Norwegian Biochemical Society

The 57th NBS Contact Meeting 2022

Clarion Hotel The Edge, Tromsø

February 10 - 13, 2022

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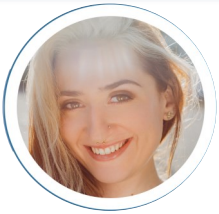
Special Thanks to This Years Planning Committee

Andrea Iselin Elvheim

Bjarte Aarmo Lund



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Hanna-Kirsti Schrøder Leiros

Kristin Lode

Ole K. Greiner-Tollersrud

Sweet Yibio Beyene

Valentyn Oksenych

Veronica K. Pattersen

Message from Your Planning Committee

Dear NBS friends and Colleagues,

Welcome to the 57th NBS Contact Meeting hosted this year in Tromsø! We are so excited for a weekend filled with making connections and scientific discussion. Planning an event during covid times has not been easy but we are happy that we were able to make it happen! Even in covid times, we aimed for a high-quality scientific program. The basis for the meeting is inspired by the previous NBS Contact Meetings consisting of plenary speakers, minisessions, and exhibitor presentations in a wide range of topics. This year in place of a Poster session we are doing Flash Talks!

While visiting Tromsø we hope you take some time to experience the beautiful city and its surroundings. Unfortunately, because of the uncertainties regarding corona-restrictions we will not organize any common social activities, so instead we have written some suggestions:

For the evening

After we have gathered for our Pole Dancing performance and local muscians we hope the fun continues! If the conditions are right, look for the Aurora Borealis! Also, Tromsø has many great bars. We have listed some of our favorites:

— Bardus bar – Great cocktails
— Agenturet – Has a separate gin bar
— Storgata camping – Lots of space and minigolf!
— Bryggeri 13: Tromsø Mikrobryggeri & Balthazar Vinbar
— Magic Ice Bar – Very close to the hotel

Saturday morning

—Tromsø alpinpark – Can be reached by bus 24 (Kroken sør) from Kystens hus to Kroken sykehjem

—Fjellheisen – Cable car up the mountain, with a view over the whole city and the surroundings. Can be reached by bus 26 (Tromsdalen via sentrum) from Torgsenteret to Fjellheisen

—Tromsøbadet – Can be reached by bus 20 or 21 from Smørtorget, or 24 from Fredrik Langes gate, to Templarheimen

—Polaria – Aquarium, within walking distance

For more options or advice please find any of the committee members and ask!

Tromsø Information

Taxi

You can order a Taxi at the Hotel desk or directly to:
Tromsø Taxi (+47) 77 60 30 00

Bus

City buses run within the city of Tromsø from around 06.00 (6am) Monday to Friday, from around 07.00 (7am) Saturday and around 08.00 (8am) on Sunday until midnight. There are one to four departures every hour.

Tickets may be purchased on board the buses, but only accept cash in NOK.

- -Single tickets bought onboard the bus cost NOK 50 for (20-66) and NOK 25 for seniors/children.
- -A day ticket (valid for 24 hours) purchased onboard the bus costs NOK 100 for adults and NOK 50 for seniors/children.

You can also get pre purchased tickets either from downloading the 'Troms Billett' app or from a ticket machine located:

- -F2 - Fr. Langesgt. F2 (by/Kiwi)
- -S1 - Sjøgata S1 (by/Peppes)
- -Giæverbukta terminal
- -UiT The arctic university of Norway
- -UNN - Universitetssykehuset Nord-Norge (University Hospital)
- -Troms fylkestrafikks customer service at Prostneset

Pre-purchased tickets are often cheaper:

- -A pre-purchased single ticket costs NOK 33 for adults and NOK 19 for seniors/children.
- -A pre-purchased day ticket (valid for 24 hours) cost NOK 100 for adults and NOK 50 for seniors/children
- -A seven-days ticket (valid for 168 hours) cost NOK 240 for adults and NOK 120 for seniors/children

Checkout the Tromsø bus website to see bus schedule times and plan your trip:

https://www.tromskortet.no/


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Thursday
February 10th

10.00	Registration—The Edge Hotel
11.00	
12.00	Lunch—The Edge Hotel
13.00	13.00-13.20—Kick-off Information—UiT Rector Dag Rune Olsen
	13.20-14.10—Harald Stenmark, UiO—*'Cellular membrane dynamics and cancer'*
14.00	14.10-14.50—Sebastian Krossa, NTNU—*'Multi-omics of prostate cancer'*
15.00	14.50-15.40—Exhibition Presentations— AH diagnostics, ChemSupport AS, CELLINK LIFE SCIENCES, MCE MedChem Express
	15.40-16.10—Coffee Break
16.00	16.10-17.30—Minisymposium — 1A Session Chair: Stephen Dela Ahator *Sybil Obuobi, Kristen Aaen, Bruna Schuck de Azevedo, and Marta Hammerstad*
17.00	16.10-17.30—Minisymposium — 1B Session Chair: Jorunn Pauline Cavanagh *Oddmund Bakke, Anett Larsen, Harini Pechiappan, Sopisa Benjakul, and Thomais Tsoulia*
	17.30-18.00—Coffee Break
18.00	18.00-19.10—Flash Talks! *Clizia Russotto, Mary Dayne Tai, Marthe Sæter, Morten Rese, Mildrid Hoff, Hibaq Farah, Inga Elise Sommer, Gro Haugseng, Greta Daae Sandsdalen, Ahmed Bargheet, Gaute Bø, Alexander Fiedler, Hendrik Langeloh, Jeanette Grunnvåg, Md Jalal Uddin, Stephen Ahator, Clement Ajayi, and Nikolina Sekulic*
19.00	19.10-19.22—Exhibition Presentations— Ortomedic
	19.22-20.00—Lorena Arranz, UiT—*'Pathophysiology of stem cells'*
20.00	20.00-21.00—Dinner—The Edge Hotel
21.00	21.00-23.00—Social Activities— The Edge Hotel *After dinner please join everyone at the Edge Hotel Bar to enjoy conversations about todays speakers and share your excitement for upcoming presentations!* *Please make sure to check out the information on Tromsø on pages 4-6!*
22.00	
23.00	

Harald Stenmark
University of Oslo

Director of the Centre for Cancer Cell Reprogramming and the Head of Department of Molecular Cell Biology
Professor in the Faculty of Medicine at the University of Oslo

Dr. Harald Stenmark recieved his degrees from the School or Pharmacy and the Norweigian Radium Hospital at the University of Oslo. He performed his post doctoral work at the Cell Biology Program in EMBL, Heidelberg. From there he returned to the University of Oslo and became a research fellow at the Norwegian Radium Hospital before eventually becoming the Head at the Institute for Cancer Research.

Cellular membrane dynamics and cancer

The main research topic of HS has been to understand how alterations in cellular membrane dynamics promote cancer progression. A central contribution was his discovery of a conserved protein domain called the FYVE domain, which binds to the membrane lipid PI3P, the catalytic product of the tumour suppressor PIK3C3. By identifying and characterizing a number of FYVE domain-containing PI3P binding proteins, HS and his co-workers have uncovered how PI3P recruits cytosolic proteins to specific intracellular membranes to control lysosomal degradation of growth factor receptors. This is important in order to understand the tumour suppressor function of PIK3C3 because lysosomal degradation of growth factors, mediated by the so-called ESCRT machinery, is a key mechanism to prevent overstimulation of cells by growth factors, a mechanism that is often inactivated in cancer. A further breakthrough was the identification of two PI3P-binding proteins that cooperate to control lysosome positioning and protrusion outgrowth, a topic of high relevance to cell signalling and cancer metastasis. The group of HS has also shown how PI3P-binding proteins regulate the final stage of cell division, cytokinesis, and demonstrated how their dysfunction can cause an abnormal number of chromosomes, a condition that promotes cancer development. An important discovery was that the ESCRT machinery mediates sealing of the newly formed nuclear envelope during cell division, and that failure of this mechanism causes DNA damage, which is a driver of cancer progression. This was followed up by a study showing that hyper-recruitment of ESCRT proteins to damaged micronuclei causes micronuclear catastrophe and chromosome damage. Recently, the HS group co-discovered ESCRT-mediated mechanisms for repair of damaged lysosomes, and sealing of autophagosomes.

Session Chair: Hanna-Kirsti S. Leiros

Sebastian Krossa
NTNU

Sebastian Krossa is a Post-Doctoral researcher on the ERC funded starting grant “ProstOmics”

Sebastian Krossa studied biochemistry and did his PhD in structural biology and X-ray crystallography at the Kiel University in Northern Germany. His PhD was focused on the structure, function and biological role of proteins related to cancer and the polyamine metabolism. He has a broad multidisciplinary methodological background with a strong emphasis on proteomics, but is also experienced in many cell biology, molecular biology, and metabolomics techniques.

Multi-omics of prostate cancer

In prostate cancer, there is a need for a reliable diagnostic tool that identify men whose PCa is likely to be most aggressive and life threatening from men with slow-growing indolent disease, causing overtreatment and reduced quality of life. To obtain meaningful biological markers in cancer research, “single” omics and “bulk” data have been studied for decades, however not proved sufficient to reveal the causal relationship between molecular signatures and the manifestation of cancer. Bulk techniques lack information about the phenotype of given cell types, and biomarkers may therefore be masked by the average data output. The investigation of multiple dimensions using multi-omics (genomics, transcriptomic, proteomics and metabolomics) and methodology using spatial resolution have the potential to uncover intricate molecular mechanisms underlying different phenotypes of cancer hallmarks.

Multi-omics methods, and especially spatially resolved analyses generate vast amounts of different types of data on cancer cells and their microenvironment. The integration of these large and complex datasets to obtain a biologically meaningful insight is one of the biggest challenges and will become especially powerful if applied directly to human tissue samples to capture the role of the tumor microenvironment that is lost in cell line studies. Novel imaging-based techniques such as matrix-assisted laser desorption ionization time of flight mass spectrometry imaging (MALDI-TOF-MSI), spatial transcriptomics and laser micro-dissected (LMD) proteomics are emerging, and they allow the spatial analysis of gene expression, protein and metabolite distribution within a single tissue sample. Our goal is to study the spatial multi-omics distribution of prostate cancer biomarkers within different cell types in several samples from each individual whole-mount prostate tissue slice.

