UCMR DAY 2024

18 January 2024, Aula Nordica

Programme - Abstracts - Posters - Participants

UCMR

Umeå Centre for Microbial Research

UMEÅ UNIVERSITY

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

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Layout: Anna Shevtsova

Welcome to the 15th UCMR Day 2024!

The UCMR Day is a one-day event full of diverse activities, where the entire community interested in microbial research and infectious diseases comes together to present their projects and interests, forge new interactions and collaborations, and be inspired to enter less familiar territories and make them bloom in unexpected directions. UCMR is a fantastic community where nothing is impossible, with research fields that span from molecules to treatment of patients, passing via the study of subcellular domains, microbial cells, and host-microbe interaction: from micro to macro.

The programme offers inspiring keynote lectures, short talks, elevator pitches, and poster presentations, but, foremost, it is an excellent opportunity for networking and initiating multidisciplinary collaborations.

Special for this year, we have an exciting panel discussion to reflect on UCMR from the past to the future, a moment to reflect on what UCMR has meant and will mean for all of us.

Panel: Bernt Eric Uhlin, Carl-Henrik Heldin, Alice Kempe, Yaowen Wu and Fredrik Almqvist

Moderator: Jennie Ekbeck

Keynote speakers are Peijun Zhang, Professor of structural biology, University of Oxford: *Visualizing virus infection in situ by cryoET*, and Maria Lerm, Professor in medical microbiology, Linköping University: *It is in your DNA – How previous exposures to infections are reflected in DNA methylation patterns*.

Remember, UCMR is all of us, and it is we who can shape it into the best melting pot for a vibrant and unique microbial research environment!

Teresa Frisan, *on behalf of the scientific organising committee:*

André Mateus, Department of Chemistry

Annasara Lenman, Department of Clinical Microbiology

Constantin Urban, Department of Clinical Microbiology

Fredrik Almqvist, Department of Chemistry

Ingrid Söderbergh, research coordinator at UCMR

Max Renner, Department of Chemistry

Oliva Wesula Lwande, Department of Clinical Microbiology

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

Yaowen Wu, Director of UCMR, Department of Chemistry

Programme

8:20 – 8:50 **REGISTRATION and MOUNTING OF POSTERS**

8:50 – 8:55 **WELCOME!**

Patrik Danielson, Dean at the Faculty of Medicine, Umeå University

8:55 – 9:15 **UCMR UPDATE**

Yaowen Wu, Director of UCMR

9:15 – 10:00 **KEYNOTE LECTURE 1**

Chair: Max Renner

Visualizing virus infection *in situ* by cryoET

Peijun Zhang, Professor of structural biology, University of Oxford

10:00 – 10:30 *Coffee break*

10:30 – 11:10 **SHORT TALKS 1** *(10 minutes talks)*

Chair: André Mateus

- **Host response to Langat virus infection at the blood-cerebral spinal fluid interface**

  Amanda Lappalainen, Department of Clinical Microbiology and Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå University

- **Regulation of morphological features and metabolic adaptations by the prokaryotic maintenance respiration share**

  Ashish Verma, Department of Ecology and Environmental Science and Umeå Marine Sciences Centre, Umeå University

- **The role of membrane complexity and heparan sulfate in the early entry stages of SARS-CoV-2 variants**

  Dario V. Conca, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine (WCMM), Umeå Centre for Microbial Research (UCMR), Umeå University

(see also **Poster #27**)

11:10 – 11:20 *Stretch your legs*

11:20 – 12:00 **SHORT TALKS 2**

- **Intravenous immunoglobin therapy for COVID-19 in immunocompromised patients: a retrospective cohort study**

  Johan Rasmuson, Department of Clinical Microbiology, Umeå University

- **A distinctive family of L,D-transpeptidases catalyzing L-Ala-mDAP crosslinks in Alpha and Betaproteobacteria**

Gabriel Torrens, Department of Molecular Biology and Laboratory for Molecular Infection Medicine Sweden, Umeå Centre for Microbial Research (UCMR), SciLifeLab, Umeå University

- **Co-infection of HAdV-F41 and AAV-2 as a possible aetiology behind acute hepatitis of unknown origin in children**

  Elin Hahlin, Department of Clinical Microbiology, Umeå University

12:00 – 13:00 *LUNCH*

13:00 – 13:30 **ELEVATOR PITCHES** *(3 minutes talks)*

Chair: Annasara Lenman

Poster #1 **Interaction of neutrophils with breast cancer cells and fibroblast in a co-culture model**

Anna Thunström Salzer, Department of Clinical Microbiology, Umeå University

Poster #2 **Breaking Barriers: A first glimpse of a Gram-positive type 4 Secretion system and a characterization of its cell-wall remodeling enzyme**

Josy ter Beek, Department of Medical Biochemistry and Biophysics and Wallenberg Centre for Molecular Medicine, Umeå University

Poster #3 **Neutrophil extracellular traps as a driving force behind severe COVID-19**

Emelie Backman, Department of Clinical Microbiology and Umeå Centre for Microbial Research (UCMR), Umeå University

Poster #4 **Ribosome clustering and surface layer reorganization in the microsporidian host-invasion apparatus**

Himanshu Sharma, Department of Molecular Biology and Department of Medical Biochemistry and Biophysics, Laboratory for Molecular Infection Medicine Sweden (MIMS), Centre for Microbial Research (UCMR), Science for Life Laboratory, Umeå University

Poster #5 **Deciphering heterogeneous populations of infecting bacteria with a novel bacterial single-cell RNA-seq method**

Joram Kiriga Waititu, Department of Molecular Biology and Umeå Centre for Microbial Research (UCMR), Integrated Science Lab (IceLab), Umeå University

Poster #6 **High content screening of natural compounds against Chikungunya virus infection identifies a steroidal lactone withaferin A as an antiviral agent**

Kiran Bala Sharma, Regional Centre for Biotechnology, Faridabad, India, and Department of Medical Biochemistry and Biophysics, Umeå University

Programme

Poster #7	**A shield against the mutants: Effects of different vaccine regimens against novel SARS-CoV-2 variants on the antibody levels and survival of nursing home residents** Remigius Gröning, Department of Clinical Microbiology, Umeå University
Poster #8	**Chasing Better Drug-like Properties and Challenging Resistance Development By Increasing 3D Complexity of 2-Pyridone Antibiotics** Victor Hellgren, Department of Chemistry, Umeå University
Poster #9	**How infection with respiratory syncytial virus alters the mechanics of live Hep-2 cells** Sarah F. Verbeek, Department of Clinical Microbiology and Wallenberg Centre for Molecular Medicine, Umeå University
Poster #10	**The complex interplay between alphaviruses and cells: identification of host factors and viral evolution** Tessy A. H. Hick, Department of Clinical Microbiology and Umeå Centre for Microbial Research (UCMR), Wallenberg Centre for Molecular Medicine (WCMM), Umeå University
13:30 – 13:40	*Stretch your legs*
13:40 – 14:20	**SHORT TALKS 3**

- ***In vitro* reconstitution reveals membrane clustering and double-stranded RNA recruitment by the enteroviral AAA+ ATPase 2C**

  Kasturika Shankar, Department. of Medical Biochemistry and Biophysics and Wallenberg Centre for Molecular Medicine, Molecular Infection Medicine Sweden, Umeå University

- **Assessing the behavior of the genotoxin-producing *Salmonella enterica* in pro-carcinogenic mouse models**

  María López Chiloeches, Department of Molecular Biology and Umeå Centre for Microbial Research (UCMR), Umeå University

- **Simultaneous Membrane and RNA Binding by Tick-Borne Encephalitis Virus Capsid Protein**

  (see also **Poster #28**)

  Pulkkinen L. I. A., Department of Medical and Translational Biology, Umeå University

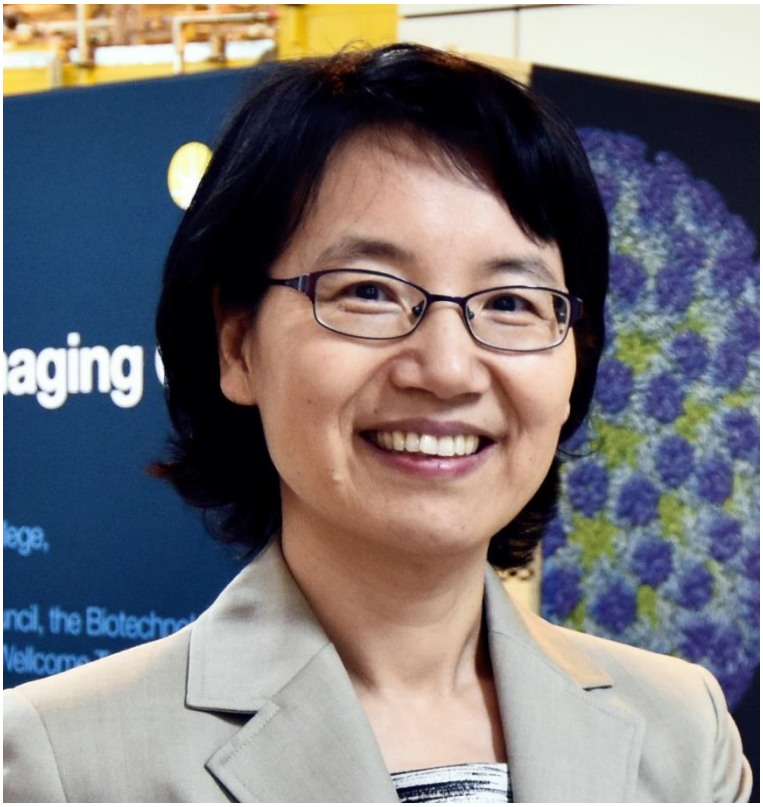
14:20 – 16:00	**POSTER WALKS and Coffee and Cake** Six groups led by PIs for organised walks (ca 35 min) *Posters are shown in Brashörnan* *Coffee and cake are served in the Lounge*

