sulfation patterns of heparin in the first step of binding to cell surface, offering an important functional implication of GAGs for the viral attachment which may influence entry.

Bacteria protein MakA causes suppression of tumour cell proliferation via inhibition of PIP5K1α/Akt signalling

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Recently, we demonstrated that a novel bacterial cytotoxin, the protein MakA which is released by *Vibrio cholerae*, is a virulence factor, causing killing of *Caenorhabditis elegans* when the worms are grazing on the bacteria. Studies with mammalian cell cultures *in vitro* indicated that MakA could affect eukaryotic cell signalling pathways involved in lipid biosynthesis. MakA treatment of colon cancer cells *in vitro* caused inhibition of growth and loss of cell viability. These findings prompted us to investigate possible signalling pathways that could be targets of the MakA-mediated inhibition of tumour cell proliferation. After exposing *C. elegans* and cancer cells to MakA, the protein expression of PIP5K1α, pAkt, and other cell cycle proteins was measured by immunoblot analysis. Additionally, flow cytometry and clonogenic assay were performed to analyze the MakA-mediated effects on cell cycle progression and suppression of tumour cell proliferation, respectively. Furthermore, immunofluorescence analysis was conducted to detect the cell proliferation marker ki67 after MakA treatment of HCT8 colon cancer cells. Initial *in vivo* studies with MakA producing *V. cholerae* and *C. elegans* suggested that the MakA protein might target the PIP5K1α phospholipid-signalling pathway in the worms. Intriguingly, MakA was then found to inhibit the PIP5K1α lipid-signalling pathway in cancer cells, resulting in a decrease in PIP5K1α and pAkt expression. Further analyses revealed that MakA inhibited cyclin-dependent kinase 1 (CDK1) and induced p27 expression, resulting in G2/M cell cycle arrest. Moreover, MakA induced downregulation of Ki67 and cyclin D1, which led to inhibition of cell proliferation.

This is the first report about a bacterial protein that may target signaling involving the cancer cell lipid modulator PIP5K1α in colon cancer cells, implying an anti-cancer effect.

Poster #8 (Group A)

Antimicrobial properties and antibiotic resistance in honey

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Honey is produced by honeybees and is a complex mixture of different types of sugar, acids, minerals, vitamins, amino acids, pollen, microbes, and, depending on the environmental field, environmental hazards, such as pesticides. Honey has been regarded as a health promoter for thousands of years in different human cultures all around the world. However, it is only during the last decades that science has started to explore some of the parameters in honey that may promote health (for humans as well as for bees). In this study, we explored the microbiological activities in honey from different parts of Sweden and a selection of other countries.

Two different strategies were used: i) explore the microbiological composition in honey and its antimicrobial properties; ii) explore the antibiotic resistance (AR) properties of honey and microbes isolated from honey. These two strategies were analyzed via microbiological, chemical, and molecular biological methods. A large part of the tested honey samples and microbial isolates from these showed both antimicrobial as well as AR properties against different model species and complex samples such as activated sludge from the municipal wastewater treatment. Further studies are needed to explore the mechanism and role of these properties – and what kind of implications this will have for human consumption and usage of honey and for bee keeping management.

Applications of *One Health* Principles to Spaceflight

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Nowhere is human health more intimately linked with that of the environment than aboard a spacecraft. Most obviously, humans are affected by the lack of gravity and the increased radiation in space, exhibiting alterations in nearly every aspect of the body, from muscle and bone wasting to immune system suppression and thus more susceptible to microbial infections. However, astronauts' wellbeing also depends on the many different physical systems within the vehicle. For instance, the antimicrobial capacity of various surfaces may affect the risk of infection, while the built environment of the spacecraft can affect mental health.

This connection between health and environment has long been recognized through the framework of *One Health*. On Earth, *One Health* advocates for the promotion of human health alongside that of other organisms such as microbes and the environment, highlighting the inextricable links between the three. One good example of this is the incorporation of phototrophs such as plants, as this framework can be applied to leverage the environment of the spacecraft to address current and future mental and physical health challenges as humans venture farther beyond Earth. Other possible avenues for exploration include the integration of beneficial microbes and phototrophs on the spacecraft environment for food, probiotics, medicine and other essential products, as well as the creation of areas that foster community within the crew while still maximizing available space in the vehicle. Collaboration between engineers, astronauts, medical professionals, and natural scientists will be critical to ensuring a continued healthy human presence in space.

Membrane-assisted assembly and selective secretory autophagy of enteroviruses

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Enteroviruses are non-enveloped positive-sense RNA viruses that cause diverse diseases in humans. Their rapid multiplication depends on remodeling of cytoplasmic membranes for viral genome replication. It is unknown how virions assemble around these newly synthesized genomes and how they are then loaded into autophagic membranes for release through secretory autophagy.

Here, we use cryo-electron tomography of infected cells to show that poliovirus assembles directly on replication membranes. Pharmacological untethering of capsids from membranes abrogates RNA encapsidation. Our data directly visualize a membrane-bound half-capsid as a prominent virion assembly intermediate. Assembly progression past this intermediate depends on the class III phosphatidylinositol 3-kinase VPS34, a key host-cell autophagy factor. On the other hand, the canonical autophagy initiator ULK1 is shown to restrict virion production since its inhibition leads to increased accumulation of virions in vast intracellular arrays, followed by an increased vesicular release at later time points. Finally, we identify multiple layers of selectivity in virus-induced autophagy, with a strong selection for RNA-loaded virions over empty capsids and the segregation of virions from other types of autophagosome contents.

These findings provide an integrated structural framework for multiple stages of the poliovirus life cycle.

Streptococcus pyogenes FASII regulator FabT avoids unnecessary energy expenditure and is required for human tissue infection

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Membranes are a universal barrier to all cells. In most known bacteria, a phospholipid membrane structure comprises a polar head and apolar fatty acid (FA) chains. In streptococcaceae and enterococci the FA synthesis pathway (FASII) is controlled by the transcriptional repressor FabT. *Streptococcus pyogenes fabT* mutants are associated with virulence defects in *in vivo* animal models. We conducted an in-depth study using transcriptomic, lipidomic, metabolomic and cellular biology approaches to decipher the consequences of *fabT* mutation on *S. pyogenes* virulence.

Our results demonstrate on an *ex vivo* model of infection the virulence defect of a *fabT* mutant strain on human tissue. They also confirm that FabT adjusts membrane FA composition in response to environmental FAs by modulating FA chain length and composition. Moreover, a *fabT* mutant led to a specific downshift in the proportion of cardiolipin. *fabT* deregulation of fatty acid synthesis further led to defects in adhesion and growth in the presence of eucaryotic cells, growth during which wild-type and *fabT* mutant strains. The *fabT* growth defect is linked to an increased susceptibility to cell-elicited toxicity and to energy consumption due to unnecessary FASII activity, constituting a fitness defect. This establishes the link between the status of the FASII transcriptional repressor FabT and virulence.

Towards structural characterization of diacylglyceryl transferase (Lgt) from *P. gingivalis*

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Lipoproteins of Gram-negative pathogens carry out various functions from nutrient uptake to invasion and virulence. Before they are localized to the outer membrane, three step post-translational lipid modifications occur in the inner membrane. Diacylglyceryl transferase (Lgt) an integral membrane protein catalyzes the first step by recognising the 'lipobox' motif ($[LVI]^{(-3)}[ASTVI]^{(-2)}[GAS]^{(-1)}C^{(+1)}$) of prolipoproteins and transferring the diacylglyceryl group from phosphatidylglycerol (PG) to the thiol group of the conserved cysteine residue in the lipobox sequence. In *Porphyromonas gingivalis* (*P. gingivalis*) bacteroidete, the fimbrial subunits lack the lipobox motif but the first cysteine from the N-terminal is diacylated by Lgt.

To understand the substrate recognition and catalytic mechanism of Lgt (from *P. gingivalis*), we have recombinantly expressed and purified the enzyme. CD measurements of purified Lgt suggested that it is 93.0% α-helical and folded. Mass photometry results resemble protein-detergent complex. Crystallization experiments resulted in microcrystals that diffracted to low resolution. Optimization of crystallization condition for structural characterization is being carried out.

Overall, these results lay the foundation for structural work that will enhance our knowledge on enzyme mechanics and aid in developing new antibiotics.

In vivo transcriptomics of *Salmonella* Typhimurium persistence

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Two pathogenic serovars of *Salmonella enterica* is of importance globally causing two distinct diseases: Broad host range adapted non-typhoidal serovar *S.* Typhimurium causing a self-limiting gastroenteritis and human adapted typhoidal serovar *Salmonella* Typhi causing typhoid fever with a potentially fatal outcome. In typhoidal and non-typhoidal *Salmonella* infected individuals of which 3.5 percent and 2.2 percent become asymptomatic chronic carriers of the disease where the bacteria persist in gallbladder and liver. Studying *S.* Typhi infections in mouse models are challenging since it is a human adapted pathogen. There are many studies that successfully have revealed some of the single genetic determinants of acute salmonellosis in mice. However, the adaptational requirements of persistent bacteria in different niches is not fully understood.

