Disruptive Technologies for Drugging the Undruggable

SciLifeLab Science Summit 2025

POSTER ABSTRACTS

Posters on 2nd and 4th floor
Posters with odd numbers presented at 10:50-11:30
Posters with even numbers presented at 14:40-15:25

SciLifeLab

CELLINK
A BICO COMPANY

CYTENA
A BICO COMPANY

DISPENDIX
A BICO COMPANY

ECHO
A BICO COMPANY

BERNER
LAB

BioNordika

BRIDGE
SWEDEN

CHEMPARTNER
DEDICATED TO LIFESCIENCE

EpiStat
Epidemiology & Statistics

eppendorf

KEM
EN
TEC
Nordic

LabLife.

Ramcon

SHIMADZU

SB
SinoBiological

Teknolab
Sorbent
när special
är standard

TWIST
BIOSCIENCE

Company exhibition

Located on floor 3

Research Posters

Research Infrastructure Posters

Research posters

1. Multiomic analysis by Teton CytoProfiling for drug screening applications at NGI

Abrahan Hernandez Hernandez (abrahan.hernandez@scilifelab.se), National Genomic Infrastructure, SciLifeLab, Karolinska Institutet

The AVITI24 platform, integrated with Teton CytoProfiling chemistry, enables high-throughput, multiomic drug screening by combining cellular imaging and sequencing in a single workflow. Utilizing a compartmentalized flowcell, the system supports co-culture of diverse cell types and simultaneous spatial profiling of RNA (up to 350 transcripts), proteins (200 protein targets), and cell morphology (using Cell Paint) at subcellular resolution. This enables detailed insights into signaling pathways, receptor activation, and compensatory mechanisms such as apoptosis evasion. The platform's scalability and flexibility - supporting millions of cells and customizable panels - make it ideal for iterative testing of drug combinations, doses, and mechanisms of action in both immortalized and primary cell lines.

2. AlphaFold accelerated discovery of psychotropic agonists targeting the trace amine–associated receptor 1

Alejandro Diaz (alejandro.diaz@icm.uu.se), Uppsala University

Artificial intelligence is transforming protein structure prediction, with major implications for drug discovery. We compared virtual screening outcomes using AlphaFold and homology models of the G protein–coupled receptor TAAR1. Over 16 million compounds were docked, and 62 top-ranked hits were tested. AlphaFold models led to a higher hit rate and identified the most potent agonists. One compound showed favorable properties and antipsychotic-like effects in mice. These findings highlight AlphaFold's potential to enhance ligand discovery.

3. BRIDGE Sweden paves the way for biopharmaceuticals from research to clinical trials

Alexandra Patriksson (alexandra.patriksson@testacenter.com), Testa Center

4. Antiviral nucleotide analogues trap chemoresistance factor SAMHD1 in an inactive tetrameric state

Christopher Dirks (christopher.dirks@scilifelab.se), Karolinska Institutet

Objectives: SAMHD1 is a deoxynucleoside triphosphohydrolase (dNTPase) that regulates cellular dNTP levels and has recently been implicated as a resistance factor to nucleoside analogue chemotherapy. This makes SAMHD1 an attractive target for inhibitor development, but no cell-active compounds have been reported so far. In this project we therefore aim to improve our understanding of SAMHD1 biology, especially of its allosteric activation,

oligomerisation and catalytic mechanisms. We aim to diversify the modes of inhibition towards SAMHD1 and provide a starting point to rationally develop allosterically targeting molecules.

Methods: We use biochemical and biophysical methods to study modulators of SAMHD1 activity. This includes enzyme activity assays (high-throughput biochemical endpoint assay & continuous NMR-based kinetic assay), a competitive binding assay to study allosteric site affinity as well as assays to study SAMHD1 oligomerisation (chemical crosslinking, mass photometry).

Results: Antiviral guanine nucleotide analogues Acyclovir- and Ganciclovir-triphosphate can mimic the physiological allosteric activator GTP, inducing the formation of SAMHD1 homotetramers. However, depending on the analogue activator, the resulting catalytic activity can be drastically diminished and, in some cases, abolished. Furthermore, we observe dNTPase activity with mixtures of physiological and non-natural allosteric site 1 activators, suggesting the formation of mixed occupancy homotetramers.

Conclusions: By studying the modulation of SAMHD1 dNTPase activity via nucleotide analogues we identify new avenues of inhibiting this challenging target by trapping it in a stable, inactive tetramer. In addition, we present a novel mode of SAMHD1 allosteric activation through a combination of endogenous activators and nucleotide analogues. We thereby improve our understanding of the allosteric activation process and of SAMHD1's dNTPase activity in the context of its oligomerisation.

5. Cortical suppressors of chromosomal instability in healthy and tumorous epithelia

Christos Samakovlis (christos.samakovlis@scilifelab.se), Stockholm University

Chromosomal instability (CIN) and epithelial polarity-loss are hallmarks of cancers. We have addressed functional links between CIN and cortical epithelial proteins in healthy and tumorous Drosophila tissues. Double knockdown of members of the Par3 and the pins/LGN/GPSM2-mud/NuMA modules in large polyploid stem cells in the larval airways, synergistically induced multipolar spindles accompanied by micronuclei formation at interphase. Additionally, compound inactivation of tumor suppressors, adhesion and junctional proteins or cytoskeletal components enhanced multipolar spindle formation.

In smaller, cuboidal cells of diploid wing imaginal discs, triple knockdowns of members of the Par complex, the pins/LGN/GPSM2-mud/NuMA module and the scribble module synergistically induced lagging or bridged chromosomes and micronuclei but not multipolar spindles. In neither tissue, CIN induction grossly disrupted epithelial architecture.

Oncogenic Ras (RasV12) overexpression either in polyploid or diploid progenitors induced CIN, which was further enhanced by knockdown of cortical suppressors. Single and synthetic

knockdowns of the human orthologs of the fly CIN suppressors in a colorectal cancer cell line similarly boost CIN phenotypes including chromosomal bridges, lagging chromosomes and micronuclei in cancer cells. We reveal a fundamental role of epithelial cortical proteins in chromosomal integrity, in addition to their functions in cell polarization and spindle orientation. Deregulation of the cortical protein modules may interfere with cortical pulling forces on astral microtubule balancing the tension on spindle or kinetochores, suggesting a causative link of perturbed cell polarity and adhesion with CIN in cultured cells or tumors.