Programme

16:00 – 16:45	**PANEL DISCUSSION – UCMR 20 YEARS** *Moderator:* **Jennie Ekbeck**, former CEO of Umeå Biotech Incubator, Business coach LEAD *Panel members:* **Fredrik Almqvist**, Director of UCMR **Yaowen Wu**, Director of UCMR 2020-2023 **Bernt Eric Uhlin**, Director of UCMR 2008-2019 **Carl-Henrik Heldin**, Chair of UCMR Executive Board 2009-2021 **Alice Kempe**, Chair at Kempestiftelserna
16:45 – 17:00	**Post your thoughts on UCMR!**
17:00 – 17:45	**KEYNOTE LECTURE 2** *Chair: Oliva Wesula Lwande* **It is in your DNA – How previous exposures to infections are reflected in DNA methylation patterns** **Maria Lerm, Professor in medical microbiology, Linköping University**
17:45 – 18:00	**CONCLUDING REMARKS and short talk/poster reward** *Chair: Teresa Frisan*
18:00 – 18:30	*Reception with mingle* *The reception takes place in the Lounge*
18:30 – 21:00	*DINNER* *The dinner takes place in the Universum restaurant*

Peijun Zhang

Professor of Structural Biology at the University of Oxford, UK, and the founding director of the UK National Electron Bio-imaging Centre at the Diamond Light Source



Title of the lecture:

"Visualizing virus infection *in situ* by cryoET"

Abstract: Retroviruses, such as human immunodeficiency virus 1 (HIV-1), contain mature capsids that enclose the viral RNA genome, enzymes, and accessory proteins. The assembly, maturation, and stability of the viral capsid are critical to the viral replication life cycle. Furthermore, the surface of the viral capsid serves as a primary interaction interface between the virus and the host cell, for both host defense proteins and virus dependence factors. We are developing cutting-edge cryoEM technologies that bring unprecedented resolution and enable in situ structures of large assemblies and complexes to decipher their underlying functional roles.

I will describe HIV-1 mature and immature capsid assemblies and interactions with host factor IP6 and cyclophilin A, highlighting unexpected novel interactions that are critical for these host factors to stabilize the HIV-1 capsid and prevent its pre-mature disassembly. I will also present our recent in situ studies of SARS-COV-2 infection and COVID vaccine, as well as the architecture of native chromatin fibers in intact T-cells relevant to HIV-1 infection and integration.

Maria Lerm

Professor in Medical Microbiology, Linköping University, Sweden

Title of the lecture:

"It is in your DNA – How previous exposures to infections are reflected in DNA methylation patterns"

Abstract: The epigenetic information encoded in the DNA methylome is reflecting environmental exposures like genomic data reflects heredity. We have uncovered how infectious diseases causes unique imprints in DNA methylation patterns, allowing us to trace disease mechanisms and identify biomarkers. I will present our findings on DNA methylation changes during tuberculosis exposure, post-covid and neuroborreliosis and hope to inspire others to investigate the epigenome changes triggered by other infections.

Short talks

The best short talk will be given an award sponsored by UCMR and Agrisera.

Poster walks

The poster session takes place **14:20-16:00 in Brashörnan**. It starts with organised poster walks to enhance interactions between the attendees and the poster presenters. The posters have been divided 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 is led by two PIs, and one group spend 7 minutes on each poster, in total 4-5 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 up to 35 minutes.

After 35 minutes, all attendants can move freely in Brashörnan and view/discuss all the posters before the start of the next session.

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

	Poster walk group A	**Poster walk group B**	**Poster walk group C**
Panel members	**Kemal Avican** **Ya-Fang Mei**	**Madeleine Ramstedt** **Matthew Francis**	**Ellen Bushell** **Johan Wikner**
Poster #, Poster presenter	#P1, Anna Thunström Salzer #P2, Josy ter Beek #P3, Emelie Backman #P4, Himanshu Sharma	#P5, Joram Kiriga Waititu #P6, Kiran Bala Sharma #P7, Remigius Gröning #P8, Victor Hellgren	#P9, Sarah F. Verbeek #P10, Tessy A.H. Hick #P11, Dmitry Malyshev #P12, Fouzia Bano #P13, Girish Malagi

	Poster walk group D	**Poster walk group E**	**Poster walk group F**
Panel members	**Magnus Wolf-Watz** **Maria Fällman**	**Björn Schröder** **Elisabeth Sauer**	**Magnus Evander** **Sun Nyunt Wai**
Poster #, Poster presenter	#P14, Ashish Verma #P15, Katarina Danskog #P16, Leyden Fernandez #P17, Malgorzata Graul #P18, Marie Berit Akpiroro Peters	#P19, Marie N. Sorin #P20, Marta Nieckarz #P21, Maud Mutsaers #P22, Oytun Sarigöz #P23, Thibault Duteil	#P24, Zeinab Razooqi #P25, Timir Baran Sil #P26, Ummehan Avican #P27, Dario V. Conca #P28, Lauri Pulkkinen

Poster List

Poster walk groups	Poster number	Elevator pitch	Presenter's name	Poster title	Research group
A	**#P1**	Yes	**Anna Thunström Salzer**	Interaction of neutrophils with breast cancer cells and fibroblast in a co-culture model	Constantin Urban
A	**#P2**	Yes	**Josy ter Beek**	Breaking Barriers: A first glimpse of a Gram-positive type 4 Secretion system and a characterization of its cell-wall remodeling enzyme	Ronnie Berntsson
A	**#P3**	Yes	**Emelie Backman**	Neutrophil extracellular traps as a driving force behind severe COVID-19	Constantin Urban
A	**#P4**	Yes	**Himanshu Sharma**	Ribosome clustering and surface layer reorganization in the microsporidian host-invasion apparatus	Lars-Anders Carlson
B	**#P5**	Yes	**Joram Kiriga Waititu**	Deciphering heterogeneous populations of infecting bacteria with a novel bacterial single-cell RNA-seq method	Kemal Avican
B	**#P6**	Yes	**Kiran Bala Sharma**	High content screening of natural compounds against Chikungunya virus infection identifies a steroidal lactone withaferin A as an antiviral agent	Lars-Anders Carlson
B	**#P7**	Yes	**Remigius Gröning**	A shield against the mutants: Effects of different vaccine regimens against novel SARS-CoV-2 variants on the antibody levels and survival of nursing home residents	Mattias Forsell
B	**#P8**	Yes	**Victor Hellgren**	Chasing Better Drug-like Properties and Challenging Resistance Development By Increasing 3D Complexity of 2-Pyridone Antibiotics	Fredrik Almqvist
C	**#P9**	Yes	**Sarah F. Verbeek**	How infection with respiratory syncytial virus alters the mechanics of live Hep-2 cells	Marta Bally
C	**#P10**	Yes	**Tessy A.H. Hick**	The complex interplay between alphaviruses and cells: identification of host factors and viral evolution	Gisa Gerold, Magnus Evander
C	**#P11**		**Dmitry Malyshev**	Robust and highly sensitive detection of spore biomarker CaDPA using SERS	Magnus Andersson
C	**#P12**		**Fouzia Bano**	Sulfation patterns of heparan sulfates regulate the interactions of human	Marta Bally

Poster List

Poster walk groups	Poster number	Elevator pitch	Presenter's name	Poster title	Research group
				papillomavirus to glycosaminoglycan for entry	
C	**#P13**		**Girish Malagi**	Adaptive Immune Response to Repeated mRNA Vaccination in the Older Population	Mattias Forsell
D	**#P14**		**Ashish Verma**	Extensive prokaryotic maintenance respiration in the sea influenced by osmoregulation	Johan Wikner
D	**#P15**		**Katarina Danskog**	Cellular Receptors for Species D Human Adenovirus	Niklas Arnberg
D	**#P16**		**Leyden Fernandez**	Multimodal deep learning to predict protein function in bacteria	Maria Fällman
D	**#P17**		**Malgorzata Graul**	Comparison of entry efficiency of different species of *Filoviridae*-derived VLP	Marta Bally
D	**#P18**		**Marie Berit Akpiroro Peters**	Unveiling the function of NUP98 and Mediator Complex interactions in the Flavivirus life cycle	Marta Bally
E	**#P19**		**Marie N. Sorin**	Structural and mechanistic basis of key intracellular steps of enterovirus infection	Lars-Anders Carlson
E	**#P20**		**Marta Nieckarz**	Toward novel antibacterial targets: decoding the cell wall biology of *Salmonella* Typhimurium	Felipe Cava
E	**#P21**		**Maud Mutsaers**	Invasive Asian urban malaria mosquito *Anopheles stephensi* can result in re-emergence of O'nyong-nyong virus	Olivia Wesula Lwande
E	**#P22**		**Oytun Sarigöz**	Diatom biofilms induce the aggregation of clay and sand in sedimentary environments	Kemal Avican
E	**#P23**		**Thibault Duteil**	Diatom biofilms induce the aggregation of clay and sand in sedimentary environments	Madeleine Ramstedt
F	**#P24**		**Zeinab Razooqi**	*Aggregatibacter actinomycetemcomitans* and *Filifactor alocis* as Associated with Periodontal Attachment Loss in a Cohort of Ghanaian Adolescents	Anders Johansson
F	**#P25**		**Timir Baran Sil**	Characterization of UV-induced spectral and morphological changes in bacterial spores	Magnus Andersson