In this study, we have established two different chronic mouse infection models where *S.* Typhimurium persists in the intestine and/or in liver for more than 6 weeks. To understand the molecular mechanisms of bacterial long-term residence, we applied global transcriptomic analysis of bacteria from those infected tissues. This was achieved by developing bacterial RNA enrichment methods both pre- and post-library preparation concerning the very small ratio of bacterial RNA in the infected tissues. A novel bioinformatic pipeline was also developed for applications of *in vivo* transcriptomic data. Analysis of differentially regulated genes revealed transcriptional reprogramming of several different pathways such as virulence and metabolic processes. Deletion of selected genes of which transcription is consistently distinct in different tissues has been performed and resulting mutant strains will be tested in the mouse infection models.

Structural and antibiotic binding comparison of LolA and LolB from Gram-negative pathogens

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The bacterial cell envelope plays an important role in maintaining viability, growth, and pathogenicity. Gram-negative bacteria contain double cell membranes in contrast to gram-positive bacteria. The outer membrane of gram-negative bacteria is often associated with pathogenicity and serves as a barrier against antibiotics. Lipoproteins are one of the most important structural and functional components of the cell envelope. They perform various physiological functions and have virulence-associated roles like antigenicity and colonization. **L**ocalization **o**f **l**ipoproteins is essential for outer membrane biogenesis which is carried out by the **Lol** pathway. Lol pathway consists of - an integral membrane complex LolCDE (ABC transporter), a periplasmic chaperone LolA and an outer membrane-anchored receptor LolB. LolB is anchored on the outer membrane and transfer lipoproteins from LolA to the inner leaflet of the outer membrane.

We have solved the crystal structure of LolA from *Vibrio cholerae* and *Porphyromonas gingivalis* and LolB from *Vibrio cholerae*. This has broadened the present repertoire of LolA/B structures across the broad phylum of gram-negative bacteria and enabled us to examine distinct structural features. Interestingly, we found that LolB *Vibrio cholerae* binds only with LolA from *Vibrio cholerae* (30.2 ±0.28 μM) but not with LolA *P.gingivalis*. We have also performed ITC analysis to check binding affinities of Lol protein with antibiotic Polymyxin B. LolA *P.gingivalis* K_d 13.8 ±0.36 μM, is higher than LolA *Vibrio cholerae* K_d 56 ± 0.63 μM. This could be due to relative bigger, thus accessible hydrophobic core of LolA *P.gingivalis*. These results encourages to also check binding of polymyxin derived antibiotics like polymyxin nonapeptide, colistin or NAB7061, SPR741/NAB741 with LolA.

The emergence of multidrug resistance demands the search for new antibacterial targets; structure determination of Lol Machinery members lays foundation for drug development with help of structure-based drug design. Disruption of an essential Lol machinery could destabilize the cell wall of *V.cholerae* and *P.gingivalis* making them susceptible to available antibiotics.

Poster #15 (Group B)

Protein-lipid interaction at low pH induces oligomerization of the MakA cytotoxin from *Vibrio cholerae*

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The α-pore-forming toxins (α-PFTs) from pathogenic bacteria damage host cell membranes by pore formation. We demonstrate a remarkable, hitherto unknown mechanism by an α-PFT protein from *Vibrio cholerae*. As part of the MakA/B/E tripartite toxin, MakA is involved in membrane pore formation similar to other α-PFTs. In contrast, MakA in isolation induces tube-like structures in acidic endosomal compartments of epithelial cells in vitro. The present study unravels the dynamics of tubular growth, which occurs in a pH-, lipid-, and concentration-dependent manner. Within acidified organelle lumens or when incubated with cells in acidic media, MakA forms oligomers and remodels membranes into high-curvature tubes leading to loss of membrane integrity. Cutting edge imaging techniques including i) spinning disc confocal microscopy, ii) scanning electron microscopy (SEM), iii) transmission electron microscopy (TEM), iv) cryo-electron microscopy (cryo-EM), and v) fluorescence microscopy were used to visualize the kinetics of MakA induced membrane tubulation. A 3.7 Å cryo-electron microscopy structure of MakA filaments reveals a unique protein-lipid superstructure. MakA forms a pinecone-like spiral with a central cavity and a thin annular lipid bilayer embedded between the MakA transmembrane helices in its active α-PFT conformation.

Our study provides insights into a novel tubulation mechanism of an α-PFT protein and a new mode of action by a secreted bacterial toxin.

Recruitment of apolipoprotein E facilitates Herpes simplex virus 1 release from infected cells

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Over two decades, epidemiological studies have revealed that interactions between human polymorphic apolipoprotein 4 (ApoE, isoform 4) and herpes simplex virus type 1 (HSV1) associate with higher risk of Alzheimer's disease, a serious and increasing issue among elder populations worldwide. Nevertheless, little is known about the mechanisms behind ApoE-HSV1 interactions at molecular levels. Here, we investigate the effects of ApoE on the HSV1 infectious life cycle in *in vitro* cell experiments. Analysis of HSV1 growth curves shows that HSV1 production is promoted in presence of any of the three ApoE isoforms, with ApoE3 or 4 demonstrating more efficient pro-viral effects than ApoE2. When added prior to infection, ApoE inhibits viral attachment but does not affect viral entry. qPCR-based quantification reveals that harboring ApoE2, 3, or 4 leads to an increase of HSV1 extracellular release but unchanged levels of viral genome copies within cells or on cell surface, indicating that virus replication, assembly, or transport to cell membrane are not affected.

Further test of virus release directly demonstrates that HSV1 detachment from cell surface is promoted by ApoE. Subsequent results reveal that ApoE is not only present in purified HSV1 particles produced in ApoE expressing cells after ultra-centrifugation but also able to incorporate into HSV1 particles after purification, suggesting that harboring ApoE may promote HSV1 detachment. With the help of total internal reflection microscopy (TIRFM), this hypothesis was tested by quantifying interaction kinetics and apparent affinity between of HSV1 and native supported lipid bilayer. HSV1 particle decorated with ApoE demonstrates higher dissociation rate constants (k_{off}) and less irreversible binding to the membrane, which is in line with the promoted virus release. Similar interaction kinetics have also been tested between non-decorated HSV1 particles and membranes harboring ApoE but revealed no difference in the kinetics of virus particles on membranes with ApoE present or absent. Overall, our results provide new insights into the roles of ApoE during HSV1 infections, which is worth to be considered when studying their involvement during AD development.

HybridVI: Variational Autoencoder with a von Mises-Fisher and Gaussian latent space

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Single-cell analysis is challenging due to the large number of molecules, usually DNA or RNA, present for each cell. This creates a need for dimension reduction to visualize and interpret the data. This is commonly done with linear methods such as PCA, or non-linear methods such as UMAP or t-SNE. However, recent years have seen a rise in utilizing machine learning. One of the most promising methods is the generative method, Variational Autoencoders (VAEs).

Briefly explained, autoencoders work by compressing data to a lower dimension and then reconstructing it back to the original dimension. The goal is that the output should be as similar as possible to the input. All autoencoders create a compressed version of the data by utilizing latent variables. VAEs stand out from other autoencoders by further regularizing these latent variables, forcing them to stay close to a simple distribution.

These latent variables usually belong to a Gaussian distribution. However, in this case the goal was to have latent variables that belong to two different distributions. The reasoning behind this is to be able to utilize kmariaown biological processes in the data. In this study the focus is on cell cycle genes. Evidence suggests that cell cycle genes are expressed in a periodic manner. The goal was to utilize this information by mapping these genes to latent variables with a circular distribution.

Implementing these ideas resulted in HybridVI, a VAE with latent variables from two distributions: Gaussian and von Mises-Fisher. Initial results indicate that when finding clusters, a VAE with one Gaussian latent space performs better. We are currently investigating how this additional information affects the model's biological interpretation.

Structural insights of Mak proteins from *Vibrio cholerae*

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Vibrio cholerae is one of the known gram-negative bacterial pathogens responsible for diarrhea in humans. Cholerea toxin (CT) and toxin regulated pili (TCP) are the main factors to cause the disease. Most *V. cholerae* strains lack CT genes yet they are considered pathogenic as they cause other complications such as skin, wound and gastrointestinal infections. Dongre *et al.* discovered new virulence factors of *V. cholerae* called motility associated killing (mak) factors using *Caenorhabditis elegans* as an infection host model. The mak operon encodes five proteins namely MakA, MakB, MakC, MakD and MakE. The MakA, MakB and MakE are soluble proteins and upon interaction with the host membrane they form the MakABE tripartite complex which is cytotoxic. The structures of MakABE tripartite complex and MakC are not known.

Here, in my project I aim to get the structural insights of MakC and MakABE pore complex to understand the molecular detail of interactions amongst Mak proteins and the host membrane. For this, I use two major macromolecular structural determination techniques i.e., X-ray crystallography and electron microscopy. So far we have solved the structure of MakC by single-wavelength anomalous diffraction. Additionally, the MakABE protein complex has been made in the presence of lipid extract and detergent for single particle cryo-EM analysis. The outcome of this will aid to understand bacterial pathogenesis and to design new therapeutics against bacterial infections.

Temperature and nutrient control of aquatic prokaryotic maintenance respiration, growth efficiency, and community composition

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Mesocosm experiments are used in microbial ecology to understand causality between natural processes not readily disentangled by field data. Prokaryotes constitutes one third of the aerobic respiration in the marine ecosystems and the bioenergetic cost can be attributed to maintenance activities and biomass synthesis. Knowledge of prokaryotic maintenance respiration is limited in natural ecosystems and its estimation is valuable with respect to understanding regulation of respiration and the adaptation strategies of prokaryotes in cold and low productive ecosystems. Recent evidence from a field study in the marine environment suggest that specific maintenance respiration rate account for more than half of annual prokaryotic respiration and is regulated by specific growth rate.