Session Chair: Hanna-Kirsti S. Leiros

Message from Our Exhibitors

AH diagnostics

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NERLIENS MESZANSKY

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Presentation by Feri Shahin

Session Chair: Hanna-Kirsti S. Leiros

Minisymposium 1A

Session Chair: Stephen Dela Ahator

16.10-16.25 Biohybrid Nucleic Acid carriers for the treatment of persistent infections

Presented by: **Sybil Obuobi**, UiT—The Arctic University of Norway
Coauthors: A. Ngoc Phung, K. Julin, E. G. A., Fredheim, M. Johannessen, and N. Skalko-Basnet

ABSTRACT

Antimicrobial resistant infections are a formidable threat to modern medicine. With a drying antibiotic pipeline, urgent actions are needed to spur research and develop new antibiotics. Because new treatments alone are not sufficient to combat superbugs, it is imperative to safeguard our current last-line antibiotics and to offer innovative strategies that speed up the clinical translation of newer agents. Unfortunately, many antimicrobials suffer poor in vivo stability, low permeability across biological barriers and high systemic toxicities. Against this backdrop, drug delivery nanocarriers fabricated from natural polymers hold immense promise to overcome these challenges. To emulate nature's precise spatial co-localization of bioactive molecules, deoxyribonucleic acid (DNA) based nanocarriers are an attractive platform for antimicrobials given their high polyanionic character, in vivo biodegradability and biocompatibility. To guard against untimely release from these carriers, biohybrids were developed in this project using DNA nanoparticles coated with neutral or zwitterionic lipids. The neutral hybrids exhibited a significantly higher size of 225.20±1.91 nm and high drug entrapment (76.59±3.44 %). On the other hand, the zwitterionic nanoparticles displayed sizes of 217.00 ± 0.59 nm and a neutral zeta potential (0.023 ± 0.018 mV) which drastically increased to 15.23 ± 0.20 mV at acidic pH (5.5). In vitro examinations revealed that the zwitterionic biohybrids had a high binding affinity to various components of the biofilm matrix unlike the neutral formulations which were only responsive to lipase enzymes. Both carriers sustained the release of vancomycin, had high biocompatibility and were effective against persistent intracellular (neutral biohybrids) or biofilm (zwitterionic biohybrids) infections. These results highlight these materials as a feasible platform for the delivery of antibiotics against persistent intracellular and biofilm infections.

16.25-16.40 Engineering of a long-acting and hyperactive coagulation factor IX by fusion to a human albumin variant with improved ability to engage FcRn

Presented by: **Kristin Hovden Aaen**, University of Oslo
Coauthors: Jeannette Nilsen, Silvia Lombardi, Mattia Ferrarese, Torleif Tollefsrud Gjølberg, Francesco Bernardi, Mirko Pinotti, Alessio Branchini, Jan Terje Andersen

ABSTRACT

Frequent injections of recombinant coagulation factor IX (FIX) is used for treatment of the bleeding disorder hemophilia B. However, its therapeutic effect is hampered by a very short half-life of only 18-22 hours. A solution to this has been to extend its half-life by genetic fusion with human serum albumin (HSA). This strategy relies on the capacity of the fusion protein to undergo pH-dependent cellular recycling mediated by the neonatal Fc receptor (FcRn), which is responsible for the three-week long half-life of HSA. However, the half-life extension gained is far from that of natural albumin, which still limits the therapeutic window of FIX-HSA. As such, there is a need for strategies that can further prolong its half-life. To meet this challenge, we designed a novel FIX-HSA fusion protein where we synergistically engineered both fusion partners. First, we inserted a naturally occurring amino acid substitution in FIX (R384L), named Padua, which resulted in 7-fold increased hyperactivity. Second, we combined FIX Padua with an engineered human albumin variant (QMP), which improved binding to human FcRn (hFcRn) with more than 300-fold. When tested in hFcRn transgenic mice, the Padua-QMP fusion showed a 2.7-fold longer half-life compared with the WT counterpart. As such, we report on an engineered FIX-HSA fusion protein with improved coagulation activity combined with extended half-life. These favorable properties may result in a wider therapeutic window where injections may be taken less frequently.

Minisymposium 1A

Session Chair: Stephen Dela Ahator

16.40-16.55 **Cancellation due to covid. Stay healthy and Safe**

16.55-17.10 **Structure-based drug design of 1-Deoxy-D-xylulose 5-phosphate reductoisomerase inhibitors**

Presented by: **Bruna Schuck de Azevedo**, University of Bergen
Coauthors: Vipul Panchal, & Ruth Brenk.

ABSTRACT

The advent of antibiotics has reduced morbidity and mortality due to bacterial infections across the globe, but their increasing use has enabled the development and emergence of resistant microorganisms. Thus, there is an urgent need for antibiotics. The discovery of inhibitors of the 2C-methyl-D-erythritol 4-phosphate (MEP) pathway could help to fuel the antibiotics drug discovery pipeline. The MEP pathway generates precursors of isoprenoids which are natural products essential for many human pathogens. As this pathway is absent in humans, its enzymes are attractive targets for new antibiotics. In this project, we focus on the second enzyme of the MEP pathway, 1-deoxy-D-xylulose 5-phosphate reductoisomerase (DXR). A protocol for the expression and purification of DXR from *M. tuberculosis* and *P. aeruginosa* was established. Currently, the proteins are being used to create a binding assay using Bio-layer interferometry and to screen our in-house fragment libraries. Fragment screening allows a more efficient probing of the chemical space when compared to “drug-like” molecules by detecting small molecules (≤300 kDa) that often have binding constants in the micromolar to millimolar range. Since the size of the chemical space for fragments is much smaller than that of larger compounds, a wider coverage can be obtained by using considerably smaller libraries than in conventional compound screening. The identified fragments will be validated and promising hits will be used as starting points to design potent DXR inhibitors. Hopefully, the discovered ligands will contribute to paving the way towards future antibiotics.

Minisymposium 1A

Session Chair: Stephen Dela Ahator

17.10-17.25 **Redox protection of pathogens by low molecular weight thiols**

Presented by: **Marta Hammerstad**, University of Oslo
Coauthors: Ingvild Gudim, and Hans-Petter Hersleth

ABSTRACT

The growing antimicrobial resistance evolving among pathogens increases the need to search for new antimicrobial targets, including enzymatic networks involved in the maintenance of a pathogen's redox homeostasis. Low G+C Gram-positive *Firmicutes,* such as the clinically important pathogens *Staphylococcus aureus* and *Bacillus cereus*, use the low-molecular weight (LMW) thiol bacillithiol (BSH) as a defense mechanism to buffer the intracellular redox environment and counteract oxidative stress encountered by human neutrophils during infections. The enzyme Bdr has recently been shown to function as an essential NADPH-dependent reductase of oxidized bacillithiol disulfide (BSSB) resulting from stress responses and is crucial in maintaining the reduced pool of BSH and cellular redox balance. In this work, we present the first crystallographic structures of two Bdr enzymes. Our analyses reveal a uniquely organized biological tetramer; however, the monomeric subunit has high structural similarity to other flavoprotein disulfide reductases. The absence of a redox-active cysteine in the vicinity of the FAD isoalloxazine ring implies a new direct disulfide reduction mechanism, which is backed by the presence of a potentially gated channel, serving as a putative binding site for BSSB in proximity to the FAD cofactor. We also report enzymatic activity for Bdrs from *S. aureus* and *B. cereus*, which along with the structures provide important insight into a new class of FAD-containing NADPH-dependent oxidoreductases, related to the emerging fight against pathogenic bacteria.

Coffee Break

Minisymposium 1B

Session Chair: Jorunn Pauline Cavanagh

16.10-16.25 **A new twist to regulation of endosomal maturation**

Presented by: **Oddmund Bakke,** University of Oslo

ABSTRACT

Rab5 and Rab7a are the main determinants of early and late endosomes and are important regulators of endosomal progression. The transport from early endosomes to late endosome seems to be regulated through an endosomal maturation switch, where Rab5 is gradually exchanged by Rab7a on the same endosome. Here, we provide new insight into the mechanism of endosomal maturation, for which we have discovered a stepwise Rab5 detachment, sequentially regulated by Rab7a. The initial detachment of Rab5 is Rab7a independent and demonstrates a diffusion-like first-phase exchange between the cytosol and the endosomal membrane, and a second phase, in which Rab5 converges into specific domains that detach as a Rab5 indigenous endosome. Consequently, we show that early endosomal maturation regulated through the Rab5-to-Rab7a switch induces the formation of new fully functional Rab5-positive early endosomes. From these data we may combine the maturation theory with the endosomal shuttling model and explain the maintenance of endosomal homeostasis. In molecular terms basically nothing is known about regulation of endosomal maturation. The invariant chain (Ii, also known as CD74) is responsible for sorting MHCI and MHCII, to immunoendosomes. When Ii is expressed, endosomal maturation and proteolytic degradation of proteins are delayed. We identified that a SNARE, Vti1b, essential these Ii-induced effects. Vti1b binds to Ii and, truncated Ii lacking the cytoplasmic tail, , relocates Vti1b to the plasma membrane. Our results suggest that Ii, by interacting with the SNARE Vti1b in antigen-presenting cells, directs specific Ii-associated SNARE-mediated fusion in the early part of the endosomal pathway leads to a slower endosomal maturation. We postulate speculate from this that trafficking receptors may interact with the sorting machinery and individually make its unique intracellular membrane dependent pathway.

16.25-16.40 **Role of liver sinusoidal endothelial cells in elimination of virus**

Presented by: **Anett K. Larsen,** UiT—The Arctic University of Norway

Coauthors: Javier Sánchez Romano, Jaione Simón-Santamaria, Kim E. Mortensen, Bo Göran Ericzon, Peter McCourt, Hans H. Hirsch, Christine H. Rinaldo, Bård Smedsrød, Karen K. Sørensen

ABSTRACT

The NRC-funded project SECVIR aims to investigate how viral particles are eliminated by the liver. The liver sinusoidal endothelial cells (LSECs) are pivotal as scavengers of circulating large molecules and nanomaterials. Hence, we hypothesize that LSECs are key contributors in the cellular arm of the antiviral innate immune system. Most viruses taken up by LSECs will likely be eliminated through the effective degradative endolysosomal apparatus, but certain viruses may escape. To date it is unclear to what extent LSECs can support productive infections with pathogens like BK polyomavirus (BKPyV) and human betaherpevirus 5 (HHV-5), although it has been suggested that LSECs may serve as a latent reservoir of other viruses including herpesviruses. HHV-5 was efficiently cleared from blood within 30 min in the mouse model and preliminary results show high uptake of viral particles in liver and spleen. Association of BKPyV with fenestrated hLSECs was verified by the use of super-resolution light microscopy, whereas internalization of viral particles was confirmed using transmission electron microscopy. Internalized viruses were found travelling along microtubules before colocalizing with the endoplasmic reticulum. Newly synthesized early (from both BKPyV and HHV-5) and late viral proteins (from BKPyV) were detected 48 hours post challenge. The production of mature viral particles is yet to be confirmed, however, our findings are noteworthy as productive infection in primary hLSECs has previously not been reported.