6. Fighting cancer with the 1-2-punch approach by identifying drug combinations that improve current cancer therapies

Daniela Hühn (daniela.huhn@scilifelab.se), Karolinska Institutet

Resistance to therapy has been estimated to contribute to treatment failure in up to 90% of cancer patients and remains one of the fundamental challenges in cancer. Drug tolerant and senescent cells accumulate as a consequence of many cancer therapies and are thought to contribute to therapy resistance. Accordingly, “to develop ways to overcome cancer’s resistance to therapy” was one of the 10 recommendations made from the Blue Ribbon Panel associated to the Cancer Moonshot initiative of the National Cancer Institute. In this regard, one specific idea is to find combinations that can eliminate the cancer cells that resist the initial treatment. This is the basis of the so-called “one-two-punch” strategy for cancer therapy, which aims to maximize the efficacy of the initial treatment and thereby reduce tumor relapse.

To advance this concept, we have developed a high-throughput phenotypic drug screening platform to identify novel one-two punch strategies across various cancer types and in combination with approved therapies. This innovative approach enabled us to discover novel senolytics - drugs that specifically target senescent cells. These senolytics have shown remarkable potential in enhancing the anticancer effects of senescence-promoting drugs, such as the CDK4/6 inhibitor Palbociclib. We are expanding our research by testing the efficacy of our candidate compounds in combination with other therapeutics across multiple cancer cell lines, with a particular focus on breast and lung cancers.
By addressing the critical issue of therapy resistance, we aim to contribute significantly to the advancement of cancer treatment and potentially increase survival rates for patients.

7. Capturing Conformational Change : A Strategy for Drugging Dynamic Transporters, Receptors and Ion Channels*

Darko Mitrovic (darmi@kth.se), KTH Royal Institute of Technology

The most pharmacologically relevant membrane proteins—receptors, transporters, and ion channels—function by cycling through multiple conformational states. These transitions are essential to their biological roles but have remained difficult to exploit therapeutically due to their transient, context-dependent nature. We present a novel, disruptive technology that combines bioinformatics, enhanced molecular dynamics simulations, and deep learning to overcome these limitations and enable rational drug design for dynamic proteins traditionally considered "undruggable."

We begin by extracting the key collective variables (CVs) governing conformational transitions. Through tailored deep learning architectures and optimization of CV spaces, we significantly accelerate sampling and convergence, enabling accurate free energy landscape reconstruction within hundreds of nanoseconds. These landscapes reveal not only the thermodynamics of state transitions but also expose transient, cryptic pockets unique to specific functional states.

Building on this, we introduce an inverse design strategy for targeting transporters, receptors or channels. By simulating pocket conformations and inverting their key physicochemical properties—such as electrostatic potential, hydrophobicity, and flexibility—we construct a 3D "pocket fingerprint" dataset. Validated to transporters like OCT1, this approach allows for the targeted design of substrates and modulators with state-specific affinities, opening new paths for therapeutic intervention in diseases linked to transporter dysregulation.

Our method demonstrates that it is possible not only to simulate conformational change with family-wide generalizability for selectivity but also to harness these insights to high-affinity design drugs for targets previously deemed too dynamic or structurally elusive. This work presents a concrete strategy for drugging the undruggable—by capturing motion, quantifying it, and designing ligands to match.

8. AI pathogenic target discovery and drug design

Diandra Daumiller (diandra.daumiller@scilifelab.se), Stockholm University / SciLifeLab

Peptides are a promising class of drugs, exhibiting advantageous properties such as low toxicity, high specificity and stability. Nowadays some Artificial Intelligence (AI) models can rapidly predict protein structures and design peptide binders towards protein targets. Our lab recently introduced EvoBindRare, the first AI model for protein structure prediction and design with 20 canonical plus 29 noncanonical amino acids. Furthermore, AI can also help the

*Selected talk, presented on stage at 14:10

identification of new therapeutic targets by predicting the structural interface of protein-protein interactions. Here we present a comprehensive study leveraging AI protein structure prediction to first discover novel target interfaces and then design selective peptide binders towards them. This approach was applied to Human Herpesviruses, resulting in the identification of a new binding interface and the rapid generation of an effective peptide inhibitor of viral infection using our EvoBind platform. These findings show the significant potential of AI in the drug development process and suggest promising avenues for future model improvements. The final goal? Providing a universal AI tool for designing drugs towards the undruggable!

9. SciLifeLab PULSE

Disa Hammarlöf (disa.l.hammarlof@scilifelab.se), SciLifeLab

10. Poster withdrawn

Poster withdrawn

11. New insights into Epidermolysis Bullosa with MAvatar Discovery: A case study

Feride Eren (feridee.er@gmail.com), Mavatar AB

Epidermolysis Bullosa (EB) is a rare connective tissue disorder manifesting with extreme skin fragility and blister formation in response to minor friction or physical trauma. It has several major subtypes defined by their skin separation level, and the main causes for the disease are mutations in genes encoding proteins essential for skin integrity, specifically those involved in dermal-epidermal adhesion (reviewed by Has et al). Currently, disease treatment focuses on managing symptoms such as wound care and preventing infections.New therapeutics focus on gene therapy and protein replacement ,where finding new protein targets holds an important place.

Using our Mavatar Discovery Platform, which enables disease modeling at the systems level through analysis of thousands of transcriptomics datasets, we aimed to identify context-specific gene networks for EB.

We used our comprehensive skin disease data resource, including conditions such as psoriasis, atopic dermatitis, and blistering disorders, to create gene networks for different EB targets. Investigation of EB simplex focusing on KRT5 and KRT14 mutations created a network including COL17A1, ITGB4, LAMB3, and LAMC2 in close proximity—genes associated with junctional EB mutations. Discovery identified 40 genes

interacting strongly with EB associated genes – known to be involved in cornified envelope formation, amino acid metabolism, estrogen signaling, and PI3K-Akt signaling pathways. These findings align with previous expression-based drug repositioning studies in EBS which identified upregulated inflammatory pathways and mTOR/PI3K upstream regulators as important pathways to the disease.