In the present study, temperature control and nutrient additions were used to simulate winter and summer conditions to control prokaryotic specific growth rates and validate the relationship observed in the field. The patterns of the specific respiration and growth rates observed in the field could be reproduced in the experiment. Based on repeated measures ANOVA, temperature was the main controlling factor for several prokaryotic variables with no significant interaction effects with nutrients. Maintenance respiration accounted for 75% and 15% of cell-specific respiration corresponding to winter and summer conditions, respectively. A global range of prokaryotic growth efficiencies (0.05-0.57) and specific growth rates (0.06-2.7 d^{-1}) were obtained. Metabarcoding of 16S rRNA gene revealed different taxonomic diversity with specific taxa associated with maintenance and growth conditions, respectively. In summary, the prokaryotes efficiently increased their biomass in summer conditions, while the high respiration in winter conditions was attributed to maintenance activities.

This experimental design provides further opportunity to investigate prokaryotic maintenance activities in aquatic environments by gene expression, proteomics, and electron microscopy.

Genetic dissection of LD-transpeptidation in *Agrobacterium tumefaciens*

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Agrobacterium tumefaciens is a rod-shaped, plant pathogenic bacterium that grows from a single pole. This species lacks many of the canonical cell wall assembly proteins, including MreB, RodA and PBP2 that are essential for cell elongation in *E. coli*. The structure of the peptidoglycan produced by *A. tumefaciens* also differs; it is more highly crosslinked and enriched for the LD-type of crosslink. LD crosslinks are formed between the L and D chiral centres of mDAP by LD transpeptidases (LDTs). The genome of *A. tumefaciens* encodes 14 putative LDTs and are thought to play an important role in achieving unipolar growth. In this project, we have systematically examined the role of each of these proteins in peptidoglycan assembly, maintenance of cell shape and their role in sustaining a cell wall that protects cells from injury.

We show that LD transpeptidation is essential for *A. tumefaciens* fitness. The activities of LDTs are partially redundant, but that a subset, specific to *A. tumefaciens* and closely related species, are necessary for maximal fitness, maintaining cell shape and sustaining unipolar growth.

Poster #21 (Group C)

Association between *Legionella* species and humic substances during early summer in the northern Baltic Sea

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Climate change is projected to cause alterations in northern coastal systems, including humification and intensified nutrient loads, which can lead to ecosystem imbalances and establishment of new bacterial species. Several potential pathogens, such as different species of *Legionella,* hide in the environment between infections, some by living inside protozoan host cells. Knowledge about the occurrence of *Legionella* in natural waters is missing, which disable risk assessments of exposure. We performed a study of the species diversity of *Legionella* in the northern Baltic Sea (Gulf of Bothnia) during early summer to map their occurrence and to identify possible environmental drivers.

We detected *Legionella* and potential protozoan hosts along gradients of the Gulf of Bothnia. We also for the first time present third generation full-length 16S rRNA amplicon sequencing (Nanopore) to resolve environmental species classification of *Legionella*, with a method suitable to study all bacteria. Our data show that full length 16S rRNA sequences is sufficient to resolve *Legionella* while the standard short Illumina sequences did not capture the entire diversity. For accurate species classification of *Legionella*, harmonization between the Nanopore classification methods is still needed and the bias toward the well-studied *Legionella pneumophila* need to be resolved. Different *Legionella* species occurred both in the Bothnian Sea and in the Bothnian Bay and their abundance were linked to humic substances and low salinity. The relative abundance of *Legionella* was higher in the humic-rich northern waters of the Bothnian Bay. The link between *Legionella* species and humic substances may be indirect via promotion of the heterotrophic microbial food web, allowing *Legionella* species and similar bacteria to establish. Humic substances are rich in iron, which has been shown crucial for growth of *Legionella* species and other pathogens. Considering climate change projections in this regional area, with increased humification and freshwater inflow, this bacterial niche containing potential pathogens might become more widespread in the future Baltic Sea.

This study demonstrates the significance of DNA sequencing to monitor public health relevant bacteria like *Legionella* species in the environment. Including sequencing of bacteria and protozoa in the environmental monitoring programs could be used to identify ecosystem imbalances, which enable appropriate responses to emerging diseases.

Tissue-specific effects of genotoxigenic *Salmonella* during *in vivo* infection

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Bacterial genotoxins (BTGX) are effectors that induce DNA damage in host cells via their cdtB subunit, whose activity is similar to the mammalian DNase I. Among the different families of BTGX, we are interested in the typhoid toxin (TT) which is encoded by typhoidal and non-typhoidal *Salmonella* serovars. Upon induction of DNA damage, the DNA Damage Response (DDR) is activated. However, if the DNA damage is beyond repair, most cells undergo a permanent, pro-inflammatory cell cycle arrest known as senescence. Besides, some damaged cells which present pro-carcinogenic features can proliferate. Even though that these features suggest that BTGX play a role as cancer promoters/inducers, it is unlikely this would be their main biological function.

To study the role of the TT during infection, we established an *in vivo* infection model where we used a modified *Salmonella* Typhimurium strain that expresses the TT. At 10 days post-infection, we assessed the effect of the TT on the host immune response, induction of DDR and senescence, and the presence of the bacteria in both colon, as the main site of infection, and liver, where *Salmonella* disseminates and persists in chronic carriers. We described the population of immune cells and activation of DDR by immunofluorescence as well as further characterised the expression of specific cytokines and the phenotype of senescence by *in situ* transcriptomics using multiplex RNAscope™. Previous studies in our group described the immunomodulatory effects of the TT and induction of DNA fragmentation and senescence in colon. Here, we showed that colonic DNA damage was associated with the presence of *Salmonella* in genotoxigenic-infected mice and coupled with higher expression levels of the inflammasome sensor *Aim2*, which is associated with detection of cytosolic DNA. This promoted the production of type I *Ifn* and induction of the DNA-damage downstream target p53. On the other hand, induction of DDR in liver was toxin-independent and the TT seemed not to have a contribution on the pro-inflammatory landscape nor on induction of senescence. Interestingly, we observed that TT promoted the expression of the inflammasome sensor *Nlrp3*, which is activated upon different cellular stimuli like ROS production.

Together, this study describes the tissue-specific consequences of DNA damage induced by genotoxigenic-producing *Salmonella* highlighting the complex interplay between the immunological landscape and induction of senescence.

A systems biology approach to uncover bacterial stress responses linked to infections

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Gene products involved in the stress responses of bacteria in stressful host environments are attractive targets for antimicrobials. However, our knowledge about such responses is still limited. In order to reveal stress responses in a diverse range of human bacterial pathogens, we cataloged gene expression profiles of 32 human bacterial pathogens under 11 infection-relevant stress conditions for 105 088 genes, deposited in the PATHOgenex RNA atlas. We will now complement this dataset with *in vivo* gene expression profiles of bacterial pathogens in different infected human specimens. To dissect the complexity and extract important information from the rich and solid datasets at both the single cell and population level, we will apply systems biology, which integrates biological and medical sciences with mathematical and computational disciplines by employing different approaches, including trained artificial intelligence algorithms.

We aim to (i) identify active bacterial molecular pathways in difficult-to-treat polymicrobial and biofilm infections, (ii) predict gene products critical for infections, and (iii) test promising candidates as antimicrobial targets in *in vivo* adapted model systems. To reach those aims, we are currently developing state-of-the-art molecular biology tools and methods to reach bacterial transcriptomes at the infection site at both population and single cell levels. During and after high-throughput data generation, we will employ mathematical and computational models to analyze and reveal hidden information that could be useful to identify potential target genes for combating difficult-to-treat bacterial infections.

Study of the SaPI2 De-repression Complex

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Staphylococcus aureus Pathogenicity Islands (SaPIs) are mobile genetic elements that spread by hijacking compatible (helper) bacteriophage particles. SaPI quiescence is maintained during homeostasis by the SaPI-encoded repressor protein known as SaPI Transcription Leftward (Stl). Stl serves as the SaPI's master repressor by blocking transcription of the excision, replication, and packaging (ERP) machinery. Different Stls from different SaPIs can bind to specific groups of phage-derived proteins, leading for the formation of complexes that result in the release of Stl from the ERP promoter region, initiating the SaPI ERP cycle. This interaction therefore directly disinhibits SaPI activity while also serving as an indicator of SaPI-phage compatibility.

The Stl from SaPI2 binds annealases, which are ubiquitous proteins that participate in DNA recombination by catalyzing ssDNA base pairing and annealing. The staphylococcal temperate bacteriophage 80α carries the annealase Sak, which has been shown to interact with SaPI2-Stl and lead to SaPI2 mobilization. Annealases typically have a C-terminal domain (CTD) responsible for protein-protein interactions, however these have usually not been seen in structures either due to the CTD's inherent flexibility or because it prevents crystallization. Removal of Sak's CTD has been shown to prevent SaPI2 induction *in vivo*, meaning this domain is likely responsible for 80α-Sak/SaPI2-Stl complexing.

Through cryoEM, we show that Sak (24kDa+6xHis-tag) has a typical annealase disposition with a homoheptadecameric toroidal/ring-like structure of ~450kDa that dimerizes upon addition of ssDNA. Mixing Sak and Stl creates complexes over 1.4mDa where Stl (26kDa) binds and stabilizes Sak's CTD, making it visible for the first time.