Minisymposium 1B

Session Chair: Jorunn Pauline Cavanagh

16.40-16.55 **Effects of Nano-and Microplastics on Inflammatory Responses in Macrophages in vitro.**

Presented by: Harini Pechiappan, NTNU
Coauthors: Felicity Ashcroft, Berit Johansen, and Martin Wagner

ABSTRACT

Humans can be exposed to nano- and microplastics (NMPs) via diet, inhalation, and possibly dermal routes, but the risk to human health is unclear. Phagocytic cells, for instance, macrophages play important roles in innate immunity and present a possible cellular target for NMPs with the potential to induce inflammation. Here, we used the THP-1 monocytic cell line, undifferentiated and differentiated to a macrophage-like phenotype (THP-1-macs), to investigate how exposure to NMPs derived from three different polymers (polymethyl methacrylate, polystyrene, and polyvinyl chloride) affects the inflammatory responses in a phagocytic cell type. Exposure of THP-1 monocytes and THP-1-macs to the highest particle concentrations of NMPs caused little/no cytotoxicity up to 48 h. At 72 h, exposure to all three NMPs reduced the viability of THP-1-macs while polystyrene NMPs were cytotoxic in the THP-1 monocytes, only. Using non-cytotoxic concentrations, we showed by confocal laser scanning microscopy that THP-1-macs internalize all three NMPs as early as 30 min after exposure. Pro-inflammatory responses, measured by NF-κB translocation, expression of genes associated with macrophage activation (CCL2, TNFα, IL-12), and release of cytokines (TNFα and IL-6), were consistently induced in THP-1 -cells by treatment with lipopolysaccharide (LPS) but not by exposure to the NMPs. In contrast, exposure to NMPs suppressed the LPS-induced responses and the IFNγ-induced release of TNFα and IL-6. In summary, we show that the internalization of NMPs from three different polymers was not associated with a pro-inflammatory response in THP-1 cells. Cytokine release in response to LPS and IFNγ was, however, suppressed by NMP exposure, and we thus conclude that the capacity for NMPs to suppress innate immune cell activation merits further investigation.

16.55-17.10 **Engineered ACE2 for blockage of SARS-CoV-2 infection**

Presented by: Sopisa Benjakul, University of Oslo
Coauthors: Aina Karen Anthi, Anette Kolderup, Marina Vaysburd, Heidrun Elisabeth Lode,, Aleksandr Ianevski, Denis Kainov, Karine Flem Karlsen, Siri Sakya, Mari Nyquist-Andersen, Torleif Tollefsrud Gjølberg,, Donna Mallery, Morten Carstens Moe, Magnar Bjørås, Inger Sandlie, Leo C. James, and Jan Terje Andersen

ABSTRACT

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has caused a global pandemic, with substantial impacts on immunocompromised patients who are at high risk of severe coronavirus disease 2019 (COVID-19). Despite the approval of several first-generation vaccines, these patients may not necessarily raise sufficient immune protection. As such, SARS-CoV-2 infection may lead to prolonged viral shedding and the development of immune escape mutant variants. Thus, there is an urgent need for both prophylactic and therapeutic treatment options for immunocompromised patients. To meet this challenge, we have engineered a recombinant fusion protein consisting of dimeric angiotensin-converting enzyme 2 (ACE2), the principle host receptor for SARS-CoV-2. This recombinant ACE2 is tailor-made for favorable pharmacokinetic properties and allows for needle-free delivery across selective mucosal barriers. Importantly, the engineered ACE2 fusion efficiently blocks cellular SARS-CoV-2 infection. As such, our long-acting soluble ACE2 design should be an attractive therapeutic candidate for COVID-19.

Minisymposium 1B

Session Chair: Jorunn Pauline Cavanagh

17.10-17.25 **Early transcriptome responses to Piscine orthoreovirus-1 in Atlantic salmon erythrocytes compared to salmonid kidney cell lines**

Presented by: Thomais Tsoulia, Norwegian Veterinary Institute

Coauthors: Arvind Y.M. Sundaram, Stine Braaen, Øyvind Haugland, Espen Rimstad, Øystein Wessel and Maria K. Dahle

ABSTRACT

The intensification of salmonid farming has increased the importance of fish welfare monitoring. Fish red blood cells (RBC) are nucleated, and in addition to their function in gas exchange they have been characterized as mediators of immune responses. Salmonid RBC are the major target cells of Piscine orthoreovirus (PRV), a virus associated with heart and skeletal muscle inflammation (HSMI) in farmed Atlantic salmon. In the present study, mRNA transcriptome responses were explored in RBC from individual fish, kept ex vivo and exposed to purified PRV for 24 hours. The responses were also studied in macrophage-like salmon head kidney (SHK-1) and endothelial-like Atlantic salmon kidney (ASK) cells, none of which support PRV replication. The antiviral response to PRV was stronger in the RBC and SHK-1 cells, while ASK cells were not significantly responsive. In particular, the transcriptome analysis of RBC revealed that 46 genes were significantly upregulated (≥2-fold upregulation), including interferon regulatory factor 1 (IRF1) and interferon-induced protein with tetratricopeptide repeats 5-like (IFIT9). However, several interferon-regulated antiviral genes which have previously been reported upregulated in PRV infected RBC in vivo (myxovirus resistance (Mx), interferon-stimulated gene 15 (ISG15), toll-like receptor 3 (TLR3)), were not significantly induced after 24h of virus stimulation. These results confirm that RBC are involved in the innate immune response to viruses, but with a delayed antiviral response. A notable difference is that interferon regulatory factor 1 (IRF-1) is the most strongly induced gene in RBC, but not among the significantly induced genes in SHK-1. Putative differences in the binding, recognition and response to PRV, and any link to effects on the ability of PRV to replicate remains to be explored.

Coffee Break

Thanks again to our session sponsor!

Flash Talks!

Each Presenter will have 3 minutes to present their work. This will be great practice for their elevator speech!

Presenter	Title
Clizia Russotto	Identification of new putative ligands for the GnRH receptor
Mary Dayne Tai	The co-chaperone DNAJC12 binds to and stabilizes tyrosine hydroxylase
Marthe Sæter	Investigation of hydroxymethylbilane synthase mutants and intermediates associated with acute intermittent porphyria
Morten Rese	Diversity in myoglobin function
Mildrid Hoff	Exploration of an alginate lyase targeting the biofilm in *Pseudomonas aeruginosa*
Hibaq Farah	Characterization of a β-1,3-glucanase from *Pseudomonas aeruginosa*
Inga Elise Tollan Sommer	Screening for modulators of vesicular monoamine transporter 2 activity in transfected Hek293 cells using a fluorescent substrate.
Gro Haugseng	The role of protein-protein interactions in neurodegenerative disease
Greta Daae Sandsdalen	FISH&CRISPR: Discovery and characterization of CRISPR-associated endonucleases from low-temperature organisms
Ahmed Bargheet	Gut microbiota and antibiotic resistome changes in the first year of life of preterm infants supplemented by probiotics while at NICUs
Gaute Bø	Investigating the mechanisms of colonization resistance in the early life human gut microbiome: a metabolomic approach
Alexander Fiedler	The early gut and skin microbiomes of Atlantic salmon yolk sac fry originating from two different microbial source communities
Hendrik Langeloh	Depletion of immobilized crude oil in temperate Norwegian waters
Jeanette Slettnes Grunnvåg	Mining of enzyme targets for clinical control of vancomycin-resistant *Enterococcus faecium*
Md Jalal Uddin	Identification Of Serine Hydrolase Virulence Factors In Methicillin-resistant - *Staphylococcus aureus* By Using Carmofur-derived Activity-Based Probe
Stephen Dela Ahator	Identification of TCS kinases involved in host-microbe interactions
Clement Ajayi	Activity-based protein profiling of *Staphylococcus aureus* exposed to blood
Nikolina Sekulic	PX Oslo - UiO Structural Biology Core Facilities: Who are we and how can we help you?

Session Chair: Valentyn Oksenych

Message from Our Exhibitor

Ortomedic

Presentation by Thommie Karlsson

Session Chair: Valentyn Oksenych

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Norwegian Graduate School in Biocatalysis

NERLIENS MESZANSKY

Lorena Arranz
UiT

Dr. Lorena Arranz is the Group Leader of Stem Cell Aging and Cancer Research Group

She is part of the Department of Medical Biology, UiT—The Arctic University of Norway

Dr. Arranz graduated in Biology (Biomedicine) and obtained her PhD in Physiology at the Universidad Complutense de Madrid (UCM). Under the supervision of Professor Monica De la Fuente, she studied the contribution of fully differentiated myeloid cells to the aging process, with a focus on oxidation and inflammation. Her PhD Thesis was awarded as the best presented at the Faculty of Biology (UCM) in 2009. She then moved to Fundacion Centro Nacional de Investigaciones Cardiovasculares (CNIC) as Postdoctoral Associate. Working in the group of Dr. Simon Mendez-Ferrer, she identified for the first time the critical role played by the haematopoietic stem cell niche in the pathogenesis of the haematological malignancies classified as myeloproliferative neoplasms. In November 2014, Lorena joined the Department of Medical Biology at the University of Tromsø

Pathophysiology of stem cells

Long life expectancy is resulting in aged societies and a remarkable increase in age-related diseases, including cancer. Stem cells self-renew and provide the source for replenishing mature cells in the organism throughout its life. These fascinating abilities ensure tissue regeneration, but must be fine-tuned regulated as their imbalance may contribute to both, ageing and cancer. Stem cell behaviour results from integration of cell-autonomous signals and extracellular cues received from the stem cell niche. Our research interest focusses on these processes that control stem cell behaviour. Taking the hematopoietic system as the primary model, my group aims at understanding the mechanisms contributing to stem cell malignant transformation using an integrative perspective that considers cell-autonomous alterations in the stem cell and remodeling of the stem cell niche. We will present observations on the potential role of the bone marrow innervation, inflammation and signaling oncometabolites in myeloid leukaemias. Our goal is the identification of novel therapeutic targets of clinical interest that will help improve survival rates and quality of life in patients of haematological malignancies.