Our systems-level approach successfully identified interconnected gene networks across EB subtypes, revealing shared molecular pathways that could serve as therapeutic targets, including the cornification and estrogen pathways. These findings demonstrate the potential of computational network analysis in therapeutic discovery.

12. Integrated Hit Discovery Pipeline Targeting Protein–Protein Interactions*

Florian David (davidfl@chalmers.se), Chalmers University of Technology

Innovative drug discovery frameworks that integrate high-throughput screening, data-driven analytics, and systematic validation are laying the groundwork for the next generation of therapies.

Building on this vision, we developed a comprehensive drug discovery workflow, tailored to address protein-protein interaction targets emerging as promising opportunities for therapeutic intervention. The workflow brings together large-scale library screening with quantitative evaluation, data prioritization, and functional follow-up studies. As part of this approach, we engineered a target-sensing biosensor module and successfully deployed it to examine a large library of variant compounds.

With its integrated design, our pipeline offers a flexible and powerful framework for hit identification, enabling faster discovery cycles and broadening the landscape of future therapeutic development.

* Selected talk, presented on stage at 14:10

13. Development of affibody molecules to monitor immune activation

Hugo Olsson (hugoo@kth.se), KTH Royal Institute of Technology

We have developed immune receptor-targeted affibodies that allow the monitoration of immune activation in vivo. This way, thanks to the short half-life of affibodies in circulation, you can acquire same-day radioimages of immune cell activation in response to cues such as immunotherapy for cancer. Crucially, this activation will be resolved both temporally and spatially. Alternatively, our binders could be coupled to a warhead in order to treat immune overactivation in the context of autoimmune disease. The poster summarizes how we've used E. coli display to develop our binders and shows promising data from in vivo studies carried out in mice.

14. Label-free target identification with proteome-wide biophysics

Ilaria Piazza (ilaria.piazza@scilifelab.se), Stockholm University and Karolinska Institutet

Different environmental cues such as stress, nutrients or drugs, trigger rapid adaptive responses that allow to maintain cellular homeostasis. One of the fastest cellular responses to the environment is the binding of small molecules to proteins. These molecular interactions produce allosteric effects, which means that they trigger a variation of protein activity as a consequence of a conformational change. Allosteric interactions are thus essential for life and can modulate both the metabolic status of the cells and gene expression. My group focuses on the development of new MS-bases proteomics technologies to study global structural changes in the proteome. I will describe the applications and latest updates of a proteomic technique called LiP-MS to discover novel protein-drug interactions in microbial and human organisms.

15. In silico protocols to predict stability of amorphous solid dispersion

Jingwen Chen (chenjingwen_apply@163.com), Uppsala University

Amorphous solid dispersions (ASDs) are widely used to enhance the solubility of poorly water-soluble drugs. However, their physical instability, particularly recrystallization, remains a major formulation challenge. To support early-stage screening, we developed an integrated in silico approach that combines molecular dynamics (MD) simulations with machine learning (ML) to predict ASD stability. ML models were trained on a combination of physicochemical descriptors and a comprehensive set of MD-derived features that capture intermolecular interactions and molecular mobility. Incorporating MD features improved prediction accuracy to 85.95%, with feature importance analysis identifying hydrogen bonding and API–excipient contacts as key stability predictors. These findings highlight the value of MD-informed features in enhancing both model performance and mechanistic interpretability.

Building on this framework, we integrate coarse-grained modeling and imaging data to study phase separation in ASDs across multiple scales. Stimulated Raman scattering microscopy enables real-time visualization of dynamic processes, including water ingress, drug–excipient mixing during dissolution, and both amorphous–amorphous and liquid–liquid phase separation. Dissipative particle dynamics simulations complement these observations by modeling mesoscale transformations over extended spatiotemporal windows. Coupling simulation and imaging enables construction of multimodal ML models that capture both molecular-level interactions and macroscale transformation patterns, providing novel validation for highly coarse-grained models.

16. Targeting the "undruggable" cancer-associated protein MYC

Lars-Gunnar Larsson (Lars-Gunnar.Larsson@uu.se), Uppsala University

Deregulation of the *MYC* family oncogenes (*MYC*, *MYCN*, *MYCL*) is implicated in more than half of all tumors, and are often associated with aggressive disease, resistance to therapy and poor prognosis. MYC is therefore a highly warranted target in cancer therapy. Our long-term goal is to develop potent and selective MYC inhibitors with minimal side effects for treatment of patients with MYC-driven cancers. However, MYC has been considered “undruggable”. We have outlined the following strategy to solve this problem: 1) Identification of small molecule inhibitors of the interaction between MYC and MAX, which is strictly required for MYC function. 2) PROTACs targeting the MYC proteins for degradation. We previously identified the molecules MYCMI-6 and -7 that bind MYC and inhibit MYC:MAX interaction (Castell et al. 2018, 2022). A chemistry program to develop these compounds towards clinical drugs has been initiated in collaboration with MyCural Therapeutics AB. PROTACS are synthesized in collaboration with CBCS, SciLifeLab. In preliminary experiments, several new compounds with improved drug characteristics as well as PROTACs show promising activity and selectivity in several types of cancer cells. These are now being further validated for specific anti-MYC activity using various assay systems, including effects on MYC-dependent cell growth/viability, MYC:MAX interaction, regulation of MYC target genes, MYC binding in biophysical assays, target engagement in cells, MYC protein ubiquitylation and degradation and inhibition of MYC/MYCN-driven tumor growth using mouse tumor models.
We hope the new anti-MYC molecules generated in this project will contribute to improved clinical treatments for patients with MYC-driven cancer in the future.

17. Search for dimerization-biased ligands of CCR2: a novel approach to target an elusive receptor family

Lauri Urvas (lauri.urvas@icm.uu.se), Uppsala University

As an important member of the chemokine system, the C-C type chemokine receptor 2 (CCR2) plays a key role in monocyte trafficking and is implicated various diseases including inflammatory and autoimmune diseases, cancer and atherosclerosis. Despite the development of several series of antagonists and the availability of detailed knowledge of the receptor 3D structure, there has been limited success in showing efficacy in clinical trials.