Enterovirus 2C protein and cellular membrane: what could go wrong??

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The *Enterovirus* genus of the Picornaviridae family includes non-enveloped, positive-sense single stranded RNA (ssRNA) viruses. This genus of virus cause many clinically relevant diseases like poliomyelitis by poliovirus (PV), cardiomyopathy by coxsackie B3 (CVB3), common cold rhinoviruses (RVs) and meningitis Enterovirus 71 (EV 71). And most of the diseases caused by this genus has no vaccine or drugs against them.

The 7.5 kb genome encodes a polypeptide which is subsequently cleaved to yield viral proteins. These viral proteins hijack the cellular machinery and modify the membranes of the Golgi and ER to give rise to Replication complexes (RCs). It is on these membranous platform that the virus replication and assembly takes place. One such viral protein is 2C. A hexameric, AAA+ ATPase and SF3 helicase which play an important role in viral uncoating, biogenesis of RCs and virus assembly. One of the most interesting feature of 2C is its N-terminal which has two amphipathic helices consisting of 40 amino-acids. The sequence of the amphipathic helix is conserved in all Enteroviruses. So, 2C has potential to be a pan-Enteroviral drug target.

My research question is to investigate the membrane -binding of 2C and how this affects the function of the protein. Initial results suggest that amino acids 12 to 40 are not only important for membrane binding but also for hexamerization. And the protein binds better to negatively-charged lipid head group found in the ER and prefers membrane with high curvature. I was also able to demonstrate that the protein can recruit RNA on the membrane which is very important for its helicase activity.

This study will open doors for designing drugs to inhibit the interaction between 2C and cellular membranes. Thereby, contributing to curb *Enterovirus* infections.

The effect of the lipid membrane environment on the binding kinetics of norovirus to glycolipids

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Human noroviruses (HuNoV) are one of the leading causes of gastroenteritis worldwide, causing an estimated 200.000 deaths annually. Despite significant effort, the processes leading to initial infection of susceptible cells are not fully understood, and the main viral receptor(s) are still unknown. The virus primarily infects epithelial cells in the gastrointestinal tract, and susceptibility is strongly correlated with the presence of histo-blood group antigens (HBGAs) found among other on glycosphingolipids. However, the full relationship between HuNoV infectivity and HBGA expression is still being researched. Susceptibility to infection depends on which HBGAs are expressed, but not all HBGA-carrying cells in the human body are susceptible to infection.

In this project, we studied how the lipid membrane environment affects the interaction between HuNoV and glycolipid-bound HBGAs. To this end, we focused on the H-type 1 glycan, a HBGA known to be recognized by HuNoV. We incorporated H-type 1 into synthetic membranes of various composition and compared attachment and detachment behaviors to examine how different lipid components influence the binding affinity. To do so, we utilized a biosensing platform involving supported lipid bilayers to model the plasma membrane, and TIRF microscopy to quantify the binding kinetics of fluorescent HuNoV to the membrane-embedded H-type 1. This way, we could measure, in real-time, on a single particle scale, viruses binding to the surface and their residence times, giving us independent measures for the on- and off-rates.

Our results show that membrane-cholesterol significantly enhances binding, whereas membranes produced from lipid extracts, more closely resembling the lipid composition of native plasma membranes, have an inhibitory effect. These results give us a better insight of the initial stages of infection and the tropism of the virus in general.

Evaluating the role of NUPs in TBEV infection

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laviviruses are arthropod-borne viruses that constitute a major global health problem, with millions of human infections annually and no antiviral treatment currently available. Tick-borne encephalitis virus (TBEV) is the most common tick-born flavivirus. It is a small (40-60 nm), spherical, lipid enveloped, RNA virus that internalizes by endocytosis, replicates in replication "vesicles", that are formed in the endoplasmic reticulum (ER), and further assembles and matures across the ER and the Golgi until its egress. Viruses use both viral and cellular factors to complete their life cycle, yet several of these host factors remain unidentified and uncharacterized. Nucleoporins (NUPs) have previously been suggested to be important in the life cycle of several RNA viruses, but still not a lot is known about their role in flavivirus infection.

Here, we identify NUP153 and NUP98 to be important for TBEV infection. We observe that the proteins are recruited to the replication site, where NUP153 interacts with C (capsid protein), NS3 and NS5, while NUP98 co-localizes with dsRNA at the replication vesicles. Using CLIP (Cross-link Immunoprecipitation) we also observe that both NUP153 and NUP98 are able to bind viral RNA. Furthermore, mass spectrometry analysis revealed a slight shift in the interactome of both NUP153 and NUP98, yet they do not interact with each other. Interestingly NUP98 is able to bind to proteins that are part of the Mediator complex, which is involved in the regulation of the transcription machinery and has previously been mainly studied in the context of DNA viruses, yet their function in RNA viruses is not known.

Taken together our results suggest that NUP153 and NUP98 are recruited to the site of replication to mediate important functions in the viral life cycle. The data indicates that NUP153 might mediate the transport of viral RNA from the replication vesicles to the assembly site, while NUP98 on the other hand seem to be more involved in the viral replication.

Transient Glycolytic Complexation of Arsenate Enhances Resistance in the Enteropathogen *Vibrio cholerae*

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The ubiquitous presence of toxic arsenate (As^{V}) in the environment has raised mechanisms of resistance in all living organisms. Generally, bacterial detoxification of As^{V} relies on its reduction to arsenite (As^{III}) by ArsC, followed by the export of As^{III} by ArsB. However, how pathogenic species resist this metalloid remains largely unknown.

Here, we found that *Vibrio cholerae*, the etiologic agent of the diarrheal disease cholera, outcompetes other enteropathogens when grown on millimolar concentrations of As^{V}. To do so, *V. cholerae* uses, instead of ArsCB, the As^{V}-inducible *vc1068-1071* operon (renamed *var* for vibrio arsenate resistance), which encodes the arsenate repressor ArsR, an alternative glyceraldehyde-3-phosphate dehydrogenase, a putative phosphatase, and the As^{V} transporter ArsJ. In addition to Var, *V. cholerae* induces oxidative stress-related systems to counter reactive oxygen species (ROS) production caused by intracellular As^{V}. Characterization of the *var* mutants suggested that these proteins function independently from one another and play critical roles in preventing deleterious effects on the cell membrane potential and growth derived from the accumulation As^{V}. Mechanistically, we demonstrate that *V. cholerae* complexes As^{V} with the glycolytic intermediate 3-phosphoglycerate into 1-arseno-3-phosphoglycerate (1As3PG). We further show that 1As3PG is not transported outside the cell; instead, it is subsequently dissociated to enable extrusion of free As^{V} through ArsJ. Collectively, we propose the formation of 1As3PG as a transient metabolic storage of As^{V} to curb the noxious effect of free As^{V}.

This study advances our understanding of As^{V} resistance in bacteria and underscores new points of vulnerability that might be an attractive target for antimicrobial interventions.

Characterization of a primase-polymerase encoded by Phage-inducible chromosomal islands

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Staphylococcus aureus Pathogenicity Islands (SaPIs) are a group of the phage-inducible chromosomal island (PICI) family of mobile genetic elements. SaPIs are quiescently integrated in the host genome and after induction by a bacteriophage, replicate autonomously due to their own replication module consisting of a primase (Pri), a self-loading helicase (Rep), and a SaPI's origin of replication. The functions of Reps have been studied previously but those for Pri are still mysterious. For example, Pri and Rep are essential in replication studies in a suicide plasmid; however, a deletion mutant of *pri* does not show any defect when SaPI replication is measured in its physiological cycle suggesting that host primase or polymerase might complement in the absence of Pri in the SaPI cycle but not in a suicide plasmid.

To better understand the particularities of Pri in the context of SaPI replication, here we have characterized Pri (Pri1) present in SaPI1. Based on sequence homology, Pri1 belongs to the PriCT_1 domain-containing protein. It is composed of a core catalytic N-terminal domain (NTD) and DNA binding C-terminal domain (CTD) and is a dimer in solution based on gel filtration. Truncation versions of Pri showed that the presence of CTD (Pri1Δ-NTD) is responsible for the dimerization whereas its absence (Pri1Δ-CTD) generated a soluble monomer. The Pri1 could synthesis a primer *de novo* using a single-stranded DNA (ssDNA) template that contains the sequence of 5'-d(GTG)-3', indicating the primase activity. We analyzed Pri1 in a DNA primer extension assay, showing that it extends the primer incorporating both deoxyribonucleotides (dNTPs) and ribonucleotides (NTPs). We observed high promiscuity in its terminal transferase activity, being able to extend ssDNA with both NTPs and dNTPs. Furthermore, deletion of CTD did not affect the polymerase activity, but completely abolished the primase and terminal transferase activities, suggesting that CTD is critical for the primase and the terminal transferase activities. Additionally, mutation of a conserved residue, Asp 92, resulted in the loss of all three activities.

Altogether, this novel Pri1 harbors multifunction of primase activity, polymerase activity, and terminal transferase activity.

Undecaprenyl phosphate translocases confer conditional microbial fitness

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The microbial cell wall is essential for cell shape maintenance and resistance to external stressors. The primary structural component of the cell wall is peptidoglycan (PG), a glycopolymer with peptide crosslinks located outside of the cell membrane. PG biosynthesis and structure are responsive to shifting environmental conditions such as pH and salinity, but mechanisms underlying such adaptations are incompletely understood. Precursors of PG and other cell surface glycopolymers are synthesized in the cytoplasm and then delivered across the cell membrane bound to the recyclable lipid carrier undecaprenyl phosphate (C55-P). The transporter protein(s) that return C55-P to 31 the cytoplasmic face of the cell membrane have been elusive.