Session Chair: Valentyn Oksenych

Friday
February 11th

09.00	09.00-09.50—Nina van Sorge, Amsterdam—*'Sweet appearances: bacterial surface glycans at the host-pathogen interface'*	
10.00	09.50-10.40—Jan Terje Andersen, Oslo—*'Design of biologics with better performance'*	
	10.40-11.00—Coffee Break	
11.00	11.00-11.55—Flash Talks! *Camilla Angeltveit, Ole Golten, Trond Moe, Philipp Garbers, Tamjidmaa Khatanbaatar, Runa Wolden, Martin Christensen, Kamilla Nygård, Melanie Engelfriet, Anna Frida Forsberg, Julie Elisabeth Heggelund, Leif Lindeman, Ingrid Lovise Augestad, Ataur Rahman, Heidi Hillier, and Yanwu Guo*	
12.00	11.55-12.20—ELIXIR Norway—*'Introduction to data management and national resources'*	
	12.20-13.00—Exhibitor Presentations— CENTRE FOR DIGITAL LIFE NORWAY · Inter Instrument AS · Novogene	
13.00	13.00-14.00—Lunch—Clarion Hotel	
14.00	14.00-15.15—Minisymposium — 2A Session Chair: Sybil Obuobi *Gabriel Almeida, Jonathan Hira, Typhaine LeDoujet, Lisa Tietze, and Madeleine Gundersen*	14.00-15.15—Minisymposium — 2B Session Chair: Marta Hammerstad *Nikolina Sekulic, Sebastian Krossa, Torkild Visnes, Mads Bengtsen, and Erik Hjerde*
15.00	15.15-15.35—Coffee Break	
16.00	15.35-16.40—Minisymposium — 3A Session Chair: Oddmund Bakke *Xian Hu, Kang-Xuan Jin, Kristian Prydz, & Svein Støve*	15.35-16.40—Minisymposium — 3B Session Chair: Nikolina Sekulic *Rafal Ciosk, Laura Bacete, Daniela Sueldo, & Manuel Serif*
	16.40-17.10—Coffee Break	
17.00	17.10-17.50—Dan Kastner, NIH—Digital—*'The Systemic Autoinflammatory Diseases: Cutting the Gordian Knots of Inflammation with the Shears of Genomics'*	
18.00	17.50-18.30—Paul Saftig, Kiel—Digital—*'Lysosomes in Health and Disease'*	
19.00	18.30-19.30— Podium Discussion—Inflammation— *Ole K. Greiner-Tollersrud, Dan Kastner, Paul Saftig, Terje Espevik, and Harald Stenmark*	
20.00	19.30-21.00—Dinner—The Edge Hotel	
21.00	21.00—The Edge Hotel— *We hope everyone will join us at the Edge Hotel Bar to enjoy casual discussions about todays speakers! Please make sure to check out the information on Tromsø on pages 4-6!*	

Nina M. van Sorge
Amsterdam UMC

Dr. Nina M. van Sorge is a Professor of Translational Microbiology and the Head of the Netherlands Reference Laboratory for Bacterial Meningitis

Nina van Sorge studied Pharmacy at Utrecht University obtaining her PharmD degree in 2001. She performed her PhD research at the University Medical Center Utrecht in the labs of Prof. J. van de Winkel and Prof. J. Wokke on the topic of autoantibodies in the pathogenesis of Guillain-Barre syndrome. After a short-term overseas fellowship and a mini-postdoc studying bacteria-lectin interactions at Utrecht University, she took up a second post-doc position in the lab of Prof. V. Nizet at UCSD to expanded her knowledge in bacterial pathogenesis. She returned to the UMC Utrecht on a MCSA Fellowship in 2012 and started her own research group in 2014. In 2020, she moved her research group to Amsterdam UMC studying bacterial pathogenesis with a special focus on bacterial glycosylation in this process.

Sweet appearances: bacterial surface glycans at the host-pathogen interface

One of the prime targets for the development of new therapeutic interventions against S. aureus are the wall teichoic acids (WTAs). WTAs are cell wall expressed glycopolymers that are critical for bacterial physiology, antibiotic resistance and colonization. Commonly, *Staphylococcus aureus* expresses WTA composed of a polyribitol-phosphate backbone modified by D-alanine. In addition, WTA display structural variation through glycosylation, which depends on the activity of three dedicated enzymes: TarM, TarS and TarP. This presentation will highlight our findings related to the interaction of WTA glycotypes with innate receptors and human antibodies. First, we have identified human Langerin, a receptor unique to skin epidermal Langerhans cells (LCs), as a receptor for β-linked GlcNAc on *S. aureus* WTA. Recognition by langerin has consequences for induced immunological responses both in vitro and *in vivo* and is abrogated by the presence of common single-nucleotide polymorphisms in the ligand binding domain of langerin6. Second, we have developed and used chemically-synthesized polyribitol-phosphate molecules to dissect interactions between WTA and human antibodies. With these new glycobiology reagents, we have mapped the WTA antibody repertoire in plasma from healthy individuals and patients with culture-confirmed bacteremia with the aim to identify correlates of disease or protection. In addition, we employed fully-defined synthetic WTA molecules to unravel the structural details of the interaction with WTA-specific monoclonal antibodies. This structural information may guide the engineering of WTA-specific mAbs with defined properties for therapeutic applications to treat or prevent *S. aureus* infections.

Session Chair: Valentyn Oksenych & Veronica K. Pettersen

Jan Terje Andersen
University of Oslo

Professor Jan Terje Andersen is the Group Leader of the Adaptive Immunity and Homeostasis Research Group at the Oslo University Hospital

Jan Terje Andersen is a professor in biomedical innovation at Department of Pharmacology, the Faculty of Medicine at University of Oslo, and a research group leader at Department of Immunology, Oslo University Hospital. Jan Terje Andersen, has obtained the Fridtjof Nansen Prize for Early Career Achievements, Oslo University Hospital Early Career Award and is a member of The Young Academy of Norway. He is heading the Laboratory of Adaptive Immunity and Homeostasis, which is studying the cellular processes and molecular interplay underlying the functions of the two most abundant proteins in blood, albumin and IgG. By combining structural and biophysical approaches with cellular and *in vivo* studies, the laboratory is using this in-depth knowledge to design novel albumin and antibody molecules with improved functions, as part of versatile biomedical technology platforms. The laboratory is highly innovative and is the research group at the University of Oslo and Oslo University Hospital with most registered innovations at the technology transfer office, Inven2. The laboratory is extensively collaborating with biotech and pharmaceutical companies. Jan Terje Andersen has obtained the Fridtjof Nansen Prize for Early Career Achievements, Oslo University Hospital Early Career Award and is a member of The Young Academy of Norway.

Design of biologics with better performance

The plasma half-life of the two most abundant proteins in blood, IgG and albumin, is roughly 3 weeks at average. This has made IgG the natural choice for development of antibody therapeutics, while albumin is increasingly used as a fusion partner. Remarkably, the plasma half-life of these unrelated proteins is prolonged by a common cellular receptor, FcRn. I will discuss how in-depth insights into the structural and cellular mechanisms that govern the functions of FcRn pave the way for engineering of albumin and antibody based molecules with improved binding and transport properties. These molecules are attractive building blocks in tailored design of protein-based biologics and subunit vaccines.

Session Chair: Valentyn Oksenych & Veronica K. Pettersen

Flash Talks

Each Presenter will have 3 minutes to present their work. This will be great practice for eveyones elevator speech!

Presenter	Title
Camilla Fløien Angeltveit	The application of LPMOs during enzymatic saccharification of woody biomass
Ole Golten	Python coding for high-throughput analysis of stopped-flow spectroscopic data that shed light on the redox chemistry of lytic polysaccharide monooxygenases
Trond Moe	Hole hopping through tryptophan chains in LPMOs
Philipp Garbers	Biorefining of Enzymatically and Microbially Tailored Side Streams from Plant Protein Production
Tamjidmaa Khatanbaatar	Structure-function analysis of bifunctional metabolic enzymes from the shikimate pathway
Runa Wolden	Characterizing antimicrobial bacteriocin activity in *Staphylococcus haemolyticus*
Martin Christensen	The role of capsule variants in *Staphylococcus haemolyticus* on immune response and biofilm production
Kamilla Nygård	Characterization of methyltransferase-like21C (METTL21C) in muscle
Melanie Engelfriet	Functional studies in *C. elegans* to elucidate the role of METTL13 in mRNA translation during health and disease.
Julie Elisabeth Heggelund	Phage display to improve the lives of celiac disease patients
Leif Lindeman	Gamma radiation induces locus specific changes to chromatin accessibility in zebrafish embryos
Ingrid Lovise Augestad	Inosine independent roles of Endonuclease V
Ataur Rahman	Antimicrobial and antibiofilm potential of marine bacteria
Heidi Hillier	Characterizing Enzymes in The Ectoine Biosynthesis Pathway
Yanwu Guo	Genetic Mapping by Whole-genome sequencing

Session Chair: Valentyn Oksenych & Veronica K. Pettersen

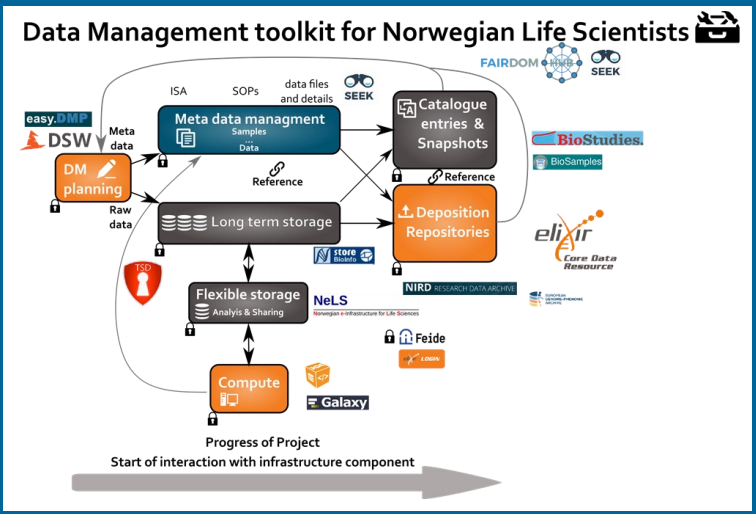
ELIXIR Norway

Presentation by: **Korbinian Bösl**

Major funding agencies require the successful planning and implementation of Data Management. In addition to obtaining funding, good Data Management also makes research more efficient, facilitates collaboration and saves time and resources.

ELIXIR Norway and the Centre for Digital Life Norway have established an End-to-End toolkit, covering the whole data life cycle from proposal to publication, providing practical, existing solutions, available to Norwegian Life Scientists to make their research FAIR (Findable, Accessible, Interoperable and Reusable).

We provide access to the Data Steward Wizard tool as one solution to identify data management needs, plan Data Management budgets and to produce a Data Management Plans (DMPs) according to the specifications by filling an interactive, well-documented questionnaire.



NeLS - the Norwegian e-Infrastructure for Life Sciences is a versatile and backuped storage solution for Life Science data. NeLS is directly integrated with Galaxy, an open, web-based platform for accessible, reproducible, and transparent computational biological research, without the need of programming skills. We have further developed a integration of NeLS with FAIRDOMHub/SEEK, a resource platform for managing data-sets and models in projects and sharing at publication.