Poster List

Poster walk groups	Poster number	Elevator pitch	Presenter's name	Poster title	Research group
F	**#P26**		**Ummehan Avican**	*In Vivo* Transcriptomics of Chronic *Salmonella* Typhimurium Infection	Maria Fällman
F	**#P27**	Short talk	**Dario V. Conca**	The role of membrane complexity and heparan sulfate in the early entry stages of SARS-CoV-2 variants	Marta Bally
F	**#P28**	Short talk	**Lauri Pulkkinen**	Simultaneous Membrane and RNA Binding by Tick-Borne Encephalitis Virus Capsid Protein	Richard Lundmark

Interaction of neutrophils with breast cancer cells and fibroblast in a co-culture model

Anna Thunström Salzer[a], Samuel Skoluda[a], Constantin Urban[a]

[a]Department of Clinical Microbiology, Umeå University

Neutrophils are part of the innate immunity and they defend us against microbial infections by moving towards a gradient of chemokines to the site of infection. In the tissue they perform phagocytosis, produce reactive oxygen species and can be triggered to release neutrophil extracellular trap. Patients with cancer often show elevated levels of neutrophils in their blood count but the role that neutrophils play in the setting of cancer is still largely unknown. It has been found that communication between cancer cells and the fibroblasts surrounding them alters the metabolic interaction into a synergy that favors the cancer cells and make them grow at the cost of the fibroblasts. The cancer cells uses oxidative stress to reprogram the fibroblasts. We use a co-culture model with breast cancer cells and fibroblasts and add neutrophils from healthy donors to investigate the interaction between cancer cells, fibroblasts and neutrophils with the aim to understand if neutrophils are being activated and if they alter the interaction between cancer cells and fibroblasts. When we examined the supernatant from the co-culture with a multiplex we found a pro-inflammatory environment that is chemoattractant to neutrophils measured with a transwell system. When using sea horse to evaluate the metabolic profile of the cells we found that neutrophils alter the mitochondrial metabolism of the co-culture and the glycolysis of fibroblasts. Our findings contribute to the understanding of interaction between innate immunity and cancer which improve the possibility of finding new tools in the fight against cancer.

Breaking Barriers: A first glimpse of a Gram-positive type 4 Secretion system and a characterization of its cell-wall remodeling enzyme

Josy ter Beek, Wei-Sheng Sun, Krishna Chaitanya Bhattiprolu, Anaïs Lamy, Ronnie P-A Berntsson

Department of Medical Biochemistry and Biophysics, Umeå University, Sweden and Wallenberg Centre for Molecular Medicine

Type 4 Secretion Systems (T4SSs) are a main driver for the spread of antibiotic resistance genes and virulence factors in bacteria. T4SSs are versatile and megadalton sized complexes that facilitate the transfer of proteins and DNA from a donor bacterium to a recipient cell, which can be from a different species. Studies in the past decades have advanced our understanding of how these systems work in Gram-negative bacteria. However, so far little is known about their structure and function in Gram-positive bacteria, while these bacteria account for most hospital acquired infections. These bacteria also have a much thicker cell-wall barrier, that will have to be crossed by the DNA-protein substrate.

Our lab studies the T4SS that is encoded on the conjugative pCF10 plasmid from the commensal Gram-positive bacterium and opportunistic pathogen *Enterococcus faecalis*. By negative stain electron microscopy, we have obtained the first images of a Gram-positive T4SS channel. We have also characterized a hydrolase that is self-regulating to just make a local hole in the (cell-)wall.

Neutrophil extracellular traps as a driving force behind severe COVID-19

Emelie Backman[a], Remigius Gröning[a], Alicia Edin[b], Hinnerk Eilers[c], Anna Lange[d], Clas Ahlm[a], Mattias N.E. Forsell[a], Sara Cajander[d], Johan Normark[a], Constantin Urban[a]

[a]Department of Clinical Microbiology and Umeå Centre for Microbial Research (UCMR), Umeå University, Umeå, Sweden
[b]Department of Surgical and Perioperative Sciences, Umeå University, Umeå, Sweden
[c]Laboratory Medicine, Region Västerbotten, Norrland University Hospital, Umeå, Sweden
[d]Department of Infectious Diseases, Faculty of Medicine and Health, Örebro University, Örebro, Sweden

Severe cases of Coronavirus disease 2019 (COVID-19) are caused by a dysregulated immune response characterized by a inflammatory state that can lead to severe complications like thrombotic events and acute respiratory distress syndrome (ARDS). Neutrophil extracellular traps (NETs) are web-like structures of DNA, decorated with antimicrobial proteins, normally released to trap and clear microbial pathogens, and known contributors to both states. Several studies have implicated NETs in COVID-19-related pathology; however, information about affected patient groups and underlying mechanisms are not yet fully understood. In this study, we corroborate the presence of elevated plasma levels of the specific NET marker MPO-DNA in patients with severe COVID-19. Notably, we show that NET levels are specifically higher in plasma samples from men with severe COVID-19, as compared to samples from women in the same category. Neutrophils from healthy donors treated with plasma from patients with severe COVID-19 formed significantly more NETs indicating that factors in plasma contribute to excessive NET formation. A comprehensive cytokine analysis revealed that several pro-inflammatory cytokines are specifically upregulated in plasma from patients with severe COVID-19. Moreover, we identified important mediators of COVID-19-induced NET formation using inhibitors of reactive oxygen species (ROS), protein kinase B (Akt) and spleen tyrosine kinase (Syk) pathways which are all implicated in NET formation. Finally, treatment with recombinant severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) spike protein further increased NET formation indicating that increased numbers of immune complexes, via Fc receptors can trigger NET formation.

Ribosome clustering and surface layer reorganization in the microsporidian host-invasion apparatus

Himanshu Sharma[1,2,#], Nathan Jespersen[1], Kai Ehrenbolger[1,2], Lars-Anders Carlson[2], Jonas Barandun[1,#]

[1]Department of Molecular Biology, The Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå and Centre for Microbial Research (UCMR), Science for Life Laboratory, Umeå University, 90187 Umeå, Sweden.
[2]Department of Medical Biochemistry and Biophysics and The Laboratory for Molecular Infection Medicine Sweden (MIMS), Wallenberg Centre for Molecular Medicine, Umeå Centre for Microbial Research (UCMR), Umeå, University, 90187 Umeå, Sweden.

Microsporidia are a group of obligate intracellular parasites that infect hosts ranging from arthropods to nematodes to chordates. Microsporidia also exemplify extreme reductive evolution, wherein deletion of biochemical pathways and structural elements, otherwise considered essential, has produced the smallest known eukaryotic genomes, degenerated mitochondria, and a highly miniaturised cytoplasmic ribosome. In contrast to these drastic omissions, microsporidia have also evolved specialised mechanisms to precisely tune their cellular apparatus in nutrient-limiting conditions and for invading and hijacking host cell systems. Among these, a microsporidia-specific organelle, the polar tube, is particularly noteworthy. For host cell invasion, microsporidia translocate their entire cytoplasmic content through this thin, hollow superstructure, the polar tube. To achieve this, the polar tube transitions from a compact spring-like state inside latent spores to an unusually long needle like tube capable of long-range cargo delivery. The unique mechanical properties of the tube enable an explosive transition from compact to extended state and allow for so far unknown morphological changes during the rapid cellular cargo translocation process. Here I describe these unusual transitions of the polar tube using in-situ cryo-electron tomography to unravel the ultrastructural changes during cargo transport. We captured cargo-filled states with a unique ordered arrangement of clustered hibernating microsporidian ribosome and an empty post translocation state of the tube with a reduced diameter but a thicker wall. Collectively, our work provides comprehensive data on the novel hibernation strategies of microsporidia and host invasion mechanisms by unravelling the basis for the efficient transport of infectious cellular cargo.