Here, we identify the 32 DUF368-containing and DedA transmembrane protein families as candidate C55-P translocases, filling a critical gap in knowledge of the proteins required for the 34 biogenesis of microbial cell surface polymers. Gram-negative and -positive bacteria 35 lacking their cognate DUF368-containing protein exhibited alkaline-dependent cell wall and viability defects, along with increased cell surface C55-P levels. pH-dependent synthetic genetic interactions between DUF368-containing proteins and DedA family members suggest that C55-P transporter usage is dynamic and modulated by environmental inputs. C55-P transporter activity was required by the cholera pathogenfor growth and cell shape maintenance in the intestine. We propose that conditional transporter reliance provides resilience in lipid carrier recycling, bolstering microbial fitness within and outside of the host.

Investigating Key Microbiota Molecules to Rescue Western-style Diet-induced Mucus Defects in Mice

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Mucus is a gel-like structure lining the gastrointestinal tract. It serves as the first barrier preventing the colonization of pathogenic bacteria. Goblet cells, the main mucin producers of the gut, continuously secrete their content. In the distal part of the colon, where bacterial load is highest, the production and expansion of mucus are important processes to lower the risk of inflammation and infection. The consumption of a Western-style diet (WSD), low on fibers and high in fat and simple sugars, impairs colonic mucus properties and changes bacterial composition in mice. In previous experiments, high-fiber-derived human microbiota, or specific bacteria alone, was observed to prevent WSD-induced mucus impairments. However, the mechanistic role of diet and bacterial stimuli on goblet cell and mucus function is not fully understood. Therefore, we aim to identify key molecules, such as secreted metabolites, important for mucus property regulation. We demonstrate the impact of specific bacteria on mucus function in mice and look at the direct effects of single metabolites. A specialized *ex vivo* mucus measurement technique is employed on mice colonic tissue to assess mucus thickness and growth alternations. Clues towards key microbiota molecules and metabolites are gathered through 16S microbiota sequencing and targeted metabolic profiling of fecal and mucus samples, respectively. Subsequently, the *ex vivo* technique allows us to explore the direct effects of suspected mucus growth stimulants.

Our findings help to understand interactions between diet, host, and microbes. In the future, identified key molecules may serve in the care of WSD-associated diseases.

Chemo-optogenetic systems for reversible control of protein function in live cells

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Optogenetic systems using photosensitive proteins and chemically induced dimerization (CID) approaches are powerful tools to elucidate the dynamics of biological systems and to dissect the complicated biological regulatory networks. We and others have developed chemo-optogenetic systems using photocaged and/or photocleavable chemical dimerizers, which have an improved dynamic range and a very rapid turn-on/off speed. However, photolysis of photolabile groups is not reversible, therefore, these systems are usually subjected to only one-time control by light, which limits the scope of application in biological processes that require cycling or gating.

Herein, we present a chemo-optogenetic system with photoswitchable CIDs (sCIDs) that can undergo multiple rounds of light-induced dimerization, enabling reversible control of cellular processes by light. We use this system to reversibly control protein dimerization, protein localization, and positioning of organelles in cells with light. We envision the chemo-optogenetic approaches using sCIDs could open a new avenue for spatiotemporal control of protein or cargo distribution, which could be highly valuable to dissect a wide range of dynamic cellular processes.

Combination of gallium citrate with linezolid or levofloxacin potentiate growth inhibition of drug resistant *Mycobacterium tuberculosis* and results in differential metabolome changes

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Tuberculosis, an airborne infection, affects 10 million people and results in the deaths of 1.5 million people per year. A major challenge in treating tuberculosis is the increasing antibiotic resistance of *Mycobacterium tuberculosis* (*Mtb*), the bacterium that causes the disease. Tuberculosis is treated with a combination of four active antibiotics depending on the drug resistance profile of Mtb strain., The development of drug-resistance in *Mtb* has made it necessary to find new antibiotics to ensure availability of 4 active drugs for an effective 4-drug regimen.

In this study we aimed to compare the growth inhibition and potential metabolome changes in a pre-extensively drug resistant (pre-XDR) *Mtb* strain when it is cultured in the presence of gallium in combination with established anti-tuberculosis drugs linezolid and levofloxacin. The pre-XDR strain was grown using the BACTEC 960 system in different experimental conditions and cells were harvested and analyzed using gas chromatography mass spectrometry. We applied PCA and OPLS-DA to follow metabolite patterns at different conditions. The results showed that the pre-XDR *Mtb* strain responded to gallium treatment in a dose-dependent manner and showed increased growth inhibition when combined with traditional antibiotics. Maximum growth inhibition was achieved with the combination of gallium and levofloxacin. Different growth conditions were associated with distinct metabolite patterns, indicating that the combination of gallium with antibiotics results in metabolome alteration. A significant increase of aconitate was observed in all samples containing gallium, which may be due to inhibition of aconitase, an enzyme containing a Fe^{3+} cluster.

The importance of dietary fibre for gut health

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With growing concerns surrounding Western style diet (WSD) consumption, it is important to fully understand how this diet can impact gut health. Gut microbiota is strongly shaped by diet and thusly the disconcertingly low fibre content in the WSD presents a major problem. The colon mucus layer, composed of mucus glycans, is a crucial line of defense in the gut, which prevents microbiota interaction with the colonic epithelium. However, in low fibre conditions members of the microbiota forage on mucus glycans to meet their nutritional needs, resulting in a damaged mucosal barrier and diminished gut health. It is therefore crucial to further our understanding of how mucosal barrier integrity is compromised and explore approaches to address this problem.

Here we investigate dietary fibre impact on microbiota composition and mucus function by utilising human-to-mouse faecal microbiota transplantation (FMT) of material from human volunteers consuming habitual or high fibre diets. We see that high fibre-driven microbiota is sufficient to protect from the harmful effects of the WSD by rescuing the defective mucus growth rate normally observed in WSD-fed mice. Our findings also confirm the strong influence of diet on microbiota composition and activity, as well as the importance of dietary fibre, which recapitulates the shortcomings of the WSD.

Expanding our understanding of gut, microbiota and diet interactions is crucial for improving gastrointestinal health and may allow tailoring of therapeutic approaches for diseases such as obesity and Inflammatory Bowel Disease, while also promoting maintenance of gut health in the general population.

In-situ structural analysis of the chikungunya virus double-stranded RNA

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The chikungunya virus (CHIV) is a mosquito-borne virus belonging to the alphavirus genus. It is responsible for the chikungunya fever characterized by fever, rashes and crippling arthralgia with symptoms lasting up to several months. There are no treatments nor vaccines available. The CHIKV is a positive sense single-stranded RNA virus (+ssRNA) that is able, as a majority of +ssRNA viruses, to hijack the host cell membrane to replicate its genome. In the case of the CHIKV, it is the plasma membrane that is remodeled. This virus forms organelles called "spherules" in which its genome is replicated. In spherules, the genomic RNA is present as a double-stranded RNA (dsRNA) replication intermediate. In a previous study, using cryo-electron tomography, we could trace the dsRNA in tomograms and estimate that there is a single copy of dsRNA present in spherules. Little is known about the spatial organization and structure of the dsRNA in spherules. In this study, we investigated the conformations taken by the dsRNA within spherules using subtomogram averaging and study the packing of the dsRNA in these replication compartments using the traced dsRNA.

We found that the dsRNA occupies homogenously the volume of spherules and has a preferred orientation. Subtomogram averaging yielded five structures highlighting different conformations taken by the dsRNA. This study is the first reporting the organization of the CHIKV dsRNA in spherules and our methodology could be applied to the dsRNA replication intermediate in replication compartments of other +ssRNA viruses.

Metabolic cooperation in dual-species biofilms related to catheter associated urinary tract infections

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Catheter Associated Urinary Tract Infections (CAUTI) are prevalent chronic conditions that may contribute to morbidity and death. *Escherichia coli* and *Pseudomonas aeruginosa* are among the most common bacterial species causing CAUTI that frequently are isolated from patients. Dual species infections and interactions between these two species within biofilms are not well understood. The aim of the study is to understand metabolic relationships between the two species. Four pairs of *E. coli* and *P. aeruginosa*, previously co-isolated from patients with CAUTI, were used. *E. coli* K-12 and *P. aeruginosa* PA01 were used as control strains. Bacterial biofilms were cultured in two different culture media: artificial urine medium (AUM) — nutrient-limited medium, and Iso-Sensitest, Oxoid™ (ISO) — medium containing glucose and amino acids/peptones. The ratio of *E. coli* and *P. aeruginosa* cells within biofilm and metabolite profiles in dual-species biofilms in presence of different nutrients were measured. A sequential culture of *E. coli* and *P. aeruginosa* in the same media (with a filtering step) was done to analyse the preferred type of nutrients and potential cross-feeding in the clinical pairs. The number of viable bacterial cells (VBC) of each species within biofilms were assessed by standard microbiological methods. Biofilm metabolites were analysed by gas chromatography–mass spectrometry (GC–MS).