ELIXIR maintains as variety of data deposition databases that are suited to host sensitive and non-sensitive data. Usage of this deposition repositories also maximises the visibility of published research.

Session Chair: Valentyn Oksenych & Veronica K. Pettersen

Message from Our Exhibitors

CENTRE FOR DIGITAL LIFE NORWAY

Presentation by Elisabeth Hyldbakk

Inter Instrument AS

Presentation by Dominik Michael Frei

Novogene

Advancing Genomics, Improving Life

Presentation by Miranda Zhao

Session Chair: Valentyn Oksenych & Veronica K. Pettersen

Minisymposium 2A

Session Chair: Sybil Obuobi

14.00-14.15 Mucosal interactions as a bridge between past and future prophylactic phage therapy

Presented by: **Gabriel MF. Almeida**, UiT—The Arctic University of Norway
Coauthors: Lotta-Riina Sundberg

ABSTRACT

The clinical use of bacteriophages (phages) to treat bacterial infections is not a new concept. The development of phage therapy in the early 20th century was heralded as a breakthrough in medical care and phage use became routine in many countries until disappearing almost completely during the 1940s-50s as antibiotics took over. Although the efforts of Georgia, Russia and Poland are well known to this day, much of what has been achieved during the golden years of phage therapy elsewhere is under-reported or even lost. Recently we have retold the forgotten history of Brazilian phage therapy, revealing the widespread clinical use of phages in South America. Among the historical references uncovered, mentions of the prophylactic use of phages caught our attention. Given the positive impact a preventive phage therapy approach could have for mortality and morbidity related to bacterial diseases, it is puzzling why this is not pursued with more effort nowadays. The main reason might be the lack of a mechanism to justify and improve phage-based prophylaxis. Inspired by the bacteriophage adherence to mucus (BAM) model we have shown that a mucus-associated phage can prevent bacterial disease. In this system mucins have a double role: adhesion to them keeps the phage for days inside the mucosa, while mucin exposure increases bacterial virulence and at the same time its susceptibility to the mucosal phage. Thus, mucosal interactions may be the key in understanding why and how phages could be used for preventive measures. In this talk I will present highlights of the historical phage use in Brazil, link these to prophylactic phage use elsewhere, and propose a mechanism to be looked upon that could finally let us pursue preventive phage therapy through an ancient phage-metazoan symbiotic relationship.

14.15-14.30 A comprehensive workflow for dissecting phenotypic heterogeneity of isogenic bacterial pathogen population

Presented by: **Jonathan Hira,** UiT—The Arctic University of Norway
Coauthors: Bhupender Singh, Christian Lentz

ABSTRACT

In recent years, it has become increasingly evident that genetically identical populations of cells of a bacterial pathogen contain subpopulations with different function, e.g., persister cells that are not susceptible to killing by antibiotics and have been associated with chronic infections. These phenotypes are not stable and therefore, escape routine clinical diagnostics. There is a need of approaches that can rapidly determine clinically relevant parameters phenotypically rather than genetically directly from clinical specimens. In the context of an infection and antibiotic resistance, a growing interest towards understanding the molecular mechanism of a pathogen´s heterogenous phenotypes demands the use of single cell technology. Individual cells must be monitored in a high-throughput manner, which enables to acquire novel knowledge of growth dynamics, proliferation, communication, and motion of bacteria. At present, various technologies are available for single cell analysis that includes, microfluidics coupled with chemical biosensors, high resolution imaging, and next-generation sequencing. These technologies have already been demonstrated to be essential tools to quantitatively investigate individual bacterial cell within the complexity of phenotypically heterogeneous populations. Amalgamating these technologies beneath an optimized workflow can speed up the single cell analysis process. Hence, here we have illustrated an end-to-end workflow that will facilitate robust identification/isolation of subpopulations, real-time monitoring of cell physiology, growth dynamics, and profiling of subpopulation based on their transcriptome.

Minisymposium 2A

Session Chair: Sybil Obuobi

14.30-14.45 Utilizing the gut microbiome of Atlantic cod as a platform for the development of cold-active enzymes

Presented by: **Typhaine LeDoujet,** UiT—The Arctic University of Norway
Coauthors: Peik Haugen

ABSTRACT

The migrating Atlantic cod, a cold-water fish, comprises the world's largest population of Atlantic cod. It is an interesting subject for microbiota/microbiome studies because of its distinct life cycle, migration pattern, feeding resources, and economic and cultural importance. The cod intestine is of particular interest for microbiome studies because it interacts directly with its environment, and helps in several processes including digestion. During evolution, its microbiota has evolved to exploit structural features and energy resources found in the gut, and over time, genes that benefit the microbes while minimizing negative impacts on the host are enriched. Given this assumption, the gut microbiota of marine fish is expected to contain bacteria with genes highly specialized for breaking down fibrous proteins (i.e., collagen, keratin, muscles), carbohydrates (e.g., chitin) and other macromolecules. Surprisingly, this obvious source of industry-relevant enzymes and small molecules is largely overlooked. Recently, we published the bacterial diversity as well as the functional profile and metaproteome of the gut of migrating Atlantic cod. Here, we describe how those results can be used as a platform for mining of commercially interesting cold-active enzymes (e.g. chitinase, collagenase, trypsin, aminopeptidase P). In addition, we will present Aliivibrio wodanis as a production host for expressing identified cold-adapted enzymes in the gut of Atlantic cod.

14.45-15.00 Using artificial regulatory sequences to express proteins with *Vibrio natriegens*

Presented by: **Lisa Tietze,** NTNU
Coauthors: Antonia Mangold, Mara W. Hoff, Rahmi Lale

ABSTRACT

Many non-model microorganisms harbor biochemical capacities that are yet to be exploited. One thing we need to exploit non-model organisms, is access to well-characterised regulatory DNA sequences that allow us to reliably control gene expression. However, there is a limited number of well-characterised regulatory DNA sequences for non-model organisms. This limitation restricts the type of work we can do with non-model organisms. Hence, we developed a method that creates artificial regulatory DNA sequences (ARES), which can be used in any microorganism. We show that the method works in non-model organisms such as Vibrio natriegens and can also be used to fabricate inducible gene expression systems. Thus, we developed a method that will hopefully contribute to increased exploitation of underutilized non-model organisms.

Minisymposium 2A

Session Chair: Sybil Obuobi

15.00-15.15 **The effect of periodic disturbances and carrying capacity on the significance of selection and drift in complex bacterial communities**

Presented by: **Madeleine S. Gundersen,** NTNU
Coauthors: Ian Arthur Morelan, Tom Andersen, Ingrid Bakke, and Olav Vadstein

ABSTRACT

Bacteria live in complex and dynamic communities, often consisting of hundreds to thousands of different populations (species). The composition of these communities changes over time due to the four assembly processes selection, drift, dispersal and diversification. Predicting these changes are important but is challenging because the contribution of the stochastic and unpredictable process drift is understudied due to methodological problems. We wanted to understand how periodical disturbances in the bacteria's environment affected the contribution of selection and drift. Therefore, we performed a 2x2 factorial study with varying disturbance (+/-) and nutrient availability (high/low) over 50 days in marine communities without dispersal. Halfway in the experiment, we switched the disturbance regime to increase the robustness of our conclusions. We modelled the rate of change in community similarity between replicates and used this rate to quantify selection and ecological drift. Our novel approach revealed that disturbed communities were structured mainly through selective processes, whereas communities in stable environments were dominated by drift. This was concluded as the disturbed communities on average increased 0.01 day^-1 in Bray-Curtis similarity, whereas the undisturbed communities decreased by 0.007 day^-1. The study I will present is the first to show that periodical disturbances increase selection by allowing for exponential growth periods. The increased contribution of selection as a response to disturbances implies that ecosystem prediction is achievable in non-stable systems.

Coffee Break

Minisymposium 2B

Session Chair: Marta Hammerstad

14.00-14.15 Phosphorylation structurally organizes the [Aurora B/IN-box] complex and introduces a synchronized internal motion of the enzyme resulting in activation

Presented by: **Nikolina Sekulic,** University of Oslo

Coauthors: Dario Segura-Peña*, Oda Hovet, Jannine Dawicki-McKenna, Stine Malene Hansen Wøien, Ben E Black, Michele Cascella

ABSTRACT

Aurora B is a component of the chromosome passenger complex that controls multiple events during cell division. Aurora B, together with IN-box, a C-terminal part of INCENP, constitutes a catalytic component of the complex. In the cell, [Aurora B/IN-box] is activated by auto-phosphorylation while removal of the phosphoryl groups by phosphatases is inhibitory for the complex. However, the effect of phosphorylation on the structural and internal dynamic properties of the enzymatic complex is not clear. We use a combination of experimental (hydrogen-deuterium exchange, enzyme kinetics) and computational (molecular dynamics simulation) approaches to assess how the internal dynamics and function of the [Aurora B/IN-box] complex change with phosphorylation. Our results provide evidence that the unphosphorylated, inhibited form, is more entropic, with the active center only partially assembled. Auto-phosphorylation is associated with structural organization in both, the active center, and the allosteric parts of the enzyme complex. We use a chemical ligation approach to generate partially phosphorylated intermediates and assess the individual contribution of the two major phosphorylation sites: activation segment in Aurora B and TSS motif in IN-box. We found that phosphorylations in the two sites act synergistically to activate the enzyme complex and help it adopt a productive breathing motion. Our study provides a detailed insight in the allosteric communication between Aurora B and IN-box and how this communication is modulated by phosphorylation, resulting in a tight control of the enzymatic activity, an essential requirement for accurate cell division.

14.15-14.30 Inflamed normal appearing glands in the tumor microenvironment of prostate cancer are associated w/ relapse after radical prostatectomy

Presented by: **Sebastian Krossa,** NTNU

Coauthors: Maria K. Andersen, Elise Midtbust, Maximilian Weß, Trond Viset, Øystein Størkersen, Helena Bertilsson, Guro F. Giskeødegård, Morten B. Rye, and May-Britt Tessem

ABSTRACT

Investigating the molecular changes in the tumor microenvironment (TME) is crucial for understanding progression and aggressiveness of prostate cancer (PCa). We used spatially resolved gene expression (ST) data to focus on histologically normal appearing gland tissue in the TME of patients experiencing relapse after radical prostatectomy (RP). We analyzed 32 samples (cancer, close-to-cancer, and non-cancer regions from whole-mount fresh-frozen prostate slices) from patients experiencing relapse (n=5, metastasis, death) or being relapse-free (n=3) 5-12 or 12-13 years after RP, respectively. ST RNA was captured from 10 µm sections, sequenced, classified by histopathology, and normalized by number of cells per capturing area. The remaining material was used for bulk RNA sequencing. The gene set was determined by differential expression analysis, gene ontology enrichment analysis, manual refinement, and filtering based on discriminatory strength. We identified a sub-type of normal appearing glands (NAG) in relapse patients defined by a characteristic upregulation of CCL20, CXCL1/3/5/6/11/17, GABRP and downregulation of AOC1, CARTPT, CYP3A5, KRT13. Of note, the activity of this set was highest in NAG in proximity to low-grade cancer and cancer-free samples but not high-grade cancer tissue. Bulk data showed a higher activity in normal and close to cancer than in cancer samples of relapse patients. Chemokines are known to be associated with PCa growth and invasiveness and CYP3A5 is known to be downregulated in PCa tissue. Reduced activity of the epithelial phenotypic gene KRT13 might be an indicator of EMT in these NAG. Bacterial infections lead to chemokine expression and might cause PCa progression as suggested by Shrestha et al. 2021. Thus, chronic infections might be a cause of the observed inflammation. In conclusion, we found that inflamed, aberrant NAG in the TME are associated with PCa relapse and express a set of chemokines that might serve as easily accessible biomarkers in prostatic fluid.