Deciphering heterogeneous populations of infecting bacteria with a novel bacterial single-cell RNA-seq method

Joram Kiriga Waititu[1,2], Tugrul Doruk[1,2], Maria Fällman[1,2,3], Linas Mazutis[4], Johan Henriksson[1,2,3], Kemal Avican[1,2]

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[3]Department of Molecular Biology, The Laboratory for Molecular Infection Medicine Sweden (MIMS), Umeå, Sweden
[4]Institute of Biotechnology, Life Sciences Centre, Vilnius University, Vilnius, Lithuania.

Bacterial populations exhibit significant heterogeneity, allowing distinct cell subgroups to thrive in diverse environments. This environmental organized heterogeneity prompts bacteria to respond to dynamic changes through unique transcriptional programs, posing substantial challenges in assaying individual cells uniformly. Recent advancements in single-cell techniques, particularly single-cell RNA sequencing (scRNA-seq), offer a powerful means to unravel cellular heterogeneity during infections and discern subpopulation variations that influence individual cell outcomes. However, current bacterial scRNA-seq methods suffer from the low number of cells being sequenced and the low number of transcripts detected in each cell due to leakage of nucleic acids due to mechanical disruption of cell walls during permeabilization for subsequent enzymatic reactions. Therefore, we are developing a novel bacterial scRNA-seq method by using advantage of semi-permeable hydrogel microcapsules (SPCs), which allows encapsulation of bacterial cells by microfluidics device and multiple enzymatic reactions once the cells are lysed inside SPCs. The methodology involves fixing bacterial cells using paraformaldehyde (PFA) and encapsulating individual bacterial cells in SPCs for single-cell analysis. Subsequent steps within the SPCs encompass molecular biology procedures such as cell lysis, rRNA depletion, mRNA polyadenylation, cell barcoding, cDNA synthesis, and PCR, alongside split-pool barcoding techniques utilizing barcoded oligos specific to the bacterial cell. Following encapsulation, the SPCs undergo a series of processing steps, including breaking the capsules, demultiplexing the cells, and employing computational techniques to cluster cells based on differentially expressed genes. This comprehensive approach overcomes technical obstacles associated with sequencing bacterial cells, enabling the sequencing of over a hundred thousand cells. This provides valuable insights into the heterogeneity and dynamics of sub-populations. Taken together, this study provides valuable insights that offer a deeper understanding of the molecular mechanisms underlying bacterial heterogeneity during infection, thus opening advancements in our understanding of bacterial pathogenesis.

Keywords: Bacteria; single-cell RNA sequencing; SPCs; microfluidics

High content screening of natural compounds against Chikungunya virus infection identifies a steroidal lactone withaferin A as an antiviral agent

Kiran Bala Sharma[a,c*], Khashpatika Ganesh[a], Subramani Chandru[a], Sapna Sehrawat[a], Kanchan Bharadwaj[a], Ghanshyam Sharma[a], Shouri Kushal[a], Archana Rout[a], Mitul Srivastava[b], Pankaj Kumar Sahoo[a], Saurabh Chugh[b], Ramandeep Singh[b], Deepti Jain[a], Shailendra Asthana[b], Renu Wadhwa[d], Sudhanshu Vrati[a]

[a] Regional Centre for Biotechnology, Faridabad, India
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[d] National Institute of Advanced Industrial Science and Technology, Tsukuba, Japan

Chikungunya virus (CHIKV) is a mosquito-transmitted alphavirus, that has spread to more than 60 countries. CHIKV infection causes febrile illness, myalgia, and debilitating joint pain which in many patients advances to a painful chronic stage. The frequent outbreaks and large-scale epidemics of CHIKV across the world pose a significant public health burden, which substantiates the need for effective antiviral therapeutics. We performed a cellular imaging based-high content screening of natural compounds against CHIKV and identified withaferin A (WFA), a steroidal lactone isolated from the plant Withania somnifera, also known as Ashwagandha, as a potent antiviral agent. The results indicated that WFA restricted CHIKV infection at the early stage of replication by targeting CHIKV nsP2 protease activity. WFA was seen to mount inhibitory effect due its oxidizing property as the addition of reducing agents completely abolished the WFA mediated antiviral activity in cell culture system and reversed the WFA mediated protease inhibition in vitro. Furthermore, it was observed that WFA significantly reduced the morbidity caused by CHIKV in the C57/BL6 mouse model. This work demonstrates the potential of WFA as an antiviral against CHIKV.

A shield against the mutants: Effects of different vaccine regimens against novel SARS-CoV-2 variants on the antibody levels and survival of nursing home residents

Remigius Gröning[a*], Peter Sundström[a], Julia Wigren Byström[a], Anders Johansson[a], and Mattias Forsell[a]

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Vaccination against SARS-CoV-2 remains to be the most effective protection against COVID-19. However, individuals older than 80 years can exhibit weaker vaccine responses and thereby continue to be at risk for severe disease and death. Especially concerning are novel virus variants, against which older vaccine batches often show insufficient protection. Therefore, the Public Health Agency of Sweden has recommended administering booster doses every six months. Nevertheless, survival and infection numbers continue to vary between these individuals. For our study, blood samples were taken every three months after the first vaccine dose up until after the sixth dose from over 3000 adults living in nursing care homes in Sweden. Using multiplex serology, we tracked anti-S and -RBD IgG antibody levels, binding the original Wuhan wild type and different Omicron variants. In addition, we acquired demographic, infection, and survival data from the Public Health Agency of Sweden. Utilizing sophisticated statistics methods, we aim to detect differences in antibody levels, survival, and infections between doses, different vaccines (Moderna/Pfizer), batches (mono-/bivalent), and demographics. Our findings could help define the risk group in more detail and offer a basis for potential new vaccine guidelines.

Chasing Better Drug-like Properties and Challenging Resistance Development By Increasing 3D Complexity of 2-Pyridone Antibiotics

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In the 2013 report on the threats of antibiotic resistance in the US from the Centers of Disease Control and Prevention (CDC) it was estimated that around 2 million infections and 23,000 deaths were caused per year in the US by antibiotic resistant microorganisms. In the subsequent report from 2019, the number of infections had increased to 3 million and the number of deaths to 48,000 exemplifying the rapid increase in antibiotic resistant microorganisms. The topic is therefore a rising concern that not only threatens to make currently treatable bacterial infections untreatable, but also to complicate medical procedures such as implant surgery where antibiotics are routinely prescribed following the procedure to avoid infections in the area. The search for new antibiotics is therefore an important task and, in our lab, we're developing substances based on a thiazolino ring-fused 2-pyridone core that can be decorated in various ways to gain antibacterial properties. The our most recent generation of analogues have had promising inhibitory activities towards gram-positive bacteria but are limited by their physicochemical properties. This has been attributed to the inherent flatness of the core due to its sp2-rich nature. Our current objective is to diversify the core while retaining activity and move away from flat-land by introducing more sp3-hybridized carbon centers. We recently discovered that the main scaffold can efficiently undergo photoinduced [2+2] cycloaddition with styrenes which gave rise to a new generation of analogues that have a more complex 3D structure and also retains antibacterial activity.

How infection with respiratory syncytial virus alters the mechanics of live Hep-2 cells

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Respiratory syncytial virus (RSV) infection can result in fatal respiratory complications in infants and elderly. No effective antiviral treatment is available. RSV has been proposed to spread through formation of long filopodia stretching to uninfected cells, and by forming multinucleate cells (syncytia) through cell fusions. Preventing these processes could be a promising antiviral strategy. Lacking, however, is a time-resolved understanding of underlying mechanisms. In this work, we use atomic force microscopy (AFM) and force spectroscopy to investigate how the morphological and mechanical properties of live cells change during infection. In agreement with our hypothesis, preliminary data suggest that RSV-infection results in overall cell stiffening, presumably due to actin overproduction, while syncytia are softer than non-syncytial cells. We will continue to investigate how promising antiviral drug candidates alter observed property changes.

The complex interplay between alphaviruses and cells: identification of host factors and viral evolution

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The population at risk of mosquito-borne alphaviruses is increasing due to spread of mosquito vectors and adaptation of viruses to new environments. Alphaviruses like o'nyong'nyong virus (ONNV) cause outbreaks of arthritic disease that represent a significant social and economic burden. To predict and prepare for these outbreaks, understanding complex virus-host interactions is critical. In this two-pronged study, we aim to identify cellular proteins (host factors) involved in alphavirus infection and characterize viral genome mutations allowing alphaviruses to adapt to host factors. Some host factors are essential for virus infection (host dependency factors), whereas others limit infection (host restriction factors). We observed that the absence or presence of a host factor prevents infection of a clinical ONNV isolate in Lunet cells (a human liver cell line), but not of cell culture adapted ONNV strains. Fusion of ONNV non-permissive Lunet cells and permissive HEK293 cells resulted in infection, suggesting the absence of a host dependency factor in Lunet cells. In addition, RNA sequencing of serial passaged ONNV revealed adaptive mutations in the viral genome, allowing infection of Lunet cells. Current research is ongoing to identify these host and viral factors in more detail and thereby create a better understanding of alphavirus infection and evolution.