We show that the number of *E. coli* VBC was higher than those of *P. aeruginosa* in dual-species biofilms cultured in ISO. The opposite was observed in AUM. Glucose was the preferred nutrient for *E. coli* and organic acids were preferred by *P. aeruginosa*. GC–MS analysis showed that organic acids (*e.g.*, succinic acid) were produced by *E. coli* as a result of glucose consumption and consumed by *P. aeruginosa* during the sequential culture. We propose that differences in metabolism between bacterial species allow them to consume urine nutrients more efficiently, which may give them an advantage living together during CAUTI.

Insights into alphaviral genome capping - *A mechanistic study*

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Alphaviruses belong to a group of arthropod-borne, bipartite positive-sense single-stranded RNA viruses (+ssRNA) that are the cause of novel and reoccurring viral outbreaks. Upon release of their +ssRNA genome into the host cell the first ORF is directly translated into the polyprotein P1234, which is subsequently cleaved in a stepwise fashion into the four individual non-structural proteins (nsPs) 1-4. Together nsP1-4 make up the replication complex located at the plasma membrane and is responsible for the replication of the viral genome. Among the nsPs, nsP1 is responsible for (1) tethering the replication complex to the plasma membrane and (2) the 5' capping of the nascent viral RNA via its methyl- and guanylyl transferase activities. Interestingly, nsP1 forms a functional dodecamer when expressed under physiological conditions, whereas when expressed in bacterial cultures in is isolated as a monomer with low basal activity.

Here, I present biochemical studies investigating the role of multimerization of nsP1 has on its activity. Using a panel of antibody fragments, I was able to identify a single antibody fragment that can activate monomeric nsP1, thereby recapitulating the activity levels seen in the assembled dodecamer.

An alternative ATG12-ATG5-TECPR1 E3-like conjugation complex regulates unconventional LC3 lipidation at damaged membranes

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Lysosomes are membrane-bound acidic organelles that play an essential role in the degradation of macromolecules received from autophagic, endocytic or phagocytic pathways. Degradation is mediated by a family of lysosomal hydrolases capable of inducing acute cell death if inadvertently released into the cytosol. To protect against the deleterious effects of lysosomal membrane permeabilization, cells employ sophisticated response mechanisms to detect, repair, remove and replace damaged lysosomes. Small membrane lesions are detected and repaired by the Endosomal Sorting Complex Required for Transport (ESCRT) machinery while more extensively damaged lysosomes are cleared by a galectin-dependent selective macroautophagic pathway (lysophagy).

In this study, we identify Tectonin beta-propeller repeat-containing protein 1 (TECPR1) as a novel player in the cellular response to lysosomal membrane damage. TECPR1 is a lysosomal protein that has been implicated in autophagosome-lysosome fusion via interaction with the ATG12-ATG5 conjugate located on autophagosomal membranes. We report a sphingomyelin-dependent enrichment of TECPR1 at lysosomes in response to lysosomal membrane damage. Enrichment occurs upstream of the galectin-dependent lysophagy pathway and recruits the ATG12-ATG5 conjugate to regulate ATG16L1-independent LC3 lipidation at the damaged membrane.

These observations identify a novel function of TECPR1 as a member of an alternative E3-like conjugation complex assembled in response to membrane damage.

Molecular mechanisms of *Salmonella* invasion and persistence via *in vivo* transcriptomics

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Salmonella enterica serovar Typhimurium is an intracellular pathogen causing gastroenteritis and severe systemic disease in humans and animals. The molecular mechanism by which this pathogen can establish and maintain infection is not completely revealed and understood. To acquire more knowledge about the critical mechanisms of the infection we applied *in vivo* transcriptional profiling of bacteria isolated from infected mice, both during early colonization and at later more chronic stages. RNA-seq of bacteria in mouse organs is a challenging task that requires significant optimizations of both protocols for bacterial RNA enrichment and further bioinformatic analyses of the samples that contain mice RNA and also the microbiota in certain organs at later time points.

Deep RNA sequencing was performed on several different tissue and cecum content samples from acute and chronic mice infection models. Optimizations were carried out for different aspects of the project regarding wet and dry lab procedures. Ribosomal RNA depletion was adjusted using a recently available rRNA depletion kit, we could improve enrichments of bacterial mRNA from infected mice organs, gaining better resolution of the transcriptomics. Data analyses were performed by several different methods overcoming the challenge of low coverage output from late time points and zero-inflated distribution of gene counts. We used imputation before applying canonical transcriptomics analyses methods and we also adapted single cell data analyses methods. This allowed us to reveal biology from the data, such as high expression of invasion related genes contributing to flagellar motility, SPI1, SPI4 pathways in cecal associated bacteria during early time points of infection. A different pattern was seen at late time points, where for example, the gene encoding the global regulator Fnr, which is important for survival in macrophages and anaerobic metabolism was highly expressed. There were also additional genes of potential interest for further investigation, among those genes can be found those that are linked to ribosome hibernation.

Overall, this study is expected to provide knowledge of genes and metabolic pathways in *Salmonella* that are essential for establishment and maintenance of infection. This knowledge would aid in identification of potential targets for future antimicrobials.

Western-style diet effect on host antimicrobial peptide expression and microbiota composition along the small intestine

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Secreted host antimicrobial peptides (AMPs) continuously protect the epithelium from the trillions of microbes residing in the intestine. While diet is considered the most substantial factor modulating microbiota composition, different dietary factors can alter the expression of AMPs, and thus, the individual contribution of AMPs and diet on microbiota composition remains unknown.

This study aimed to investigate the contribution of AMPs and diet on the mouse small intestinal microbiota composition, and explored the interplay between diet, AMPs, microbiota, and host metabolism. Mice lacking the matrix metalloproteinase 7 (MMP7), an enzyme that cleaves and activates intestinal α-defensins, were fed for 8 weeks a chow or a Western style diet (WSD), a diet rich in carbohydrates and fat but low on dietary fiber content. The composition of the small intestinal microbiota and absolute expression of several small intestinal AMPs was investigated at the lumen and at the mucosa. The overall microbiota composition was more affected by diet composition than by AMPs. However, AMPs selectively controlled the levels of specific genera at the ileal mucosa. The AMP expression in the ileum showed a clear clustering according to diet and genotype in a principal component analysis. A worsening in metabolic parameters, especially glucose, was observed in the mice lacking active α-defensins.

Taken together, diet had the strongest impact on small intestinal microbiota composition whereas AMPs had a more fine-tuning function of specific genera at the mucosa. In addition, our results highlight the protective effects of AMPs against metabolic dysfunction during WSD intake.

Peptidoglycan recycling mediated by an ABC transporter in the plant pathogen *Agrobacterium tumefaciens*

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During growth and division, the bacterial cell wall peptidoglycan (PG) is remodelled, resulting in the liberation of PG muropeptides which are typically reinternalized and recycled. Bacteria belonging to the Rhizobiales and Rhodobacterales orders of the Alphaproteobacteria lack the muropeptide transporter AmpG, despite having other key PG recycling enzymes. Here, we show that an alternative transporter, YejBEF-YepA, takes over this role in the Rhizobiales phytopathogen *Agrobacterium tumefaciens*. Muropeptide import by YejBEF-YepA governs expression of the β-lactamase AmpC in *A. tumefaciens*, contributing to β-lactam resistance.

However, we show that the absence of YejBEF-YepA causes severe cell wall defects that go far beyond lowered AmpC activity. Thus, contrary to previously established Gram-negative models, PG recycling is vital for cell wall integrity in *A. tumefaciens*. YepA is widespread in the Rhizobiales and Rhodobacterales, suggesting that YejBEF-YepA-mediated PG recycling could represent an important but overlooked aspect of cell wall biology in these bacteria.

Structural insight into filament proteins found in vesicles of enterovirus-infected cells and their involvement in enterovirus egress

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Enterovirus are small non-enveloped virus with a positive sense single stranded RNA genome. They can cause various diseases like poliomyelitis or myocarditis, respectively caused by Poliovirus and Coxsackies B3 virus. During infection, enteroviruses are known to reshape cytoplasmic membranes for their replication. Recent work done in our team through cryo-electron tomography highlighted that poliovirus assembles on replication membranes and that virus-induced autophagy strongly selects for autophagosomes carrying RNA loaded virions over other types of cargoes. Among these cargoes, some contained tightly packed bundles of protein filaments which were identified as potential decorated F-actin, suggesting a potential role of actin in the shaping of phagophores during enterovirus infection.

Thus, our aim is to confirm the nature of these filaments by acquiring a better resolved structure through cryo-electron tomography and subtomogram averaging. Moreover, autophagosomes from poliovirus infected cells will be isolated in order to do proteomic analysis of their content. Preliminary data have also highlighted three actin binding partners having an impact on viral release, thus, we will also perform live cell imaging and cryo-electron tomography studies to understand how these actin binding partners influence viral release.

Tantalosin impaires the assembly of ESCRT complex with a normal topology

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The Endosomal Sorting Complexes Required for Transport (ESCRTs) are a conserved protein machinery that plays a critical role in membrane remodeling processes such as the endosomal intralumenal vesicle (ILV) formation, enveloped virus budding, membrane repair, nuclear envelope closure, and cytokinetic abscission. ESCRT-III proteins can assemble into different structures, which make it challenging to dissect the function. In this study, by a high-content screening we identified a pseudo-natural product, termed Tantalosin (Tant), that modulates autophagy. We combined Proteome Integral Solubility Alteration (PISA) assay, cell biology method, Microscale thermophoresis (MST), electron microscopy and Hydrogen Deuterium Exchange mass spectrometry (HDX-MS) to identify the target of the compound and characterize the mode of action. Target identification and engagement suggested that the compound specifically targets increased sodium tolerance 1(IST1), a component of ESCRT-III complex.