Minisymposium 2B

Session Chair: Marta Hammerstad

14.30-14.45 Finding new small molecule inhibitors to modulate DNA repair-mediated gene regulation

Presented by: **Torkild Visnes,** SINTEF Industry
Coauthors: Camilla Olaisen*, Sheida Nadeiri, Ane Marit Waagbø, Geir Klinkenberg

ABSTRACT

DNA glycosylases excise and remove modified bases with small chemical adducts caused by oxidation, deamination, and alkylation. Whilst traditionally seen as DNA damage it is now becoming apparent that many of these modified bases are normal intermediates in gene regulation and that DNA glycosylases act as transcriptional regulators rather than DNA repair systems. In recent years, perturbation of the DNA glycosylases through small molecule inhibitors have emerged as a novel way to modulate transcription with the potential to alleviate disease. In cancer cells, DNA glycosylase inhibitors have been shown to modulate transcription of cell cycle genes conferring growth arrest 1, whereas in non-transformed cells DNA glycosylase inhibitors are shown to down-regulate transcription of pro-inflammatory cytokines, thereby attenuating airway inflammation 2–4. These, and other emerging studies, suggest there is a large untapped scientific and therapeutic potential in identifying novel DNA glycosylase inhibitors. Here, we present evidence that two other DNA glycosylases affect gene regulation by stimulating removal of transcriptional silencers from DNA promoters, thereby causing enhanced transcriptional activity of target genes. We hypothesize that inhibitors to these DNA glycosylases may be used to modulate gene regulation in a therapeutically beneficial manner and describe high-throughput screening of ~70,000 small molecules to discover novel inhibitors for two previously undrugged DNA glycosylases.

14.45-15.00 Testosterone affects and alters the epigenome in muscles.

Presented by: **Mads Bengtsen,** University of Oslo
Coauthors: Einar Eftestøl, Kamilla Nygård, Kristian Gundersen

ABSTRACT

Testosterone is an anabolic steroid that induces hypertrophy through an increase in skeletal muscle mass and protein synthesis. Due to its pronounced effect, it is used both in medical treatments and as a doping agent in sports. Interestingly, it has been shown that short term treatment with the anabolic steroid creates long-lasting effects in muscle. However, how the steroid exerts its effect on the molecular level is not fully understood. To understand in more detail how testosterone affects the skeletal muscle and the regulatory program in the muscle-specific myonuclei, we treated mice with testosterone, purified the myonuclear population and analysed the epigenetic landscape of the muscle cells. Genome-wide analysis shows that testosterone treatment induces global changes in the muscle-specific epigenome, both in promoter and distal-regulatory enhancer regions. The alterations create a complex regulatory network that can play a role in the increased hypertrophy and in the long-term physiological effects observed by the steroid.

Minisymposium 2B

Session Chair: Marta Hammerstad

15.00-15.15 **How may ELIXIR Norway help you analyse your data?**

Presented by: Erik Hjerde, UiT—The Arctic University of Norway

Coauthors: Christine Stansberg*, Morten B. Rye, David Dolan, Kjell Petersen, Kidane M Tekle, Dag Inge Våge, Eivind Hovig, Nils Peder Willassen, Pål Sætrom, Inge Jonassen, Espen Åberg, Korbinian Bösl

ABSTRACT

Did you know that you can get help analysing your biological data? ELIXIR Norway, formerly the Norwegian bioinformatics platform, offers a wide set of services, including the Norwegian e-infrastructure for Life Sciences (NeLS), various tools and data resources for analysis and storage, training, and user-support through our nationally coordinated Support desk. The Support desk is linked with local bioinformatics core facilities to support a wide range of bioinformatics needs, including experimental design, data management, advanced analysis, scripting, complex pipelines design and many more. We are competent in e.g. genomics, transcriptomics, proteomics, structural bioinformatics, assembly, variant calling, annotation, gene expression, methylation, epigenetics, statistical genomics, network analysis, metagenomics, microbial genomics. The NeLS platform allows you to store, share and analyse your data, and is linked with a national Galaxy server, where you may process and analyse data directly. We have developed Galaxy workflows for metagenomics, RNA-seq and ChIP-seq data, and have other applications in production. NeLS is also linked to the SEEK platform from FAIRDOM, to support FAIR data management. For sensitive data, we are developing secure and FAIR solutions through the national services for sensitive data (TSD). We organise regular training in using our infrastructure and workflows, and also contribute to bioinformatics courses nationally and internationally.

Coffee Break

Minisymposium 3A

Session Chair: Oddmund Bakke

15.35-15.50 Contour Learning Localization Provides Super Resolution Tracking of Endosome Maturation in Extended Field of Depth Microscopy

Presented by: **Xian Hu**, University of Oslo
Coauthors: Duarte Mateus, Anna Vik Rødseth, Vinodha Manovaseegaran, Xiaochun Xu, Felix Margadant, & Oddmund Bakke

ABSTRACT

It is a great challenge to study the rapid dynamics of intracellular membrane trafficking events in live cells. Multiple intracellular membrane trafficking pathways co-exist and interlace with each other in close proximation in time and space. On top of that, many vesicles undergo fusion and fission events and change in size, shape and even identity as they move more or less rapidly in the cytoplasm along cytoskeletal paths driven by molecular motors. All these events make tracking and analysing their behaviour using modern microscopy technology very challenging. Here we present a new imaging and tracking method tailored for intracellular trafficking vesicle tracking and how we use this method to study the coating protein dynamics during the endosome maturation process. Live cell image series obtained by the superresolution microscopy techniques(Airyscan and Live-SR) of maturing endosomes are tracked by our new ImageJ based seed-growing polygon detection algorithm. The intensities of coat proteins labelled with either the green fluorophore(GFP) or the red fluorophore(RFP/mCherry/mApple) are automatically recorded and analysed. The method was first tested with the well-studied Rab5 and Rab7 switch during endosome maturation and then applied to study the dynamics of other proteins that have been reported to interact with either Rab5 or Rab7 (EEA1, Rab9, Rab 4, Rab8, etc)during the Rab switch phase of the endosome maturation process. We found that some of the proteins are merely visiting the endosomes during the process(e.g. Rab11); but the presence of several others are in phase with the Rab switch(e.g. Rab4, Rab8 and Rab9), indicating that they may play an role in regulating the endosome maturation process.

15.50-16.05 m6A modulates cell fate in embryonic stem cells via signaling pathways

Presented by: **Kang-Xuan Jin**, University of Oslo
Coauthors: Rujuan Zuo, Konstantinos Anastassiadis, Arne Klungland, Carsten Marr, and Adam Filipczyk

ABSTRACT

N6-methyladenosine (m6A) modification affects mRNA metabolism and therefore regulates the gene expression underlying various biological processes. However, how m6A regulate pluripotency in embryonic stem cells (ESCs) remains controversial. To address this issue, we performed detailed characterization of m6A depleted mouse ESCs (mESCs) at single cell resolution. We found that immediate m6A depletion by Mettl3 knockdown promotes mESCs towards pluripotency exit. Furthermore, our live cell imaging data showed that mESCs with increased propensity towards pluripotency exit accumulate overtime. These effects mainly depend on Fgf5 mediated activation of both Erk and Akt pathways. Our data also suggested that the impaired differentiation capacity caused by Mettl3 knockout could be a stable effect of m6A depletion. This study, for the first time, uncover the essential role of signaling pathways in m6A mediated pluripotency regulation and provide a novel explanation for a long-lasting debate in the field. Besides, our results highlight the importance of single cell approaches for dissecting complex biological effects.

Minisymposium 3A

Session Chair: Oddmund Bakke

16.05-16.20 Proteoglycan synthesis in conserved oligomeric Golgi subunit deficient HEK293T cells

Presented by: **Kristian Prydz**, University of Oslo
Coauthors: Ravi Adusumalli, Hans-Christian Åsheim, Vladimir Lupashin, Jessica B Blackburn

ABSTRACT

The Conserved Oligomeric Golgi (COG) complex is an eight subunit protein complex associated with Golgi membranes. Genetic defects affecting individual COG subunits cause congenital disorders of glycosylation (CDGs), due to mislocalization of Golgi proteins involved in glycosylation mechanisms. While the resulting defects in N-and O-glycosylation have been extensively studied, no corresponding study of proteoglycan (PG) synthesis has been undertaken. We here show that glycosaminoglycan (GAG) modification of PGs is significantly reduced, regardless which COG subunit that is missing in HEK293T cells. Least reduction was observed for cells lacking COG1 and COG8 subunits, that bridge the A and B lobes of the complex. Lack of these subunits did not reduce GAG chain lengths of secreted PGs, which was reduced in cells lacking any other subunit (COG2-7). COG3 knock out (KO) cells had particularly reduced ability to polymerize GAG chains. For cell-associated GAGs, the mutant cell lines, except COG4 and COG7 KO, displayed longer GAG chains than wild-type cells, indicating that COG subunits play a role in cellular turnover of PGs. In light of the important roles PGs play in animal development, the effects KO of individual COG subunits have on GAG synthesis could explain the variable severity of COG associated CDGs.

16.20-16.35 HTP screening of the integral membrane proteins vesicular monoamine transporter 2 and mitochondrial complex I

Presented by: **Svein Isungset Støve,** University of Norway
Coauthors: Åge Skjevik, Knut Teigen, Charalampos Tzoulis, Aurora Martinez

ABSTRACT

The vesicular monoamine transporter 2 (VMAT2) is a member of the SLC18 family of transporters and responsible for the uptake of monoamine neurotransmitters such as dopamine, serotonin, histamine, epinephrine, and norepinephrine into synaptic vesicles for subsequent release upon neurotransmission. Due to the role of VMAT2 as a central regulator of monoamine homeostasis, it is an interesting potential drug target of neuronal disorders such as Parkinson's disease or for the treatment of hyperkinetic movement disorders. We have screened VMAT2 for small molecule modulators of substrate transport and identified several new inhibitors of VMAT2. These inhibitors are already approved drugs that are promising candidates for a future use in treatment of hyperkinetic movement disorders such as tardive dyskinesia. Further, by docking and MD simulations of the top ranked hit compounds, we identify VMAT2:drug binding modes and study the molecular determinants involved in inhibitor binding. Finally, we describe our efforts to identify activators mitochondrial complex I, compounds which will have a potential use in treatment of Parkinson's disease.