Here we describe a novel approach to target CCR2 by modulating its dimerization. The project is based on a fine understanding of the structure-activity relationships, defined from complementary data obtained following three axis: (1) modeling of CCR2 homodimers and CCR5/CCR2 heterodimers and their validation by cross-linking experiments, (2) curation of a dataset and docking of known CCR2 ligands to determine their binding modes, (3) experimental testing and MD simulations with a small set of CCR2 ligands representing different binding modes to probe their effect on dimerization.

Establishing the connections between the binding modes and specific functional effects will enable the virtual screening of chemical libraries to discover biased ligands, using machine learning to prioritize compounds based on interaction graph similarity to the MD-trajectories.

18. Conserved and Unique: Mapping Protein Dynamics to Rethink Drug Discovery

Lucie Delemotte (lucie.delemotte@gmail.com), KTH Royal Institute of Technology

GPCRs are important drug targets. Why do we not have more drugs targeting them, then? Structure-based drug discovery hinges on discovering ligands that bind with high affinity to a well-defined static binding pocket, thus ignoring the fact that these proteins are dynamic entities that cycle through conformational ensembles to perform their function.
We have designed a method that allows us to obtain conformational ensembles of all GPCRs of interest in all states of interest, thus bypassing the need to obtain experimental structures that can then be subjected to MD simulations.

Targeting these ensembles with structure-based drug discovery methods enables the discovery of new modulation modalities.

19. Targeting the oncogenic TGFb pathway

Maarten De Chateau (maarten.de.chateau@metacurum.com), Metacurum Biotech

Metacurum Biotech is developing a mAb targeting the oncogenic TGFb pathway. This approach is expected to avoid side-effects seen with other TGFb targeting approaches. We have completed in vivo studies in an orthotopic prostate cancer model and see promising effects on tumor growth and metastatic spread. We also have data on other solid tumors like HNC, CRC and TNBC. Moreover, we have a biomarker approach and have developed a separate mAb that can detect a marker that can show if the oncogenic TGFb pathway is active. This has been demonstrated to be the case in a majority of prostate cancer patient samples. The project is ready to go into full pre-clinical development pending financing. The company owns all IP.

20.Targeting oncogenic TGFbeta signaling in prostate cancer

Marene Inga-Britt Landström (Marene.Landstrom@umu.se), Umeå university

Transforming growth factor beta (TGFbeta plays important roles in the growth and metastasis of prostate cancer and many other tumors. Thus, development of therapeutic strategies targeting TGFbeta signaling have been pursued and entered clinical trials, however the pleotropic physiological roles of TGFbeta have limited their clinical usefulness. So far has not treatment strategies targeting TGFbeta in cancer reached clinical stage. The TGFbeta type I receptor (TbetaRI) undergoes proteolytic cleavage by ADAM17 in prostate cancer cells, and other tumor cells, generating a soluble intracellular domain (ICD) which is translocated to the nucleus. Nuclear TbetaRI-ICD promotes invasion of prostate cancer cells.

Herein we report that high expression of TbetaRI is linked to poor prognosis, and correlates with ADAM17 expression, in two different cohorts of mCRPC biopsy RNAseq. Moreover, treatment with a monoclonal antibody (mAb) that specifically prevents cleavage of TbetaRI, and thereby the formation of TbetaRI-ICD, inhibits growth and metastases of a castration-resistant prostate cancer (mCRPC) preclinical model. Notably, this therapeutic strategy does not impact TGFbeta-mediated activation of the canonical Smad pathway. Treatment with a fully humanized mAb, preventing the formation of TbetaRI-ICD, offers a potential novel precision treatment opportunity to block TbetaRI-ICD-related oncogenic responses in cancer, without disturbing physiological TGFbeta signaling.

21. Protein 3D structure determination services at Protein Science Facility

Martin Moche (martin.moche@ki.se), Karolinska Institutet

PSF MX performs 3D structure determination services using macromolecular X-ray crystallography from expression and purification of protein to 3D structural determination, deposition and structural analysis. 3D structure determination of ligand complexes is in high demand as well as developing crystal systems for fragment screening. Small Angle X-ray Scattering (SAXS) is yet another experiment that are gaining popularity among PSF clients to catch the changes in protein structural and oligomeric states in solution when varying buffer conditions.

Maintaining a dialogue with PSF MX clients are very important when taking on MX or SAXS services with ligands since the PI and his/her research group might have accumulated knowledge about their ligands from biochemical and biophysical assays that can benefit crystallization protocol and strategies.

22. The Role of Non-homologous End Joining in the Repair of Different Types of DNA Double-Strand Breaks

Mehran Hariri (mhariri96@gmail.com), Uppsala University

Anticancer agents kill cancer cells by inducing DNA double-strand breaks (DSBs). DSBs vary in complexity, posing different challenges to repair mechanisms. The primary DSB repair pathway is non-homologous end-joining (NHEJ).

However, NHEJ response to different types and complexity of DSB remains elusive. We examined agents which produce DSB and varying ratios of single-strand breaks (SSB) and base damage, calicheamicin, X-rays, phleomycin, etoposide, and temozolomide in wild-type cells and cells with knock-outs (KO) of DNA-PKcs or XRCC4 and analyzed clonogenicity, induction of prompt DSB, non-DSB clusters and DSB repair kinetics. In wild-type cells, DSB were repaired by both fast and slow repair kinetics.

However, in the absence of NHEJ there was only a fast repair phase (30-60 min), whereafter there was no repair the following 1-24 hours. X-rays and calicheamicin treatment resulted in only 20% repair in the NHEJ defective cells, whereas 40-50%, and 85% of DSB were repaired after treatment with phleomycin and etoposide, respectively.

The anticancer agent temozolomide, which do not induce prompt DSB, reduced cell survival in a dose-dependent manner but independent of NHEJ status. Non-DSB clusters, e.g., combination of two or more oxidized bases, single-strand breaks or other DNA lesion that do not form a prompt DSB, increased as the DSB:SSB ratio decreased from calicheamicin, to X-rays, phleomycin, etoposide, and temozolomide, respectively.