Tantalosin was employed as a promising chemical genetic tool that perturbs the ESCRT complex with a normal topology, which enables to dissect different functions of ESCRT-III complex in cellular membrane remodeling processes.

Role of binding avidity and membrane complexity in the attachment of SARS-CoV-2 variants

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SARS-CoV-2 still challenges the healthcare systems of many countries despite unprecedented government policies and a pervasive vaccination campaign. This can be largely attributed to the emergence of variants showing an increasing number of mutations. The highest density of mutation is observed in the spike glycoprotein (S) which is responsible for primary receptor, ACE2, engagement and membrane fusion. This suggests that the virus is rapidly evolving to optimize the interactions leading to viral entry. Differences in the affinity between S and ACE2 have been reported among variants of concern in several studies. However, ACE2 affinity poorly correlates with the increased fitness of recent variants observed in infection assays and it ignores the complex environment the virus encounters at the cell membrane and the role of possible coreceptors.

Our study aims to bridge the gap between molecular and cellular observations by addressing the influence of bond multivalency and the complex plasma membrane in the modulation of the virion binding kinetics. To do so, we employ an *in-vitro* model system that combines native supported lipid bilayers (nSLBs) derived from lung epithelial cells and single particle tracking of fluorescent virion-mimics. nSLBs incorporate cell-membrane material into synthetic lipids, preserving the composition complexity of the native membrane, while as virion-mimic we developed liposomes decorated with soluble S. These particles resemble the virion in shape and size but allow high sample purity, control of S concentration, direct comparison between commercially available S variants, and handling in BSL2 conditions.

The characterization of the liposomes' interaction with the nSLB yields a comprehensive analysis of the effect of mutations on the viral attachment process. Our results show significant enhancement in the binding affinity of the most recent Omicron variant when compared to Delta and the original Wuhan strand. Given no such increase is observed in the literature for binding to ACE2 alone, the behavior might be attributed to a shift towards more efficient use of coreceptors. This exciting prospect is being currently investigated by observing the variation in the affinity between S and possible coreceptors, e.g., heparan sulfate.

Our work ultimately shines a light on the attachment process used by more recent SARS-CoV-2 variants, and, in general, on how viruses can adapt their entry strategy over strong selective pressure.

Discovery of novel anti-chlamydial compounds through a multi-strategy screening approach

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The obligate intracellular bacterium *Chlamydia trachomatis* is responsible for over 120 million urogenital infections per year. These can cause adverse pregnancy outcomes and infertility and may increase the risk for cervical and ovarian cancers. Infections with *Chlamydia* are usually effectively treated with azithromycin or doxycycline, but these broad-spectrum antibiotics will inevitably disrupt the patient's natural microbiota and select for antibiotic resistance not only in *Chlamydia* but also in off-target bacterial populations. The availability of more specific anti-chlamydial medicines would thus benefit patient health and aid antibiotic stewardship.

To this end, we developed a multiple-readout screening assay, based on fluorescence measurements and high-content imaging, and screened a chemically diverse library of 36,785 small molecules for compounds that inhibit *Chlamydia* growth. To complement this approach, we also combined the results of the screen with literature data to develop a machine-learning-based model allowing virtual screening of millions of additional compounds. Overall, we thereby identified a set of compounds that do not resemble known antibiotics but display potent anti-chlamydial activity, with some showing half-maximal bacterial growth inhibition (IC_{50}) at sub-micromolar concentrations. Crucially, the compounds are non-toxic to mammalian host cells and do not inhibit representative bacterial species from the gut microbiota.

Currently, we are conducting in-depth studies of the most promising compounds to reveal their mode of action. Ultimately, these efforts could generate novel therapeutics or tool compounds to help treat or study this highly prevalent intracellular pathogen.

Characterization of a novel penicillin-binding protein in *Vibrio cholerae*

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Penicillin-binding proteins (PBPs) are membrane-associated proteins that catalyze the synthesis of peptidoglycan (PG), the polymer that constitutes the bacterial cell wall. Thanks to a genome wide high-throughput screening of PG composition in *Vibrio cholerae*, we were able to identify several novel genes that could play a potential role in PG homeostasis. Among these, we found a gene with locus tag *vc1321,* which was previously annotated as a hypothetical protein but showed functional and sequence homology to the other high-molecular weight PBPs in *V. cholerae*. We discovered that this unique PBP, named PBP1V (after Pennicilin-binding protein 1 in Vibrio), helps *V. cholerae* survive in low-salt conditions and overall contributes to outer membrane stability, as its deletion mutant shows a dysregulation on the lipopolysaccharide (LPS) layer.

Furthermore, through proteomic studies we have also uncovered a potential role of this protein in virulence since the deletion mutant is altered in the ToxRS virulence system and related outer-membrane proteins. PBP1V is conserved in different *Gammaproteobacteria* species, suggesting its evolutionary and biological importance. Further characterization of PBP1V might uncover novel mechanisms of how PG homeostasis and PBPs can affect central systems in bacteria such as virulence or envelope stability, as well as potentially new therapeutic targets in infection.

Nematocida displodere mechanosensitive ion channel of small conductance 2 assembles into a unique six-channel super-structure *in vitro*

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Mechanosensitive ion channels play an essential role in reacting to environmental signals and sustaining cell integrity by facilitating ion flux across membranes. For obligate intracellular pathogens like microsporidia, adapting to changes in host environment is crucial for survival and propagation. Despite representing a eukaryote of extreme genome reduction, microsporidia have expanded the gene family of mechanosensitive ion channels of small conductance (MscS) through repeated gene duplication and horizontal gene transfer. At least five copies of MscS are present in each to-date characterized microsporidian genome: One subfamily related to eukaryotic MscS and the other of bacterial origin. A single copy of the bacterially derived mechanosensitive ion channel of small conductance 2 (MscS2) is highly conserved amongst all microsporidian species sequenced to date. However, compared to its bacterial counterpart it is extremely reduced and it is unclear if MscS2 forms a channel protein and if so, what role it plays in microsporidia.

Here, we investigated the cryo-electron microscopy structure of MscS2 from *Nematocida displodere*, an intracellular pathogen of *Caenorhabditis elegans*. We purified MscS2 and used size exclusion chromatography, negative-stain transmission electron microscopy, cryo-electron microscopy, and mass photometry to analyze size and structure.

Nematocida displodere MscS2 assembles into a unique superstructure *in vitro* with six heptameric MscS2 complexes interacting through their transmembrane domains. Individual MscS2 channels are oriented in a heterogeneous manner to one another, resembling an asymmetric, flexible six-way cross joint. This highly unusual assembly provides a novel basis to design oligomers that interact through hydrophobic interfaces.

Poster #48

Umeå Postdoc Society: By postdocs, for postdocs

Umeå Postdoc Society[a]

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The Umeå Postdoc Society (UPS) is a volunteer organization founded in 2020 to represent the diverse community of postdocs in Umeå, both from the Umeå University and the Swedish University of Agricultural Sciences (SLU). Its goal is to promote networking and exchange of ideas to enhance the postdoc experience of young researchers in Umeå.

With support from some departments at the University, UPS organizes a variety of events to build camaraderie, improve soft skills, and provide a platform for overall professional development. From social events like postdoc retreat, pub nights, and hikes, to career development programs like Career Path Seminars, postdoc talks, and workshops, UPS constantly strives to make this crucial stage in every young scientist's career ever so wholesome. The society is mentored by an Advisory Board of principal investigators from across the University and receives active support from the Research Support and Collaboration Office to prepare the postdocs for the next frontier.

UPS recently joined the 'Horizontal Connection' group at the local level, collaborating with different doctoral schools and centers, and is also a part of the Swedish National Postdoc Association (SNPA) at the national level.

The society is structured into various autonomous offices run entirely by postdocs, and therefore, is always looking to diversify connections and find more postdocs to join as active members. Our efforts are planting the seeds for development of a progressive research environment in Umeå that will not only nurture the on-campus scientific talent but will also attract highly motivated postdocs to Umeå.

Poster #49

National Bioinformatics Infrastructure Sweden (NBIS)

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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 organization 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.

We provide:
• Weekly online drop-in sessions, Tuesdays at 14:00; http://meet.nbis.se/dropin. Join to discuss study design, data analysis or other bioinformatics-related questions.
• Free consultation meetings.
• Hands-on project support, ranging from assistance with smaller tasks to long-term engagement.
• Free, extensive hands-on support to a limited set of projects selected in a peer review process (enabled by a grant from Knut and Alice Wallenberg Foundation).
• Tools, data management, systems development and guidelines for the life science community.
• Introductory and advanced training events, such as workshops in RNAseq data analysis, epigenomics data analysis, tools for reproducible research, python programming, and many other bioinformatics related topics.
• The Swedish Bioinformatics Advisory program - A mentorship program for PhD students interested in guidance from a bioinformatics expert.

For more information, please visit https://nbis.se or email jeanette.tangrot@umu.se

Poster #50

Umeå Hypoxia Research Facility (UHRF)

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While oxygen is essential for survival of most organisms, it can also be toxic for others. Thus, oxygen concentrations required by different organisms and cell types can vary significantly. In this line, hypoxia is a key regulatory signal inducing cellular responses including changes in microbiota diversity, cancer progression, brain aging, etc. Thus, to generate meaningful data, research conducted under controlled oxygen concentrations is imperative.