Minisymposium 3B

Session Chair: Nikolina Sekulic

15.35-15.50 Surviving Hypothermia by ferritin-mediated iron detoxification

Presented by: Rafal Ciosk, University of Oslo

Coauthors: Tina Pekec*, Jarosław Lewandowski, Alicja Komur, Daria Sobańska, Yanwu Guo, Karolina Świtońska-Kurkowska, Jędrzej Małecki, Marcin Frankowski, Maciej Figiel

ABSTRACT

How animals rewire cellular programs to survive cold is a fascinating problem with potential biomedical implications, ranging from emergency medicine to space travel. Studying a hibernation-like response in the free-living nematode *Caenorhabditis elegans*, we uncovered a regulatory axis that enhances the natural resistance of nematodes to severe cold. This axis involves conserved transcription factors, DAF -16/FoxO and PQM-1, which jointly promote cold survival by upregulating FTN-1, a protein related to mammalian ferritin heavy chain (FTH1). We show that inducing expression of FTH1 also promotes cold survival of mammalian neurons, a cell type particularly sensitive to deterioration in hypothermia. Our findings in both animals and cells suggest that FTN-1/FTH1 facilitates cold survival by detoxifying ROS-generating iron species. We finally show that mimicking the effects of FTN-1/FTH1 with drugs protects neurons from cold-induced degeneration, opening a potential avenue to improved treatments of hypothermia.

15.50-16.05 Brillouin microscopy: a novel and powerful tool to investigate mechanical characteristics of plant cell walls

Presented by: Laura Bacete, NTNU

Coauthors: Julia Schulz, Timo Engelsdorf, Zdenka Bartosova, Lauri Vaahtera, Guqi Yan, Joachim Gerhold, Tereza Tichá, Camilla Øvstebø, Nora Gigli-Bisceglia, Svanhild Johanessen-Starheim, Jeremie Margueritat, Hannes Kollist, Thomas Dehoux, Scott A.M. McAdam, Thorsten Hamann

ABSTRACT

The plant cell wall is a complex and dynamic extracellular matrix that surrounds all plant cells. This structure forms the interface between plants and their environment, providing cells with mechanical support as well as containing the high levels of turgor pressure prevalent in plant cells. The cell wall integrity (CWI) maintenance mechanism constantly monitors the functional integrity of cell walls during cell morphogenesis and exposure to stress, initiating adaptive changes in wall composition and structure to ensure plant survival. However, despite its importance, molecular mechanisms underlying CWI maintenance are still poorly understood, as well as their effect on cell wall mechanical characteristics. A key reason for this is that it is challenging to investigate the mechanical characteristics of cell walls below the epidermis in vivo with useful temporal and spatial resolution. Brillouin microscopy is based on the so-called Brillouin light scattering effect, which allows detection of changes in the longitudinal modulus of materials and extra-cellular matrices in vivo, hence allowing the determination of mechanical characteristics such as stiffness and viscosity. We have used that approach to investigate the effects that manipulation of cell wall biosynthesis and turgor levels have on the mechanical characteristics of root cell walls in the model plant *Arabidopsis thaliana.* Our results show that the receptor kinase THESEUS1 (THE1), a key component on CWI-regulatory pathways, is involved in the coordination of the changes in cell wall mechanical proprieties in response to these stimuli. The integration of these results with existing knowledge has enabled us to develop a model on how mechano-perception contributes to CWI maintenance.

Minisymposium 3B

Session Chair: Nikolina Sekulic

16.05-16.20 Extracellular ATP induces bleaching and regulated cell death in *Arabidopsis*

Presented by: Daniela Sueldo, NTNU
Coauthors: Lucia Gimenez Lopez*, Ana Dominguez-Ferreras, Renier van der Hoorn, Vardis Ntoukakis

ABSTRACT

Plant regulated cell death involves tightly controlled signalling cascades that can be genetically or chemically modulated. Environmental stress can lead to regulated cell death, and accurate control is key to minimise tissue damage whilst conferring adaption. Despite having a significant impact on crop performance, the mechanisms that plants use to control cell death propagation are largely unknown. We hypothesise that cell-cell communication, mediated by molecules acting as 'death' or 'life' signals, is instrumental for the control of plant cell death spatiotemporal propagation. Purine nucleotides (ATP, UTP and derivatives) are well described signalling molecules in animals, where they mediate cell death upon injury and during immune responses. In plants, extracellular ATP (eATP) has been identified as a key signalling molecule involved in several physiological processes such as stomatal closure, pollen tube growth, and response to environmental stress. Cell damage (i.e., upon biotic stress) is considered a source of plant eATP and changes in eATP concentration in the apoplast lead to cell death, suggesting eATP can act as a death signal. However, the adaptive role of eATP-cell death and its relation to other types of stress-triggered cell death pathways is poorly understood. At this conference, we will discuss the effect of ATP on the viability of *Arabidopsis* seedlings and the induction of regulated cell death. Furthermore, we will present preliminary data aimed at the establishment of new model species to study eATP physiology in plants.

16.20-16.35 Identification of interaction partners of the Albino3 insertases Alb3a and Alb3b in the diatom *Phaeodactylum tricornutum*

Presented by: Manuel Serif, NTNU
Coauthors: Per Winge and Marianne Nymark

ABSTRACT

Diatoms are a group of eukaryotic microalgae of high ecological importance, contributing to up to 20% of oxygen production worldwide. They are of commercial interest for potential biotechnological applications, e.g. for use as fish feed in aquaculture due to their lipid profile or for bio-remediation of wastewater. Pigment-binding proteins of the light harvesting antenna are inserted into the thylakoid membrane via the chloroplast signal recognition particle pathway. Previous studies in plants and green algae have shown that knockout of genes of this pathway can result in strains with a truncated light harvesting antenna. These strains are valuable for biotechnological approaches as they may grow better in dense bio-reactor cultures. Two isoforms of the Albino3 insertase Alb3, named Alb3a and Alb3b, had been identified in the model diatom *Phaeodactylum tricornutum*. Knockout of Alb3a was attempted, but no bi-allelic loss-of-function mutant could be obtained, indicating that Alb3a is an essential gene. In contrast, knockout of Alb3b resulted in a pronounced change of coloration of the cells from brown to green and a strong decrease in the main light-harvesting antenna proteins was observed. However, growth rates were found to be reduced. Thus, further genes involved in this pathway need to be identified. Therefore, we are employing a proximity labeling approach to identify interaction partners of the two Alb3 proteins. *Phaeodactylum tricornutum* strains expressing fusion proteins of the respective Alb3 isoform and the peroxidase APEX2 were generated. These strains were then acclimated to medium light conditions, followed by a shift to low light conditions to induce Alb3 activity. Upon addition of the membrane-permeable substrate biotin-phenol, APEX2 biotinylates proteins in close proximity, which are then affinity purified and identified by mass spectrometry. Potential interaction partners identified will then be confirmed and characterized by generation of knockout strains.

Dan Kastner
NIH

Dr. Dan Kastner is the Scientific Director of the Division of Intramural Research of the National Human Genome Research Institute

Dr. Dan Kastner obtained his A.B. summa cum laude in philosophy from Princeton University in 1973 and a Ph.D. and M.D. from Baylor College of Medicine by 1982. After completing Internal Medicine residency and chief residency at Baylor, Dan moved to the National Institutes of Health (NIH) in 1985. Dan has won a number of awards and honors, including election to the National Academy of Sciences in 2010 and to the National Academy of Medicine in 2012, recognition as Federal Employee of the Year in 2018, and the Ross Prize in Molecular Medicine in 2019.

The Systemic Autoinflammatory Diseases: Cutting the Gordian Knots of Inflammation with the Shears of Genomics

The systemic autoinflammatory diseases are characterized by seemingly unprovoked episodes of inflammation, without the high-titer autoantibodies or antigen-specific T lymphocytes usually seen in the classic autoimmune diseases. Many of the monogenic autoinflammatory diseases are recognized as inborn errors of innate immunity. Genomic strategies have been instrumental in discovering the molecular basis of the autoinflammatory diseases. In the infancy of the Human Genome Project, the gene mutated in familial Mediterranean fever (FMF) was identified by positional cloning. The FMF gene, MEFV, encodes a protein denoted pyrin; the N-terminal ~90 residues of pyrin define a motif, the PYRIN domain, is a cognate interaction domain found in 20 human proteins involved in innate immunity. The PYRIN domain is crucial to the assembly of the inflammasome, a macromolecular complex involved in the activation of interleukin (IL)-1b, IL-18, and an inflammatory form of cell death known as pyroptosis. Shortly after the discovery of MEFV, a group of periodic fever patients clinically and genetically distinct from FMF were identified, and were found to harbor mutations in TNFRSF1A, encoding the p55 receptor or tumor necrosis factor (TNF). The discovery of the genetic basis for two distinct periodic fever syndromes, both of which could cause fatal AA amyloidosis, but neither of which featured the usual features of autoimmunity, prompted the proposal of the new disease category and the prediction that other periodic fever syndromes would be genetically related. Within about one year the gene mutated in two other periodic fever syndromes, familial cold urticaria and Muckle-Wells syndrome, was discovered by positional cloning. The causative gene, initially denoted CIAS1 but now called NLRP3, was found to encode an N-terminal PYRIN domain. Diseaseassociated mutations were found to cause excessive inflammasome activation, and patients with mutations in this gene were found to be exquisitely responsive to treatment with IL-1 inhibitors. These findings firmly established the concept that autoinflammatory diseases are disorders of innate immunity.

Session Chair: Ole Kristian Greiner-Tollersrud

Paul Saftig

Christian-Albrechts-Universität

Dr. Paul Saftig is the Director and Group Leader of the Molecular Cell Biology and Transgenic Research Group

Paul Saftig, Biochemical Institute at the University Kiel is director of the department. He is chairing a group of scientists working at projects aiming to understand the functions of lysosomes and their role in health and disease. There is a focus to decipher the (patho)physiological role of membrane components of the lysosomal compartment. Approved therapies for lysosomal storage disorders including alpha-mannosidosis were developed in the lab. New trafficking routes to the lysosomal compartment were discovered. The role of lysosomal proteins and different types of membrane-associated proteases in more common neurodegenerative disorders like Alzheimer Disease and Parkinson Disease are addional topics of interest. Dr. Saftig´s work has been presented in about 300 original publications and review articles in highly recognized scientific journals. Dr. Saftig received the highest German Alzheimer Research Award (2011) and the EU-Horizon Impact Award (2019). Dr Saftig has chaired and is still chairing a number of national and European research networks.