Poster #11 (Group C)

Robust and highly sensitive detection of spore biomarker CaDPA using SERS

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Spore forming bacteria are responsible for a vast number of infections and costly contaminations in agriculture, food industry, and healthcare. To minimize spread of spores and reduce damage, rapid field detection of spores is important. However, detection is difficult since spores are resistant to many of the bacterial disruption techniques used to bring out the biomarkers necessary for detection. Because of this, quick and effective spore disruption methods are desirable.

Here we show a highly sensitive method of detecting the biomarker CaDPA from bacterial spores using surface-enhanced Raman spectroscopy (SERS), a method utilizing laser light to identify chemical compounds. We release CaDPA from spores using sonication and collect the liquid fraction of the sample. Then, using golden nanorods optimized for SERS, we can detect CaDPA in our sample. We can dilute a sample to the level of detecting single-spore quantities of CaDPA. Crucially, we work directly with a biological spore suspension with minimal sample treatment, unlike typical SERS measurements which rely on highly purified samples or synthetized target chemicals.

Thus, we conclude that this method is a promising option for practical detection of spore contamination in a field setting.

Sulfation patterns of heparan sulfates regulate the interactions of human papillomavirus to glycosaminoglycan for entry

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Human papillomaviruses (HPVs) are small non-enveloped DNA viruses, several of which are responsible for anogenital cancers. At the initial stage of infection, HPV16, the best-studied and most prevalent cancer-causing type, interacts with heparan sulfates (HS). The interaction of HPV16 virions with HS is important for both the initial attachment to host cells and triggering a crucial conformational change in the viral capsid termed structural activation. In this context, the role of specific sulfation groups of HS in regulating these interactions is currently unknown. Here, using a combination of biochemical and biophysical assays, we find that N-sulfation is crucial but alone insufficient for binding and structural activation of HPV16 and is likely aided by 6O-sulfation, whereas 2O-sulfation is dispensable. Biophysical assays confirm the crucial role of N-sulfation in HPV16 binding both at monovalent and multivalent levels. In addition, dynamic force spectroscopy reveals that 6O-sulfation mechanically strengthens the interactions between HS and HPV16 to facilitate binding. Altogether, these results find the direct involvement of sulfation patterns of HS to HPV16 binding and structural activation and reveal how the distinct sulfation groups of HS facilitate the viral attachment to influence the entry and thus will likely play a role in determining the tropism of HPVs.

Adaptive Immune Response to Repeated mRNA Vaccination in the Older Population

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An aged immune system cannot mount an appropriate response necessary to resolve an infection. It is suggested that skewed ratio of naïve and memory lymphocytes and limited specificity of the antigen-specific receptors on B- and T-cells are one of the many causalities. As a result, prophylactic interventions like vaccines have a dampened effect to prevent new infections. There is also a blunted response to a previously experienced pathogen. An important aspect of vaccination is to generate immune memory and induce pathogen specific antibodies. The quality of antibody thus generated is dependent on the vaccine platform. A quadrivalent subunit vaccine for seasonal influenza virus generates lower titers of antibody in the older population with reduced mutations in the antigen binding site compared to a younger population. However, when they are given mRNA vaccine to prevent COVID-19 the age-related difference is evened out. In response to the newly emerging variants of SARS CoV-2 and to decrease the mortality among the older population repeated vaccination is recommended. To understand the adaptive immune response elicited to repeated mRNA vaccination by an aged immune system, we will use next generation sequencing technique to sequence genes encoding antigen specific receptors. The sequences will illuminate the quality of age dependent processes that fine tunes the antibody response. We will also utilize flow cytometry to access expansion of different subsets of B- and T-cells on repeated antigen exposure. The study aims to provide a mechanistic understanding of vaccine response in the elderly population.

Extensive prokaryotic maintenance respiration in the sea influenced by osmoregulation

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Understanding the ability of the ocean to assimilate and release CO2 and its regulation is a grand challenge in aquatic science with impact on development of hypoxia and emission of green-house gases. Microbial aerobic respiration is the major process consuming O2 and releasing CO2 in the biosphere. In this study we determined the relative importance of growth of biomass and maintenance activities for in situ respiration in sea water. The cause of respiration implies different regulation mechanism. Tritiated thymidine assimilation measured in situ growth rates while oxygen consumption was determined by sensor technique using fluorescence quenching. Collected data was analyzed by existing bacteriological model for specific respiration and growth rate. We demonstrated significant relationships in marine field samples where the prokaryotic specific growth rate predicts cell-specific respiration, in accordance with theory from culture models, over a 10- fold salinity range. This enables the first reported direct estimates of maintenance respiration in nature to show a 6-fold variation between 0.12-0.62 fmol O2 cell-1 d-1, comprising 29-72% of prokaryotic specific respiration. The lowest maintenance respiration occurred at salinity close to physiological osmolarity, suggesting osmoregulation as one of the more energy-consuming maintenance activities. A conservative global estimate of maintenance respiration accounted for 66% of the total prokaryotic respiration in the ocean´s mixed layer. This means that maintenance activities dominate the use of the energy generated by prokaryotic respiration in the sea, where osmoregulation is one significant energy consumer. Prokaryotic maintenance activities have greater importance for their ecological fitness and evolution than currently recognized.

Poster #15 (Group D)

Cellular Receptors for Species D Human Adenovirus

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The largest and most diverse group of human adenoviruses (HAdV) is species D, in part due to recombination between the three major coat proteins: the hexon, penton and fiber (1). Species D exhibit a broad tropism, can induce potent cellular immune responses, and have a relatively low seroprevalence which makes them interesting as viral vectors (2-4). The fact that they are able to infect multiple tissues is perhaps also related to the seemingly diverse receptor usage. Previous studies have suggested several proteins and glycans to act as receptors, however, there is conflicting information and little consensus regarding preferences (4-10).

We have generated A549 knockout cells lacking expression of either DSG2, CAR, CD46, or CMAS (an enzyme involved in the addition of terminal sialic acid), all of which are known HAdV receptors and some of which have been suggested as receptors for species D. These A549 knockout cells were infected with 19 diverse species D HAdVs to assess the relative importance of each receptor during infectivity.

Our results show that A549-ΔCD46 cells are significantly less susceptible to species D HAdV compared to A549-wt. Although both CAR and sialic acid have been implied as receptors for species D HAdV (7, 11, 12), neither A549-ΔCAR or -ΔCMAS were less susceptible to infection.

Fibers from species D can engage CAR, implying the possibility of it acting as a primary receptor. However, species D have been shown to have short and non-flexible fibers, and in the case of HAdV-D37 this interferes with its ability to engage CAR for cell entry (13). Furthermore, HAdV-B3 and -B7, which bind with high affinity to DSG2, can use CD46 if it is present in high density through increased avidity (14). Species D have indeed been shown to engage CD46 via the most abundant coat protein – the hexon – implying that this high avidity interaction is important for the virus infection.

Our results indicate a preference for CD46 as receptor within species D HAdVs. The fact that it interacts with the hexon, which is twenty times more abundant than the fiber, could be a defining factor in terms of receptor preferences.

Poster #15 (Group D)

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Poster #16 (Group D)

Multimodal deep learning to predict protein function in bacteria

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Approximately 20-60% of protein coding sequences within bacterial genomes remain uncharacterized. This significant knowledge gap in bacterial genomes emphasizes the importance of accurate function prediction for better understanding of bacterial physiology and adaptations to diverse environments. Given the substantial functional redundancy and genetic diversity within bacterial genomes, relying solely on the protein sequence is insufficient for obtaining new insights into bacterial protein function. To address this knowledge gap, it is essential to leverage data modalities to capture different facets of the protein functionality. We hence develop Deep Expression STructure (DeepEST), a multimodal deep learning framework designed to accurately predict protein function in bacteria. DeepEST comprises two modules. The first one consists of a multi-layer perceptron that takes gene expression and location as input features, providing evidence of the proteins functional roles in biological pathways and processes. The second module utilizes an established protein structure-based predictor, which relies on recurrent and graph convolutional neural networks, revealing insights into functional groups. We develop a novel masked loss function to fine-tune the structure-based predictor for bacterial species. DeepEST combines the two modules through a learnable weighted linear integration. The experimental results show that DeepEST consistently outperforms its constituting modules and the various comparison methods. Furthermore, it provides credible annotations for hypothetical proteins.

Comparison of entry efficiency of different species of *Filoviridae*-derived VLP

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Zaire Ebolavirus (ZEBV) was first discovered in 1976 and soon became one of the most dangerous human pathogens causing Ebolavirus disease (EVD), manifested with high fever, haemorrhage, multiorgan failure and shock. ZEBV belongs to the *Ebolavirus* genus in the *Filoviridae* family, that consists of eight genera with growing number of emerging species that vary in tropism and pathogenicity. Overall, in our work, we aim at establishing whether there is a correlation between pathogenicity of filovirus, the characteristics of their interaction with cell membrane and their entry and egress potential. To that end, we analysed the entry efficiency of various filoviruses. The highly pathogenic MARV (genus *Marburgvirus*) and ZEBV (genus *Ebolavirus*) were chosen as a representative filovirus with the highest case fatality rate (CFR), reaching up to 90%. Moreover, this study included the less pathogenic SUDV (Sudan-Ebola virus) with a CFR around 50% and two filoviruses that never caused lethal infection in humans. First was RESTV (Reston-Ebola virus) that causes only asymptomatic infections in humans and is closely related to ZEBV; second was LLOV (Lloviu virus), which is the only member of distantly related genus Cueravirus and is non-infectious to humans.