The Umeå Hypoxic Research Facility (UHRF provides a solution to this potential issue by allowing researchers to conduct their experiments under the exact oxygen concentrations that their model organisms or cell types face *in vivo*.

(UHRF) is a new research space at Umeå University launched in 2020 with the support of Kempe foundation. The main purpose of UHRF is to promote research excellence by supporting projects that require environments with highly controlled oxygen concentrations.

https://www.umu.se/en/research/infrastructure/umea-hypoxic-research-facility/

List of Participants

(*) Discussion group leaders

Last name	First name	Affiliation	Discussion group #
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Ahmad	Irfan	Molecular Biology	3
Andersson	Agneta	Ecology and Environmental Science	10
Arnqvist	Anna	Medical Biochemistry and Biophysics	**5 (*)**
Avican	Ummehan	Karolinska Institutet	2
Avican	Kemal	Molecular Biology	6
Avila-Cariño	Javier	Molecular Biology	8
Bae	Jiseon	Chemistry	7
Bala	Anju	Molecular Biology	1
Baland	Elisabeth	Chemistry / MIMS	4
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Björk	Glenn	Molecular Biology	6
Björklund	Stefan	Medical Biochemistry and Biophysics	**9 (*)**
Bodra	Nandita	Chemistry	10
Bougro	Léni	Molecular Biology	7
Bueno	Emilio	Molecular Biology	10
Bushell	Ellen	Molecular Biology	**7 (*)**
Carlson	Lars-Anders	Medical Biochemistry and Biophysics	9
Cava	Felipe	Molecular Biology	2
Chookajorn	Thanat	Molecular Biology	4
Cisneros	David	Molecular Biology	5
Corkery	Dale	Chemistry	3
Dahmane	Selma	Umeå University	10
Danskog	Katarina	Clinical Microbiology	5
Debiasi Anders	Gianluca	Medical Biochemistry and Biophysics	3
Doruk	Tugrul	Molecular Biology	5
Dörr	Tobias	Cornell University, USA	
El Arbi	Nabila	Plant Physiology	4
Elofsson	Mikael	Chemistry	8
Eriksson	Karolina	Ecology and Environmental Science	10
Erttmann	Saskia	Molecular Biology	2
Feeney	Rachel	Molecular Biology / MIMS	8
Fischer	Dominik	Molecular Biology / MIMS	5
Forró	Barbara	MIMS, UCMR	5
Francis	Matthew	Molecular Biology	**1 (*)**
Frisan	Teresa	Molecular Biology	
Giacoboni	Jessica	Chemistry	7
Gilmore	Michael	Molecular Biology	4
Gomez Sawicki	Filip	Molecular Biology	2
Guest	Tom	Molecular Biology	6

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Last name	First name	Affiliation	Discussion group #
Gylfe	Åsa	Clinical Microbiology	3
Hasanzada	Narmin	Molecular Biology	8
Henriksson	Johan	Molecular Biology / MIMS	6
Herzog	Laura	Chemistry	7
Hollopp	Isa	Chemistry	10
Holmberg	Sandra	Molecular Biology	8
Honkala	Emma	Clinical Microbiology	2
Jachmann	Lana	Molecular Biology	7
Jaiman	Deepika	Chemistry	6
Knyazeva	Anastasia	Chemistry	4
Kopeckova	Monika	Clinical Microbiology	3
Kossenko	Elisabeth	MIMS	1
Kumar	Pravin	Clinical Microbiology	9
Lambert	Clara	Molecular Biology	10
Lamy	Anaïs	Umeå Postdoc Society	1
Lappalainen	Amanda	Clinical Microbiology	4
Laurent	Timothée	Medical Biochemistry and Biophysics	9
Lee	Natuschka	Ecology and Environmental Science (but soon moving to SLU)	1
Lehotai	Nóra	MIMS	7
Li	Shuang	Chemistry	2
Li	Lingyu	Molecular Biology	3
Lindgren	Marie	Clinical Microbiology	2
Lindgren	Lena	Clinical Microbiology	5
Lindholm	Mark	Odontology	3
Liu	Bokai	Applied Physics and Electronics	8
LIU	LIFENG	Clinical Microbiology	8
López Chiloeches	María	Molecular Biology	6
Lu	Qiongxuan	UCMM	1
Lundmark	Richard	Integrative Medical Biology	3
Maccaferri	Nicolò	Physics	**10 (*)**
Masucci	Maria Grazia	CMB, Karolinska Institutet	
Mateus	André	Chemistry	**6 (*)**
Mir-Sanchis	Ignacio	Medical Biochemistry and Biophysics	7
Mishra	Maya	UPSC / Ecology and Environmental Science	6
MOREJON GARCIA	PATRICIA	Chemistry	9
Nadeem	Aftab	Molecular Biology	2
Nautiyal	Manesh	Chemistry	4
Nazemroaya Sedeh	Jasmin	Clinical Microbiology	2
Nieckarz	Marta	Molecular Biology	1
Näsman	Moa	Molecular Biology	7
Oscarsson	Jan	Odontology	**2 (*)**
Pace	Hudson	Clinical Microbiology	1
Pérez	Lucía	MIMS / Molecular Biology, Chemistry	6
Persson	Karina	Chemistry	1

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Last name	First name	Affiliation	Discussion group #
Peters	Marie Berit Akpiroro	MIMS / Clinical Microbiology	5
Pinedo	Victor	Molecular Biology	3
Prasad	Jyoti	UPSC	9
Prasoodanan	Vishnu	Molecular Biology	5
Puertolas Balint	Fabiola	MIMS / Molecular Biology	8
Puigefabregas Sieso	Alba	Ecology and Environmental Science	3
Qiao	Cuncun	Medical Biochemistry and Biophysics	4
Rafie	Karim	Medical Biochemistry and Biophysics	9
Ramstedt	Madeleine	Chemistry	**8 (*)**
Rey Varela	Diego	Molecular Biology	9
Robinson	Nicholas	Ecology and Environmental Science	1
Rodriguez Buitrago	Jhon Alexander	Chemistry	6
Rosendal	Ebba	Clinical Microbiology	6
Sandblad	Linda	Chemistry	4
Schneider	Ben	Ecology and Environmental Science	1
Schneider	Ben Silvester	Molecular Biology / Ecology and Environmental Science	3
Seddik	Noha	Molecular Biology	1
Seier	Kerstin	Clinical Microbiology	3
Selvarayan Vaz	Caraline	Ecology and Environmental Science	9
Shankar	Kasturika	Medical Biochemistry and Biophysics	3
Sharma	Atin	MIMS / Molecular Biology	6
Sharma	Himanshu	MIMS	10
Shevtsova	Anna	KBC	
Shingler	Victoria	Molecular Biology	2
Singh	Bina Kumari	Medical Biochemistry and Biophysics	1
Sixt	Barbara Susanne	Molecular Biology	10
Sokol	Dmytro	Chemistry	5
Song	Tianyan	Clinical Microbiology	8
Sorin	Marie	Medical Biochemistry and Biophysics	7
Stigbrand	Torgny	Clinical Microbiology	5
Strömberg	Nicklas	Odontology	9
Söderbergh	Ingrid	Clinical Microbiology / UCMR	
Thorsteinsson	Konrad	Clinical Microbiology	9
Toh	Eric	Molecular Biology	8
Torrens	Gabriel	Molecular biology	9
Tronnet	Sophie	Molecular Biology / MIMS	6
Tångrot	Jeanette	NBIS / Molecular Biology	7
Uhlin	Bernt Eric	Molecular Biology	1
Ullah	Nadeem	Clinical Microbiology	4
Urban	Constantin	Clinical Microbiology	5
Verbeek	Sarah	Clinical Microbiology	10
VERMA	ASHISH	Ecology and Environmental Science	10
Vikström	Linnea	Clinical Microbiology	8
Wadell	Göran	Clinical Microbiology	5

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Last name	First name	Affiliation	Discussion group #
Wai	Sun Nyunt	Molecular Biology	**3(*)**
Waititu	Joram	Molecular Biology	6
Wegler	Christine	Molecular Biology	7
Wennemo	Alfred	MIMS	5
Wikner	Johan	Ecology and Environmental Science	8
Wolf-Watz	Magnus	Chemistry	**4 (*)**
Wongkuna	Supapit	Molecular Biology	2
Wu	Yaowen	Chemistry	
Yabrag	Abdelbasset	Molecular Biology	4
Yalamanchili	Sampath Kumar	Chemistry	9
ZHANG	JUN	Chemistry	10
Zhu	Shaochun	Chemistry	7
Zlatkov	Nikola	Molecular Biology	2
Ölander	Magnus	Molecular Biology	2

Group discussions

The group discussion takes place **15:30-17:00** in the **Universum restaurant**. All participants of the conference have been divided in ten randomly mixed groups with appr. 14-15 persons in each group. The tables are marked with the group numbers, look out for yours (presented in the participant list and on the name badges).

The discussions are led by a designated PI (marked with (*) in the participant list). He/she is provided with ten questions. The group can choose which questions to address, and also address other issues that the group would like to discuss. The discussion leader takes notes and makes a summary. All summaries from all groups will be collected and distributed after the conference. In time before 17:00, the group discussion ends, and the participants gather in Aula Nordica for the second keynote lecture.

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