Notably, removal of non-DSB clusters occurred rapidly, independent of NHEJ. Although the NHEJ defective cells were hypersensitive to all agents, except temozolomide, the cell survival did not directly correlate to the DSB repair capacity of NHEJ KO cells, suggesting that the role of NHEJ-independent repair pathways vary for DSB of different complexity. Overall, DSB type/complexity affects the repair efficiency.

These insights could be vital for understanding the choice of DSB repair pathway and optimization of DNA repair.

23. Employing CETSA:registered: in primary screening for P53 binders*

Merve Kacal (merve.kacal@pelagobio.com), Pelago Bioscience AB

The confirmation of target engagement is essential for successful drug discovery, yet many programs fail to reach the clinic due to lack of efficacy or failure to show lead candidates interacting with the intended target in physiologically relevant environment. The Cellular Thermal Shift Assay (CETSA:registered:) enables direct measurement of target engagement in living cells, and its high-throughput adaptation (CETSA:registered: HT) provides a powerful platform for primary screening, increasingly exploited, with studies showing low false positive rates (Rowlands et al. 2023 PMID: 36736830).

The tumor suppressor protein p53 plays a central role in preventing neoplastic growth, underscored by the high frequency of mutations in cancers. Many mutations abolish p53's tumor-suppressive functions, while some confer pro-tumorigenic gain-of-function activities. Therapeutic strategies therefore aimed to restore wild-type function or restrict mutant p53 oncogenicity. However, p53 remains a challenging target due to its lack of enzymatic activity and accessible deep binding pockets, and to date no clinical success has been achieved. Here, we performed CETSA:registered: HT screen using Alpha SureFire:registered: technology on p53 in SK-BR-3 cells to identify novel hit compounds against mutant p53 by using our high-throughput screening platform. The screen successfully identified compounds that induced a thermal shift of mutant p53 in intact cells. These hits were further confirmed by qPCR-based efficacy assay which showed effects on p53 regulated transcription. Here, we highlight the advantages of CETSA for screening difficult-to-drug targets, and providing a rapid, target-to-hit lead generation strategy. By priotizing chemical matter that engages the endogenous target in living cells, this approach can reduce reliance on recombinant proteins or modified cell lines, offering potential cost and time savings compared to traditional high-throughput screening approaches that assess target engagement later in the pipeline.

* Selected talk, presented on stage at 14:10

24. Ynamide-based reactive matrices for the sensitive detection of carboxylic acids in MALDI Mass Spectrometry Imaging

Morgane Baudoin (morgane.baudoin@ilk.uu.se), Uppsala University

The central role of small molecule chemical messengers including neurotransmitters (NT) and neuromodulators (NM) as central drivers in neurodegenerative diseases such as Alzheimer's and Parkinson's disease (PD) is well established. However, detecting and monitoring these molecules require sensitive and selective analytical methods. Molecular Mass Spectroscopy Imaging (MSI) is an emerging and powerful tool that allows the two- and three- dimensional visualization of compounds distribution and concentration in a tissue sample. The most common MSI approach relies on Matrix Assisted Laser Desorption Ionization (MALDI-MSI), a soft ionization MS technique that enables the in situ and label-free detection of analytes in tissue sections with high sensitivity and lateral resolution. Lately, fluoromethylpyridiums (FMP) have been discovered, allowing the sensitive detection of small molecules and metabolites in MALDI-MSI. [1,2] These compounds enhance the detection of amines and phenols by the incorporation of three functional moieties: a highly reactive group for analyte capture, a permanently charged group to enhance mass-sensitivity and a conjugated chromophore to increase laser desorption.

To expand the scope of functional groups detected, we are now developing new compound classes that are able to react selectively with alcohols, carbonyls or carboxyl groups. For this purpose, the ynamide functional group has been identified as a promising reactive matrix for its reagent-free and selective reaction with carboxylic acids.[3] Preliminary experiments are showing some encouraging results in the detection of homovanillic acid as a dopamine's metabolite and malic acid, involved in the citric acid cycle, leading us to the current development of a series of analogs that are being tested.

[1] Nat Methods, 2019, 16, 1021–1028. [2] Nature Protocols, 2019, 16, 3298–3321. [3] J. Am. Chem. Soc. 2016, 138, 40, 13135–1313825.

25. Targeting loss of heterozygosity in cancer

Natallia Rameika (natallia.rameika@igp.uu.se), Uppsala University

Overcoming cancer treatment limitations by targeting emerging tumor vulnerabilities is investigated as a credible therapeutic approach. We suggest exploiting loss of heterozygosity (LOH) at highly polymorphic gene loci as a target for cancer therapy. We have investigated 8p22 and 22q13 loss in cancer and found passenger genes deletions in these loci that after LOH in a cancer cell might make it sensitive to certain medicinal treatments compared to normal cells that remain heterozygous. We showed it in proof-of-concept studies targeting polymorphic drug-metabolizing NAT2 and CYP2D6 genes and now aim to find novel targets for this approach.

We evaluated variant alleles in known cancer genes with altered functionality as well as genes located on the commonly lost chromosomal loci, which resulted in around 5500 potential new target genes for the proposed therapeutic strategy by processing ExAc and 1000 Genomes data. We further limited the selection to a ranked set of polymorphisms with a likely effect on the target gene function and performed a visual inspection of AlphaFold predicted 3D structural models of proteins for all putative target genes with around 800 SNVs over 10% frequency on chr1p21-36, chr 17p11-13, chr 3p26-11, 8p26-11 and all loci on chromosome 18. These chromosome arms were prioritized as they are frequently lost in common solid tumor types like CRC, NSCLC or OvCa. Our analysis of constitutional variants resulted in 70 putative genes of interest. As 12 genes are involved in cell cycle, DNA damage pathways we initiated the experimental work with that focus. We are testing the functional impact of common SNVs in ATR and ERCC2 genes with CRISPR SELECT methodology, where the selective advantage of particular variants for cell fitness, drug sensitivity or other features can be shown. A therapeutic approach targeting allelic diversity in a cancer cell and accounting for constitutional variants in the lost genes may serve as a new approach in cancer treatment

26. Suspension-grown 3D colonoids recapitulate human colonic epithelial drug transport, metabolism, and distribution in vitro

Rebekkah Hammar (rebekkah.hammar@uu.se), Uppsala University

Our objective was to study drug absorption, distribution, metabolism, and excretion (ADME) in a physiologically relevant colonic in vitro model. To this end, we aimed to establish three-dimensional (3D) colonoids from adult colonic stem cells. Having established this in vitro model, growth in suspension yielded apical- and basal-out 3D colonoids. Both types were subsequently validated using different microscopic and proteomic techniques combined with functional integrity, transport, and metabolism assays, using matched patient tissue and isolated primary cells for comparison. Finally, to better understand drug distribution in the colon compartment, we determined intracellular bioavailability (Fic) in colonoids using a representative drug panel. These values were benchmarked against the current standard model for Fic, Human Embryonic Kidney (HEK293) cells.