Since Filoviruses are BSL4 pathogens, we used a transcription and replication competent virus like particle system (trVLP) as a model in this study, where the particles varied in the species of the produced glycoprotein. This allows the analysis of virus entry of different species in one infection cycle under BSL2 conditions. Detection of Ebola structural proteins and titration of released VLPs allowed us to determine differences in the production rate of virus-like particles that express different type of *Filoviridae* species glycoprotein. Furthermore, co-expression of luciferase with Ebola structural genes enabled tracking of the rate of cell entry and to determine the entry efficiency of various VLPs.

The comparison of the different species of *Filoviridae* will provide understanding of why some species are more efficient in spreading and hopefully help in the preparedness for new emerging viruses.

Poster #18 (Group D)

Unveiling the function of NUP98 and Mediator Complex interactions in the Flavivirus life cycle

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Flaviviruses, such as tick-borne encephalitis virus (TBEV) and west nile virus (WNV), are arthropod-borne viruses that constitute a major global health problem, with millions of human infections annually and no antiviral treatment currently available. They are small, enveloped, RNA viruses that internalize by endocytosis, replicate in replication "vesicles", that are formed in the endoplasmic reticulum (ER), and further assemble and mature across the ER and the Golgi until their egress. Viruses use both viral and cellular factors to complete their life cycle, yet several of these host factors remain unidentified and uncharacterized.

Here we have identified nucleoporin 98 (NUP98) to be an important cellular factor in the flavivirus life cycle, by being recruited to the replication vesicles where it interacts with viral RNA. Furthermore, mass spectrometry analysis of the proteome of NUP98, has also revealed the interaction of this protein with proteins of the Mediator Complex specifically during infection. We observed that knock out of MED1 specifically results in increased flavivirus infection. Taken together, we hypothesized that NUP98 is proviral host factor that can be found at the replication vesicles, yet it also interacts with members of the mediator complex hereby enhancing flavivirus infection.

Structural and mechanistic basis of key intracellular steps of enterovirus infection

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Enteroviruses (EVs) are positive-sense, single stranded RNA virus from the Picornaviridae family. With 106 types, they can cause mild to severe diseases in human, especially children, such as common cold (Rhinovirus), myocarditis (Coxsackievirus B3, CVB3) or poliomyelitis (Poliovirus, PV). EVs are known to remodel the cytoplasm of host cells to create replication organelles allowing efficient viral genome replication. They also hijack the secretory autophagy pathway for replication and viral release. Recent cryo-electron tomography (cryo-ET) work done in the Carlson lab has highlight that PV virions directly assemble on replication membrane through a tethering complex where the tether is thought to be viral ATPase 2C. Moreover, they were able to show one class of autophagosomes is carrying bundles of filament proteins, suspected as filamentous actin (F-actin). My project will address two questions in EV replication: (i) how does the viral protein 2C assist membrane-localized assembly? (ii) what is the identity and role of the filaments in virus-induced autophagy? I will use an integrative approach of cell biology, biochemistry and structural biology and PV and CVB3 will be used as model viruses for this project. I first aim to characterize the viral assembly complex by in vitro reconstitution of the interaction between model membranes, purified 2C, capsid proteins and RNA through cross-linking mass spectrometry (XL-MS) and cryo-ET. By combining focus-ion-beam (FIB) milling and cryo-ET to perform subtomogram averaging, I will study the structure of the tethering complex and the one of the filament proteins found in autophagosomes of infected cells. In parallel, to aid the identification of the filament, I will perform proteomic studies through MS study on isolated autophagosomes infected cells. Finally, combined with knock-out cells, I will be able to identify potential protein candidates and test their effect on EV infection through cryo-ET and live cell imaging.

Poster #20 (Group E)

Toward novel antibacterial targets: decoding the cell wall biology of *Salmonella* Typhimurium

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Bacteria are surrounded by the peptidoglycan (PG) cell wall. Understanding the principles of cell wall biogenesis and remodeling is critical to find new and more effective methods of targeting this unique bacterial structure in order to treat infections. Although, the interest in the PG has increased enormously during the last decade, PG metabolism and its regulation during infection remains poorly understood in intracellular pathogenic bacteria like *Salmonella enterica* serovar Typhimurium, the causative agent of the gastrointestinal disease salmonellosis.

The aim of this project is to establish the molecular repertoire of genetic determinants of peptidoglycan biogenesis and remodeling in the S. Typhimurium to gain a deeper understanding of the principles governing cell wall homeostasis. To uncover novel PG players, we applied a unique systems-level analytical approach of the bacterial cell wall with dedicated bioinformatic tools.

Thus, using genome-wide PG profiling, we characterized the chemical compositions of cell wall of the entire non-redundant mutant library of S. Typhimurium under *in vitro* conditions mimicking the intracellular life in eukaryotic cells. We identify a repertoire of proteins important for cell wall synthesis and regulation. Among them are well-characterized PG associated proteins as well as novel proteins of unknown function in the cell wall biology of S.Typhimurium. Taken together, we present the first comprehensive, omic-scale overview of the cell wall of *Salmonella* Typhimurium and reveal novel potential antimicrobial drug targets.

Invasive Asian urban malaria mosquito *Anopheles stephensi* can result in re-emergence of O'nyong-nyong virus

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O'nyong-nyong virus (ONNV) is an arthritic mosquito-borne alphavirus. ONNV is responsible for multiple endemics in Africa, with the largest outbreak affecting over 2 million people. ONNV is restricted to Africa and is transmitted by two malaria vectors: Anopheles (An) gambiae and An. funestus. Due to globalization and urbanization as well as invasion of the 'urban' Asian malaria mosquito An. stephensi to ONNV endemic areas, there is a possible risk of introduction of the virus to other African countries and continents. An. stephensi is closely related to the primary vector of ONNV, An. gambiae, and has been established in the Horn of Africa since 2012 and is spreading further into the African continent. Since its establishment in Africa, it has increased the cases of malaria in urban areas where malaria was eliminated. We studied the possibility of ONNV infection, dissemination and transmission in the An. stephensi by performing vector competence at four different time points post-infection. From the findings, the average infection rate, dissemination rate, transmission rate and transmission efficiency, were 89.47%, 43.36%, 65.27% and 74.56% respectively. An. stephensi is a competent vector for ONNV. Since the vector prefers urban areas over rural areas, there is a high risk of the introduction of ONNV in urban areas and the expansion of the malaria foci.

Poster #22 (Group E)

Understanding bacterial adaptation strategies through bacterial *in vivo* transcriptomics

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Studying bacterial infections *in vivo* holds high importance as it offers a window into the complex environment of host-pathogen interactions, shedding light on bacterial adaptation and maintenance of infection. Even though *in vitro* studies have broadened our understanding on bacterial virulence and adaptation strategies, many questions about the behavior of bacterial pathogens *in vivo* remain unanswered. Human prosthetic joint infection (PJI) is challenging to treat and often necessitate reconstructive surgery and extended antibiotic therapy, leading to the emergence of antibiotic-resistant bacteria in hospital. These infections serve as prime models for biofilm-related infections, featuring a variety of bacterial types, enabling investigation of various bacterial adaptation strategies. To achieve this, we are developing novel methods to recover intact total RNA and enrich bacterial total RNA to obtain robust and complete bacterial transcriptomes from PJIs. Additionally, to identify distinct subpopulations of infecting bacteria based on variations in their transcriptome and genome, we will encapsulate bacterial cells in semi-permeable capsules (SPCs) and perform single cell RNA-seq and DNA-seq, which is compatible also non-culturable bacteria. Moreover, the full transcriptome and single cell genomics of bacterial pathogens present in infected specimens has the potential to help develop novel methods for detecting both culturable and non-culturable bacterial species, which could aid in the diagnosis and treatment of PJIs. The types of data to be generated by this study and computational analyses provide the foundation for the integration of bacterial genomes, and transcriptomes from infected human specimens to uncover hidden information that can be critical for understanding infection maintenance in human samples.

Diatom biofilms induce the aggregation of clay and sand in sedimentary environments

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In sedimentary environments, clay minerals and sand grains tend to be segregated by hydrodynamic processes. However, in many modern and ancient coastal sedimentary deposits, clay aggregates are observed covering the surface of sand grains. The formation of these aggregates is currently being investigated because they are regarded as potential precursors for the clay coatings abundantly found in buried estuarine sandstones. The exopolymeric substances (EPS) produced in intertidal biofilms could have a major role in the aggregation of clay and sand, although this role was never formerly demonstrated. Here, we present laboratory experiments where we produced clay-coated quartz grains similar to those observed in modern estuarine sands. These coatings were produced at ambient temperature by mixing EPS derived from intertidal diatom biofilms with clay and sand mineral standards. The standards were mixed in order to reconstitute the mineral assemblage observed in the Gironde estuary (France). The observation of sediment-EPS mixes with cryo-Scanning Electron Microscopy and Atomic Force Microscopy demonstrates that EPS form organic bridges between clay and quartz. The physico-chemical properties of EPS were characterized independently through colorimetric assays and Fourier Transform Infrared Spectroscopy. The results show that several EPS components (e.g., proteins, polysaccharides) have a potential to complex to quartz and clay minerals. Our results provide novel insights in the impact of microorganisms in sedimentary processes.