Lysosomes in Health and Disease

The pivotal role of lysosomes in cellular processes is increasingly appreciated. An understanding of the balanced interplay between the activity of acidic hydrolases, lysosomal membrane proteins and cytosolic proteins is required. About 60 lysosomal hydrolases continuously help to degrade macromolecules delivered by endocytosis, autophagy or phagocytosis. It has also been appreciated that the lysosomal membrane plays essential roles in a number of cellular events ranging from phagocytosis, autophagy, cell death, virus infection to membrane repair. An overview about the most interesting emerging functions of lysosomal membrane proteins and how they contribute to health and disease will be provided. Lysosomal storage diseases (LSDs) are characterized by disturbances in the interplay between hydrolases, membrane proteins and the cytosolic world. They are characterized by intralysosomal accumulation of substrates, often only in certain cell types. Even though our knowledge of these diseases has increased and different types of therapies have been established, many aspects of the molecular pathology of LSDs remain obscure. It will be discussed how lysosomal storage affects downstream functions linked to lysosomes, such as membrane repair, autophagy, exocytosis, lipid homeostasis, signalling cascades and cell viability. Therapies must aim to correct lysosomal storage not only morphologically, but reverse its (patho)biochemical consequences. Some of the major advantages and drawbacks of current and possible future therapies for LSDs will be discussed. It is currently explored in how far more common diseases such as cancer, cardiovascular and neurodegenerative diseases may profit from a correction of an impaired lysosomal function.

Session Chair: Ole Kristian Greiner-Tollersrud

Podium Discussion
Inflammation

Presentation by: Ole K. Greiner-Tollersrud

Panel Discussion with: Dan Kastner, Paul Saftig, Terje Espevik, and Harald Stenmark

Discovered DADA2 and pioneer of autoinflammatory diseases

Central researcher on Lysosomal Diseases

Inflammasome and Toll-like receptors

Membrane stability and repair systems

After a short presentation by Ole Kristian Greiner-Tollersrud, a multidisciplinary panel of highly recognized experts within inflammation and lysosomes, will give their view on whether the suggested function of ADA2 is compatible with its role as an anti-inflammatory protein, and in a general sense to speculate about a connection between aberrant lysosomal metabolism and activation of pro-inflammatory cytokines in myeloid cells.

In 2014 Dan Kastner at NIH and others described in New England Journal of Medicine a novel monogenic disease which they named “DADA2” (Deficiency of Adenosine Deaminase 2); since whole genome sequencing had revealed that it was caused by loss of function mutations in the ADA2 gene. This started a race to find the function of ADA2, and its anti -inflammatory activity; stimulated by the DADA2 family organization founded by Chip Chambers, a father of two DADA2 affected daughters. The current main model of ADA2 is that it is a secreted myeloid-expressed glycoprotein that functions as a down-regulator of free adenosine during inflammation and/or as a growth factor of anti-inflammatory macrophages. Recent studies in Tromsø, Oslo and Freiburg/Basel challenge the current view by showing that ADA2 is highly expressed in myeloid cells as a mannose-6-phosphate dependent lysosomal glycoprotein, that binds to terminal parts of dsDNA and can modify terminal end adenosines into inosines. Upon degradation into monomers ADA2 can act as a DNase, as shown for brain derived ADA2.

Saturday
February 12th

09.00	09.00-19.50—Matt Bogyo, Stanford—*'Chemical Probes for Imaging Cancer and Infectious Diseases'*
10.00	09.50-10.15—Exhibitor Presentations— BERGMAN DIAGNOSTIKA creoptix sensors
	10.15-11.05—Terje Espevik, NTNU—*'The double-edged sword of inflammation'*
11.00	11.05-15.00—Experience Tromsø and Northern Norway!
12.00	*Check out some ideas on page 4-6!*
13.00	  
14.00	
15.00	15.00-15.30—Coffee Break
	15.30-17.30—Panel Discussion—Career Paths and Opportunities post PhD
16.00	*Moderators: Hermoine Venter and Javier Romano*
	Join us to learn about career pathways, funding opportunities, grant writing advice, mobility, patents, and opportunities in the industry.
17.00	*Panelists: Jan Terje Andersen, Harald Stenmark, Matthew Bogyo, Hans C. Bernstein, Jeanette Hammer Andersen, Kenneth Ruud, Lisa Schroer, Bernd Ketelsen Striberny (ArcticZymes)*
	17.30-18.00—Coffee Break
18.00	18.00-19.45—NBS General Assembly
19.00	
20.00	20.15->—Banquet and Concluding Remarks
	The evening will consist of several performances so be sure to stick around to hear from our local violinist, ***Emilie Arctander****!*
21.00	*You will also not want to miss an after dinner dance routine from our local pole dancer,* ***Elise Dahl-Hansen!***

Matt Bogyo
Stanford University

Dr. Matt Bogyo is a Professor of Pathology and of Microbiology and Immunology at Stanford University

Dr. Bogyo received a B.Sc. degree in Chemistry from Bates College in 1993 and a Ph.D. in Biochemistry from the Massachusetts Institute of Technology in 1997. After completion of his degree he was appointed as a Faculty Fellow in the Department of Biochemistry and Biophysics at the University of California, San Francisco. Dr. Bogyo served as the Head of Chemical Proteomics at Celera Genomics from 2001 to 2003 while maintaining an Adjunct Faculty appointment at UCSF. In the Summer of 2003 Dr. Bogyo joined the Department of Pathology at Stanford Medical School and was appointed as a faculty member in the Department of Microbiology and Immunology in 2004. His interests are focused on the use of chemistry to study the role of proteases in human disease. In particular his laboratory is currently working on understanding the role of cysteine proteases in tumorgenesis and also in the life cycle of human parasites and bacterial pathogens. Dr. Bogyo currently serves on the Editorial Board of Biochemical Journal, Cell Chemical Biology, Molecular and Cellular Proteomics and is an Academic Editor at PLoS One. Dr. Bogyo is a consultant for several biotechnology and pharmaceutical companies in the Bay Area and is a founder and board member of Akrotome Imaing and Facile Therapeutics.

Chemical Probes for Imaging Cancer and Infectious Diseases

Hydrolases are enzymes (i.e. proteases, esterases, lipases) that often play pathogenic roles in many common human diseases such as cancer, asthma, arthritis, atherosclerosis and infection by pathogens. Therefore, tools that allow dynamic monitoring of their activity can be used as diagnostic agents, as imaging contrast agents and for the identification of novel enzymes as drug leads. In this presentation, I will describe our efforts to design and build small molecule probes that can be used to identify, inhibit and image various hydrolase targets in models of cancer and infectious disease. This will include recent advances in protease activated fluorescent probes for real-time visualization of tumors during surgery as well our efforts to identify several new classes of serine hydrolases in pathogenic and commensal bacteria. We believe many of these enzymes will represent valuable imaging and therapy targets that can be used to visualize and disrupt various aspects of colonization and community formation inside a host.

Session Chair: Christian Lentz

Message from Our Exhibitors

Presentation by Morten Thorsholt

Presentation by Edward Fitzgerald

Session Chair: Christian Lentz

Terje Espevik
NTNU

Professor Terje Espevik is the Director for the Centre of Molecular Inflammation Research and a Professor of Cell Biology at NTNU

Professor Terje Espevik research recieved his degrees from NTNU and then continued his research on the cellular and molecular mechanisms that inflammasomes, Toll-like receptors (TLRs) and the complement system are using to mount sterile and non-sterile inflammatory responses. His research group has a long track record and has made several significant contributions within innate immunity and host defence over the last 25 years. The main goal of his research is to develop new methods for better diagnosis and therapy for diseases where inflammation plays a role in the pathology. He was elected to the Norwegian Academy of Sciences.

The double-edged sword of inflammation

When an infection or injury triggers our immune system, it causes an inflammation in the affected area. Inflammation is an essential mechanism that the body uses to remove harmful substances and to start the healing process. The body regulates inflammatory responses carefully and it is generally a transient event which normally happens during an infection or an injury. But if the reaction is too powerful it can result in blood poisoning (sepsis). If the body is unable to remove harmful substances, it can lead to chronic inflammation that is seen in diseases such as arthritis and atherosclerosis. In our research, we have a focus on atherosclerosis which is a progressive chronic inflammatory disease leading to arterial lesions. During atherogenesis, cholesterol precipitates into cholesterol crystals (CC) in the vessel wall, which trigger inflammation in plaques. The mechanisms how this inflammatory response is initiated has been largely unknown. We have previously shown that CC activate the NLRP3 inflammasome with subsequent release of the pro-inflammatory cytokine IL-1β which participates in the atherogenesis. We have also demonstrated that the CC strongly activate the extracellular complement system in blood leading to a robust secondary cytokine response. Whether intracellular complement is also critical for the release of IL-1β from myeloid cells has so far not been explored. Our new data demonstrate the unexpected formation of intracellular C3/C5 convertases leading to C5a production. We have identified the complement receptor C5aR1 as modulator of mitochondrial functions and, consequently, inflammatory responses in macrophages. Together, the data suggest that targeting both extracellular and intracellular complement could be beneficial in sterile inflammatory diseases such as atherosclerosis.

Session Chair: Christian Lentz

Opportunities after your PhD

Moderators: Hermoine Venter & Javier Romano

More than 70% of all PhD students do not want an academic position following their graduation (NIFU Survey of PhD students in Norway, 2017*). Unfortunately, the education during their three to four year scientific education leaves little space to exploring the sectors outside academia. In this panel discussion we will talk about possible career pathways, funding opportunities, grant writing advice, mobility, patents, and opportunities in the industry after your PhD.

Panelist Discussion with:

Jan Terje Andersen, Harald Stenmark, Matthew Bogyo, Hans C. Bernstein, Jeanette Hammer Andersen, Kenneth Ruud, Lisa Schroer, and Bernd Ketelsen Striberny (ArcticZymes)







ArcticZymes Technologies

We will hear from a mixture of early career scientists, Administrators, Group Leaders, PhD students and representatives from industry. Hear about the adventure of how these sceintists got to where they are today and listen to Lisa Schroer who following her PhD, conducted an internship at a pharmaceutical company in Grünenthal in Mexico City to get to know not only how a company is structured, but also how and where her analytical and organizational skills that she acquired during her PhD fit into the industrial sector.

Notes

Notes

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