Colonoid morphology, barriers, and ADME proteomes were characterized and found to be comparable to those of native epithelium, best represented by primary cell isolates. Key transporters (PGp, BCRP) and phase I (CYP3A) and II (SULT1A, and UGT1A families) drug metabolizing enzymes (DMEs) were detected via orthogonal methods (global and targeted proteomics, immunocytochemistry stainings) and functionally characterized. Notably, colonoid phospholipid content and drug accumulation kinetics differed from that of HEK293 cells, most strongly impacting Fic of polar compounds. We conclude that colonoids are a physiologically relevant in vitro ADME model. Importantly, simpler models such as conventional monolayers lack the 3D geometry and in-vivo-like lipid and protein profiles displayed by the colonoids which are needed to recapitulate colonic epithelial ADME.

27. Modelling Neurological disordee using orgnoid and organoid-based chip model for drig screening

Rekha Tripathi (rekha.tripathi@scilifelab.se), KTH Royal Institute of Technology

Background: Glucose is the primary energy source for the brain, transported via solute carrier transporter family members. SLC2A1 (GLUT1) is a crucial glucose transporter at the blood-brain barrier (BBB) and cerebrospinal fluid-blood-brain barrier (CSF-BBB), facilitating glucose uptake into the brain and cerebrospinal fluid (CSF). GLUT1 plays a critical role in maintaining glucose homeostasis within the CSF, ensuring adequate energy supply to the brain. Mutations in SLC2A1 cause GLUT1 Deficiency Syndrome (GLUT1DS), leading to seizures, microcephaly, and other neurological symptoms. The standard treatment, a ketogenic diet, has limited efficacy and long-term adverse effects, necessitating alternative therapeutic approaches1,2.

Objectives: This study aims to develop a choroid plexus organoid model to investigate glucose transport in GLUT1DS.

Methods: We generated choroid plexus organoids from patient-derived GLUT1DS iPSCs and an isogenic corrected control. Organoids were characterized by morphological and metabolic differences using biochemical assays, glucose uptake studies, and omics-based analyses. Results: GLUT1DS choroid plexus organoids exhibited early cyst formation, reduced cerebrospinal fluid (CSF) production, and lower glucose levels compared to isogenic controls. Metabolic profiling confirmed altered glucose metabolism, which will be further validated using 13C-glucose uptake assays.

Conclusion: Our study establishes a clinically relevant choroid plexus organoid model to investigate glucose transport d eficits in GLUT1DS. This model provides a platform for mechanistic studies, offering new insights into therapeutic strategies for GLUT1DS.

28. Single-Cell Protein Interactomes by the Proximity Network Assay

Simon Fredriksson (simon.fredriksson@pixelgen.com), Pixelgen Technologies AB

Cellular function depends on dynamic interactions and nanoscale spatial organisation of proteins. While transcriptomic and proteomic methods have enabled single-cell profiling, scalable technologies allowing high-resolution analysis of protein interactions at omics-scale are lacking. Here we present the Proximity Network Assay (PNA), a DNA-based method for constructing three-dimensional nanoscale maps of 155 proteins in single cells without the use of optics.

PNA employs barcoded antibodies and in situ rolling circle amplification to generate >40,000 spatial nodes per cell, which are linked through proximity-dependent gap-fill ligation and decoded by DNA sequencing, forming single cell Proximity Networks.

At an estimated spatial resolution of ~50 nm, PNA captures single-cell protein abundance, self-clustering, and colocalization, validating established cell membrane protein interactions and discovering novel ones as potential future targets of bispecific antibodies. We illustrate how PNA can be used to gain insights into the molecular mechanisms of cell function through protein interactions in hematological oncology, CAR-T cell therapies, and autoimmune disease.

29. Striking twice: unbiased IGF1R downregulation through GRK2-inhibition in colorectal carcinoma

Sylvya Pasca (sylvya.pasca@ki.se), Karolinska Institutet

Background and aim: Colorectal carcinoma (CRC) remains a leading cause of cancer-related morbidity and mortality worldwide, with liver metastasis significantly impacting its outcome. CRC is characterized by aberrant activation of various signal transduction pathways, creating redundant cancer-supportive networks. Despite evidence demonstrating CRC's reliance on a hyper-functional IGF1R system, clinical trials targeting IGF1R have failed.

A possible explanation could be that, although successful anti-IGF1R therapies downregulate the receptor, this process preferentially activates a parallel cancer-protective signaling, the β-arrestin-biased signaling.

Recently, we addressed this IGF1R targeting bottleneck through GRK2/β-arrestin system inhibition by paroxetine (PX). Given CRC's hyperactive signal network, our study aimed to investigate the impact of an 'IGF1R system bias therapy'. Results and methods: In a panel of human CRC cell lines, PX caused a dose-dependent decrease in cell viability. Mechanistic insights revealed that PX specifically downregulated and inhibited downstream signaling of GRK2-dependent RTKs (IGF1R), while sparing or enhancing the signaling capabilities of GRK2-independent InsR.