Poster #24 (Group F)

Aggregatibacter actinomycetemcomitans and *Filifactor alocis* as Associated with Periodontal Attachment Loss in a Cohort of Ghanaian Adolescents

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The aims of the present study were to document the presence of *Aggregatibacter actinomyctemcomitans* and the emerging oral pathogen *Filifactor alocis*, as well as to identify genotypes of these bacterial species with enhanced virulence. In addition, these data were analyzed in relation to periodontal pocket depth (PPD) and the progression of PPD from the sampled periodontal sites during a two-year period. Subgingival plaque samples were collected from 172 periodontal pockets of 68 Ghanaian adolescents. PPD at sampling varied from 3-14 mm and the progression from baseline, i.e., two years earlier up to 8 mm. The levels of *A. actinomycetemcomitans* and *F. alocis* were determined with quantitative PCR. The highly leukotoxic JP2-genotype of *A. actinomycetemcomitans* and the ftxA a gene of *F. alocis*, encoding a putative Repeats-in-Toxin (RTX) protein, were detected with conventional PCR. The prevalence of *A. actinomycetemcomitans* was 57%, and 14% of the samples contained the JP2 genotype. *F. alocis* was detected in 92% of the samples and the ftxA gene in 52%. The levels of these bacterial species were significantly associated with enhanced PPD and progression, with a more pronounced impact in sites positive for the JP2 genotype or the ftxA gene. Taken together, the results indicate that the presence of both *A. actinomycetemcomitans* and *F. alocis* with their RTX proteins are linked to increased PPD and progression of disease.

Characterization of UV-induced spectral and morphological changes in bacterial spores

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Bacterial spores are well known for their association with food-borne illness, hospital-acquired infections, and deadly diseases e.g., Anthrax. The detection and decontamination of spores are of paramount importance, with UV-radiation mediated protocols being widely used for spore decontamination. However, unlike vegetative bacterial cells, spores are more resilient to common decontamination protocols, necessitating methods to verify spore inactivation post-decontamination. Here, we investigate the UV-radiation mediated alterations in spectral and morphological properties of bacterial spores in a dose dependent manner. We use a combination of spectroscopic techniques e.g., absorption spectroscopy, fluorescence spectroscopy, Raman spectroscopy, and morphological characterizations by scanning and transmission electron microscopy for assessment of UV-mediated changes in spores. The results from these experiments indicate dimerization of dipicolinic acid (DPA), a core component of spore, structural degradation of protein and DNA, disintegration of spore outer envelope and leaking of DPA from the core of the spores. Overall, these findings have practical applications in the development of new spore decontamination and inactivation verification methods.

Poster #26 (Group F)

In Vivo Transcriptomics of Chronic *Salmonella* Typhimurium Infection

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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% and 2.2% 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 were tested in the mouse infection models.

The role of membrane complexity and heparan sulfate in the early entry stages of SARS-CoV-2 variants

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The worldwide spread of SARS-CoV-2 has been characterised by a high mutation rate of the viral genome with the continuous emergence of variants of concern (VOCs). The spike glycoprotein (S), responsible for engaging the viral receptor ACE2, exhibits the highest density of mutations, suggesting an ongoing evolution to optimize viral entry. While previous research focused on isolated molecular interactions between S and single membrane components, our study explores how avidity and the complexity of the plasma membrane influence virus-host binding kinetics during the early stages of SARS-CoV-2 entry. We employ liposomes decorated with S from several VOCs as virion mimics in single-particle tracking studies on native supported lipid bilayers derived from pulmonary cells. Our findings reveal an increase in affinity of the multivalent bond to the cell surface for Omicron BA.1 compared to early VOCs. We show that heparan sulfate (HS), a glycosaminoglycan commonly expressed on cells' plasma membrane, is an important component for the modulation the interaction with the cell surface and we observe a shift in its role from screening the interaction with ACE2 in early VOCs to an important binding factor for Omicron. This is caused by a ~10-fold increase in Omicron's affinity to HS compared to the original Wuhan strain, as shown using single-molecule force spectroscopy. Our results indicate a transition in the variants' attachment strategy towards the use of HS as an initial docking site, which likely played a role in shaping Omicron's tropism towards infection of the upper airways, milder symptoms, and higher transmissibility.

Simultaneous Membrane and RNA Binding by Tick-Borne Encephalitis Virus Capsid Protein

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Tick-borne encephalitis virus is an enveloped, pathogenic, RNA virus in the family *Flaviviridae*, genus *Orthoflavivirus*. Viral particles are formed when the nucleocapsid, consisting of an RNA genome and multiple copies of the capsid protein, buds through the endoplasmic reticulum membrane and acquires the viral envelope and the associated proteins. The coordination of the nucleocapsid components to the sites of assembly and budding are poorly understood. We have investigate the interactions of the wild-type and truncated capsid proteins with membranes with biophysical methods and model membrane systems. We showed that capsid protein initially binds membranes via electrostatic interactions with negatively-charged lipids, which is followed by membrane insertion. Additionally, we discovered that membrane-bound capsid protein can recruit viral genomic RNA. We confirmed the biological relevance of the biophysical findings by using mass spectrometry to show that purified virions contain negatively-charged lipids. Our results suggest that nucleocapsid assembly is coordinated by negatively-charged membrane patches on the endoplasmic reticulum and that the capsid protein mediates direct contacts between the nucleocapsid and the membrane.

Host response to Langat virus infection at the blood-cerebral spinal fluid interface.

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Tick-borne encephalitis virus (TBEV) is neurotropic virus spreading to humans via infested ticks. Disease severity varies depending on virus strain and between patients, with symptoms ranging from mild flu-like illness to severe manifestations such as neuroinflammation and paralysis. The neuroinvasive capacity of TBEV is the primary risk for human health, yet the mechanism is not understood. To address this in a BSL-2 class setting, 10^5 pfu Langat virus (LGTV) was administered subcutaneously to mice deficient in type I interferon signaling (IPS1$^{-/-}$mice). This model simulates the natural route of TBEV infection and has been established to induce detectable viremia and neuroinvasion. Mouse tissues collected at 0-5 days post infection (d.p.i.) were analyzed by rt-qPCR to detect LGTV RNA. Results reveal presence of LGTV in the skin draining lymph node 1 d.p.i., the following day it had spread reaching the peripheral organs and blood. Interestingly, the 3rd and 4th ventricle choroid plexus are the first organs in the brain to display detectable levels of LGTV RNA, already at 3 d.p.i.. The choroid plexus functions as brain barrier by regulating immune trafficking and molecular exchange between the blood and cerebral spinal fluid. Therefore, we hypothesis that LGTV uses the choroid plexus as an entry site to the brain. To study this, we performed bulk RNA sequencing on choroid plexuses collected 0-3 d.p.i. from LGTV infected IPS1$^{-/-}$mice. Differential gene expression demonstrates a strong antiviral response initiated 2 d.p.i.. Moreover, among the dysregulated genes targets, which may be involved in barrier integrity, were identified.

Regulation of morphological features and metabolic adaptations by the prokaryotic maintenance respiration share

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Respiration is a basic cellular process to control the carbon assimilation and release of CO_2 in marine ecosystems. Prokaryotes exert control over ~33% of the total marine respiration. Notably, maintenance-based respiration constitutes 66% of total respiration and is particularly significant in winter and low productive conditions. This form of respiration is pivotal for sustaining maintenance activities related to prokaryotic cell integrity, macromolecule repair and osmoregulation, without directly contributing to cell growth. Both field and mesocosm experiments show that prokaryotic maintenance respiration share is significant during winter conditions, whereas the share of growth-based respiration dominates summer conditions. However, the regulatory mechanisms governing levels of maintenance activities during winter and summer conditions in marine ecosystems remain elusive. At mesocosm scale, our study revealed that differential expression of maintenance activity genes played a dominant role during winter conditions, whereas cells with significant morphological features were selected during summer conditions. The differential expression of maintenance activity genes during winter conditions was associated with osmoregulation, modification of translational machinery, ATP production, and RNA processing. The significant morphological features during summer conditions referred to cell-cell connections, membrane blebbing, and membrane vesicles. The morphology was also specific with predominance of rods and vibrion cell shape in winter and summer conditions respectively. Our findings illustrate how prokaryotes allocate resources during varying levels of maintenance activities in winter and summer conditions. This motivates further study of prokaryotic morphological features during summer, while investigations of gene expression patterns during winter conditions can advance knowledge about maintenance activities.