As InsR compensates the lost IGF1R signaling, we further evaluated additional targets, finding the greatest sensitivity displayed by PX combined with PI3K or MAPK inhibitors. In a 3D spheroid model, mimicking liver-like high IGF1, PX induced cell-death in the spheroid core but did not affect overall growth. However, double-hit treatment inhibited both growth and survival. These results were validated in vivo zebrafish xenografts, where the combination treatments restricted metastatic tumor growth. Conclusions: This study proposes a potent dual-hit strategy: the first hit (PX) targets IGF1R causing an oncogene addiction shift toward PI3K/ MAPK compensatory signaling pathways, while the second hit (PI3K or MAPK inhibitors) effectively disrupts the survival network.

30. Breaking new ground: Novel Preclinical models for Protac Evaluation

Takashi Willebrand (twillebrand@chempartner-eu.com), Chempartner

Targeted Protein Degradation (TPD) is an innovative therapeutic strategy that leverages the ubiquitin-proteasome system (UPS) to eliminate disease-relevant proteins. Key modalities in this field include proteolysis-targeting chimeras (PROTACs) and molecular glue degraders (MGDs). Unlike traditional occupancy-based inhibitors, PROTACs utilize an event-driven mechanism, enabling catalytic degradation of target proteins and expanding the therapeutic landscape to previously "undruggable" proteins. Despite their promise, PROTACs face significant development hurdles, including synthetic complexity, high molecular weight, limited cell permeability, and a constrained pool of E3 ligases. To address these challenges, ChemPartner has established an integrated preclinical platform featuring cost-effective, high-throughput in vitro and in vivo assays. This system supports structure-activity relationship (SAR) analysis and provides quantitative evaluations of efficacy, pharmacokinetics (PK), pharmacodynamics (PD), and toxicity. Our platform facilitates the rational design and optimization of PROTACs, accelerating development and reducing clinical attrition.

31. Electrophoretic Implants for in situ Precision Medicine*

Theresia Arbring Sjöström (theresia.arbring.sjostrom@liu.se), Linköping University

Biological barriers such as dense extracellular matrices, poor vascularization, and rapid clearance mechanisms all contribute to rendering large regions of tissue effectively inaccessible using systemic drug delivery approaches. Overcoming these barriers requires technologies capable of targeting defined tissue regions with high spatial and temporal precision, within effective yet safe dose levels. The central nervous system exemplifies the need for such disruptive approaches: the blood–brain barrier and delicate tissue structures prevent efficient access by conventional drugs, while neurodegenerative and oncological conditions demand precise modulation. We are developing drug delivery devices and implants that leverage polyelectrolyte hydrogels and low-current electronic control for selective transport of charged drugs. This electrophoretic approach enables programmable and durable in situ release without bulk solvent flow. It thereby minimizes off-target exposure, prevents pressure-induced tissue damage, and provides flexible temporal control, while integrating naturally with predictive computational modeling to optimize diffusional dose distribution. Using iontronic devices, we demonstrate high-precision local release of neurotransmitters and chemotherapeutics in preclinical models. These results lay the foundation for translation to more complex modalities, including in situ delivery of nucleic acid–based therapeutics, and point toward a pathway for targeting tissues and treating diseases today considered hard-to-treat or undruggable.

* Selected talk, presented on stage at 14:10

32. LigandTracer

Maria Winkvist (maria.winkvist@ridgeview.eu), Ridgeview Instruments AB

Highlight how emerging diagnostic frameworks can support the development and clinical translation of new modalities aimed at previously “undruggable” cancer targets.

33. The FragMAX platform for crystallographic fragment screening

Tobias Krojer (tobias.krojer@maxiv.lu.se), MAX IV Laboratory / SciLifeLab

The FragMAX facility supports crystallographic fragment and ligand screening at MAX IV Laboratory (Lund, Sweden), enabling the rapid identification of chemical starting points for drug discovery. The platform integrates four key components: (i) a high-throughput crystal preparation facility, (ii) a collection of fragment libraries, (iii) automated diffraction data collection at the BioMAX beamline, and (iv) software tools for large-scale data processing and analysis. Operational since 2019, FragMAX has established an international user program open to both academic and industrial research organizations. Through its modular and customizable workflows, FragMAX enables users with varying levels of expertise to routinely obtain high-quality screening hits for their targets.

34. NanoSIMS – A new tool to unveil drug targets in cells for drug discovery

Yanan Yang (yanan.yang@gu.se), University of Gothenburg

Intracellular drug concentrations and target engagement are critically important properties of a drug, however, difficult to assess in drug discovery. Recently, NanoSIMS has been presented as a powerful technique to visualize the uptake and biodistribution of drug, as well as to quantify its absolute concentration at the organelle level.

NanoSIMS is a unique secondary ion nanoprobe, capable of measuring elemental, isotopic, inorganic, organic, and biomolecular distributions with excellent sensitivity (ppm), and 2D/3D imaging with nanometer spatial resolution (50 nm laterally, 10 nm axially). NanoSIMS enables multiplexed detection of molecules at the subcellular level to reveal fundamental information of biological functions.

Here, we provide a few representative examples of NanoSIMS applications in pharmaceutical research to demonstrate the potential of the technology in drug discovery and development. Particularly, we demonstrate NanoSIMS imaging to measure the absolute concentration of an organelle-bound metabolite derived from L-DOPA drug in single organelles, to quantify the intracellular absolute concentration of oligonucleotides across organelles of human hepatocytes, investigate the impact of ligand-ASO conjugate stability on ASO subcellular

distribution, and to determine the uptake and localization of iron in lung macrophages at the organelle level.

NanoSIMS imaging, with a capability of correlating with other imaging techniques, offers a new window of opportunity to investigate drug pharmacokinetics and pharmacodynamics at the single cell level, delivering new insights into the drug trafficking and underlying mechanisms of endosomal escape.