Co-infection of HAdV-F41 and AAV-2 as a possible aetiology behind acute hepatitis of unknown origin in children

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In spring 2022, over 1000 cases of acute paediatric hepatitis of unknown origin was reported to the World Health Organization (WHO) globally. A recent publication (Ho *et al*. Nature, 2023) showed a likely association to co-infection with adeno-associated virus type 2 (AAV-2) and adenovirus type F41(AdV-F41). In this study we have isolated and characterized AdV-F41 from a patient sample and investigated co-infection with HAdV-F41 and AAV-2. Patient samples were retrieved and AdV-F41 was isolated and sequenced by NGS. Infection was visualised through confocal microscopy by immunostaining of viral capsid proteins and measured by % of infected cells. Sequencing of isolated HAdV-F41 show a high similarity to prototype AdV-F41 with some distinct differences mainly in early genes. Efficient co-infection of AdV-F41 and AAV-2 in primary hepatocytes was visualised by staining for respective virus capsid proteins. Infecting with only AAV-2 yielded no staining, confirming the need for a helper virus for productive infection of AAV-2. In conclusion, we have isolated HAdV-F41 from a child suffering from hepatitis and present genomic and phenotypical similarities and differences to a previously known prototype strain of AdV-F41. We can show that Ad41 and AAV-2 can efficiently co-infect hepatocytes, and that AAV-2 alone is not sufficient to cause infection and liver damage in immortalized hepatocytes. This study supports a role of AAV-2 and AdV-F41 as a cause of atypical paediatric hepatitis and opens up for helper-satellite viral co-infections as a more common cause of human disease than previously recognized.

A distinctive family of L,D-transpeptidases catalyzing L-Ala-mDAP crosslinks in Alpha and Betaproteobacteria

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Most bacteria are surrounded by an essential protective mesh-like structure called peptidoglycan, made of glycan chains crosslinked through short peptides by enzymes known as transpeptidases. Of these, penicillin-binding DD-transpeptidases connect adjacent peptide stems between their 4th and 3rd amino acids (4,3-type), D-alanine and a meso-diaminopimelic acid (mDAP) in Gram negatives, whereas LD-transpeptidases make the 3,3-type between mDAP^3 residues. While these two processes explain the formation of crosslinks in most bacteria, recent investigations involving non-model species have brought to light novel crosslinking mechanisms that point to the existence of less-explored groups of peptidoglycan crosslinking enzymes. Here, we present the identification and characterization of a novel LD-transpeptidase found in the acetic acid bacterial *Gluconobacter oxydans*, named LDT$_{Go}$, which performs 1,3-type crosslinks between L-Ala^1 and mDAP^3. LDT$_{Go}$-like proteins are conserved among Alpha and Betaproteobacteria species that do not encode LD3,3-transpeptidases. Using a highly active ortholog, we demonstrated *in vitro* that this enzyme can work with non-terminal peptide bonds in the crosslinking process. This property is different from the strict specificity of typical LD- and DD-transpeptidases, which only deal with terminal peptide bonds. The high-resolution crystal structure of LDT$_{Go}$ revealed significant distinctions when compared to 3,3-type LD-transpeptidases. These include a proline-rich region near the N-terminus that restricts substrate access to the active site, and an unprecedented cavity designed to accommodate both the glycan chain and the peptide stem from donor muropeptides, a feature that exhibits broad conservation among LD1,3-transpeptidases. Finally, we demonstrated the involvement of DD-crosslinking turnover in supplying the necessary substrate for LD1,3-transpeptidation. This phenomenon underscores the interplay between structurally distinct crosslinking mechanisms in maintaining cell wall integrity in *G. oxydans*.

Intravenous immunoglobin therapy for COVID-19 in immunocompromised patients: a retrospective cohort study

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Immunocompromised patients without humoral immunity despite repeated vaccinations face a high risk of persistent SARS-CoV-2 infection and poor outcome. There are limited treatment options available. Post-pandemic intravenous immunoglobulins (IVIg) contain high levels of SARS-CoV-2 neutralizing antibodies. We aimed to evaluate if IVIg could be a treatment alternative for these patients. We performed a retrospective cohort study investigating the effects of IVIg to treat COVID-19 in immunocompromised, vaccine non-responding, SARS-CoV-2 viraemic adults in the region of Västerbotten, Sweden. We analysed clinical data, viral load, and anti-SARS-CoV-2 IgG binding and neutralization levels of patient serum samples and IVIg batches. Primary and secondary outcomes were determined as clinical cure and virological cure, respectively. Sixteen patients were analysed. At baseline, before IVIg, eight individuals had severe or critical disease, and 13 were hospitalized. After a median COVID-19 duration of four weeks, a median 60g IVIg infusion significantly increased SARS-CoV-2 binding and neutralizing antibody levels, with broad in vitro activity against tested variants. The treatment resulted in abrogation of viremia in all patients and general improvement in 15 survivors. Fifteen patients achieved the primary endpoint, while 13 met the secondary endpoint of SARS-CoV-2 clearance in plasma and nasopharynx at follow-up after a median of four months. Two subjects with persistent nasopharyngeal SARS-CoV-2 carriage relapsed but were successfully retreated with IVIg. Antibodies in commercially available IVIg bound and neutralized various SARS-CoV-2 variants and were associated with several clinical benefits. Our data suggests that IVIg therapy could be a treatment alternative for COVID-19 in this patient category.

In vitro reconstitution reveals membrane clustering and double-stranded RNA recruitment by the enteroviral AAA+ ATPase 2C

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The *Enterovirus* genus of the *Picornaviridae* family includes non-enveloped, positive-sense single stranded RNA (ssRNA) viruses. This genus of viruses causes many diseases such as poliomyelitis by poliovirus (PV), cardiomyopathy by coxsackievirus B3 (CVB3), common cold by rhinoviruses (RVs) and meningitis by Enterovirus 71 (EV 71). Most of the diseases caused by caused by enteroviruses have no vaccine or drugs against them.

The 7.5 kb enterovirus genome encodes a polypeptide which is subsequently cleaved to yield viral proteins. These viral proteins hijack and modify the membranes of the Golgi and ER to give rise to Replication complexes (RCs). It is on this membranous platform that the virus genome replication and assembly takes place.

One of the proteins that are excised form the viral polyprotein 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 features of 2C is its N-terminus which has an amphipathic helix 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. We have demonstrated that a conserved glycine divides the N-terminal membrane-binding domain of 2C into two amphipathic helices, namely: AH1 and AH2. AH2 plays a key role in mediating the oligomerisation of 2C and is sufficient for its binding to membranes. On the other hand, AH1 primarily functions in orchestrating the clustering of membranes, a function not previously reported. Cryo-electron tomography revealed that multiple copies of 2C decorates the surface between vesicles and facilitates vesicle clustering. The ability of 2C to induce membrane clustering is partially reduced by nucleic acids, suggesting a mechanism by which 2C transitions from its early role in mediating interaction of replication organelles with lipid droplets to a later role where it aids in RNA replication and particle assembly.

Moreover, 2C on its own is capable of recruiting RNA to membranes especially the double-stranded RNA which is a replication intermediate during viral RNA replication. Notably, *in vitro* reconstitution experiments unveil that full-length, membrane-bound 2C is a RNA chaperone and not a helicase. In summary, we have demonstrated two previously unrecognized functions of 2C: membrane clustering and the recruitment of double-stranded RNA to membranes. The reconstitution of functional vesicles decorated with 2C serves as a valuable platform for further biochemical investigations into the protein and its involvement in enterovirus replication.

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Assessing the behavior of the genotoxin-producing Salmonella enterica in pro-carcinogenic mouse models

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Some *Salmonella enterica* serovars as Typhi and Paratyphi secrete a toxin known as typhoid toxin (TT), which induces DNA single- and double-strand breaks in host cells via their cdtB subunit, an homologue to the mammalian DNase I. Induction of DNA damage activates DNA repair mechanisms. 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 with pro-carcinogenic features overcome the tumorigenic barrier and proliferate. The induction of genomic instability by DNA-damaging toxins has been widely studied *in vitro* and *in vivo* in immunodeficient models. However, little is known about the pro-carcinogenic role of the TT in *in vivo* immunocompetent models. Here, we established two chemically-induced cancer models by using the pro-carcinogenic agent azoxymethane (AOM) alone or in combination with the colitis-inducing compound dextran-sodium sulphate (AOM/DSS) to study the contribution of infection by TT-producing *Salmonella* for cancer development. We detected a higher bacterial colonisation of the TT-producing *Salmonella* in colon, compared to the isogenic control strain in the AOM model. The increased colonisation correlated with the activation of the DNA Damage Response, as assessed by staining of the surrogate marker **γ**H2AX, mainly in cells conforming the crypts. Stromal cells underwent mostly oxidative damage, as tested by staining with the marker 8-oxoguanine. We detected a bacterial-dependent sustained inflammation in both models, characterised by the formation of lymphocytic aggregates. Only in few mice infected with the genotoxigenic *Salmonella* development of neoplasia was observed in the AOM/DSS model. The number of these aggregates was bacteria-dependent, but toxin-independent. Interestingly, and despite the strong inflammatory microenvironment upon infection, we did not observe induction of senescence in none of the conditions analysed. Our results highlight the complexity of the tissue microenvironment and point towards a fine-tuned crosstalk between the immune system and the DNA Damage Repair system to hijack tissue homeostasis during infection.

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