35. Targeted and functional delivery of cargo through pseudo-typed extracellular vesicles

Zankruti Dave (zankruti.dave@ki.se), Karolinska Institutet

The delivery of a macromolecular drug into a cell is hindered by the cellular and endosomal membranes. Extracellular vesicles (EVs) are well suited as transport and delivery vehicles; however, we are yet to discover the mechanisms which dictate their delivery to a specific cell type. Interestingly, viruses have evolved to be extremely efficient in overcoming cellular defenses against nanoparticle delivery. We have thus genetically engineered EVs to express modified targeted viral fusogenic proteins which can achieve specific functional delivery of cargo only to the cells of choice. As a proof of concept, using this strategy, we have been able to deliver functional cargo specifically to Her2+ve cells with an efficiency of up to 80%, even in cases when the Her2 positive cells constitute only 10% of the total cell population. This is comparable to the efficiency of VSVG pseudo-typed EVs which are extremely efficient at delivery (above 90%) but are not specific and hence will deliver even to wild type cells. The unique aspect of our drug delivery system is that it can be customized to target different cell types or deliver different cargoes. Using pseudo-typed nanoparticles, we have screened multiple viral fusogens and multiple receptors for efficient delivery and observed varying efficiencies of delivery to activated, as well as non-activated T cells in cultured human PBMCs, with efficiencies around 42% and 8,5%, respectively. In contrast VSVG had a delivery efficiency of 50% in activated T-cells whereas it failed to deliver in non-activated cells. I am currently optimizing the strategy for the targeted delivery of CRISPR-Cas9 using pseudo-typed EVs to these and other cell types. Our goal is to ultimately target neuroblastoma cells, but because our delivery system is modular, we can apply this technology to any target of choice.

36. Advancing Companion Diagnostics to Enable Next-Generation Oncology Therapies

Christer Ericsson, PhD, Founder and Chief Scientific Officer, SOMA Genomics AB, Solna, Sweden

Next-generation oncology therapies increasingly aim to address targets historically considered “undruggable.” To realize their full clinical potential, companion diagnostics must evolve beyond single-biomarker tests and provide a more complete picture of treatment-relevant tumor biology.

This poster outlines how advances in diagnostic frameworks can enable patient stratification for emerging therapeutic modalities, including targeted protein degraders, RNA-based therapeutics, and engineered cell therapies. By guiding therapies to patients most likely to benefit while avoiding unnecessary treatment in others, such diagnostics can improve both clinical outcomes and safety — thereby increasing the effective therapeutic index of next-generation oncology therapies.

The work highlights the role of diagnostics as a critical enabler for drugging the undruggable: not only in matching therapies to the right patients, but also in ensuring that innovations are accessible, scalable, and aligned with regulatory and health technology assessment requirements.

Research Infrastructure Posters

A. Clinical Genomics Platform

Marcela Dávila (marcela.davila@gu.se), Clinical Genomics

Our platform offers a comprehensive suite of services aimed at revolutionizing diagnostics and treatment strategies through genomic analysis.
Our specialized equipment and analytical frameworks enables us to translate new high-throughput techniques into clinical use.

We play a key role as trainers and teachers for healthcare professionals and clinical researchers in the field of genomic medicine/precision medicine.

B. DDD Biophysical screening and characterization unit

Annette Roos (annette.roos@icm.uu.se), DDD Platform

The Biophysical Screening and Characterization (BSC) Unit of the SciLifeLab Drug Discovery and development (DDD) Platform supports drug discovery projects at all stages, from hit identification to lead optimisation and development. We also support target validation, feasibility studies and in the platform's antibody projects have assisted in epitope mapping. Our main methods are SPR biosensor technology, for mechanistic and kinetic analysis of molecular interactions, and X-ray crystallography, for structural analysis of ligand-target interactions.

We have the Prometheus Panta instrument for protein quality assessment and thermal melting profiling (nanoDSF and dynamic light scattering) and a Microscale Thermophoresis instrument for equilibrium-based interaction analysis.

The DDD platform supports projects wishing to develop proximity inducing agents and at the BSC unit we have an instrument called heliX+ that can measure ternary complex formation.

C. Multi-Modal Mechanism-of-Action Elucidation at SciLifeLab

Bernhard Schmierer (bernhard.schmierer@scilifelab.se), Karolinska Institutet

The SciLifeLab platform Chemical Biology and Genome Engineering unites three specialized units that together provide a multidisciplinary environment for discovery and innovation. The platform integrates expertise in chemical biology, chemical proteomics, cell painting, and CRISPR-based functional genomics, creating a versatile set of approaches for investigating the biological effects of small molecules. By combining unbiased phenotypic profiling, genome-wide perturbation technologies, and proteome-wide target engagement methods, we aim to reveal both direct molecular targets and the broader pathways that govern compound activity.

A central focus of our work is the integration of multi-modal datasets to generate testable hypotheses, uncover mechanisms of action, and accelerate target identification. This strategy not only supports the development of high-quality chemical probes but also contributes to drug discovery efforts by linking compounds to their biological context. Applications span from fundamental studies of cellular mechanisms to the identification of candidate therapeutic targets in complex disease settings. By providing access to cutting-edge technologies and expert guidance, the platform empowers both academic and industrial researchers to advance chemical tools into meaningful biological and translational insights.

D. Scilifelab Proteomics Platform

Ronald Sjöberg (ronald.sjoberg@scilifelab.se), KTH / SciLifeLab

E. SciLifeLab CMI Platform

Rebecca Howard (rebecca.howard@scilifelab.se), KTH / SciLifeLab

The SciLifeLab CMI Platform offers experimental, technological and analytical support for cellular and molecular imaging beyond the scope of standard university resources.

F. Display and Selection Technologies

Mikael Mattsson (Mikael.mattsson@immun.lth.se), Lund University

A SciLifeLab unit for antibody research, supporting discovery and development of therapeutic antibodies. Part of the Drug Discovery and Development platform (DDD) within SciLifeLab.

G. CRISPR Functional Genomics facility

Soniya Dhanjal (Soniya.Dhanjal@ki.se), Karolinska Institutet / SciLifeLab

At CRISPR Functional Genomics (CFG), we are specialists in CRISPR technology. We are a National SciLifeLab facility that offers the entire range of CRISPR applications, from the creation of knock-out/knock-in cell models to massively parallel, genome-wide perturbation screens. We strive to make the latest technological innovations available to the Swedish research community as quickly as possible.

Examples of our toolbox include base- and prime-editing, pooled CRISPR loss- and gain-of-function screens, and coupling pooled screens with single cell transcriptomics. One emerging focus area is target identification and mode-of-action elucidation, which we pursue in collaboration with the Chemical Biology Consortium Sweden (CBCS) and the Chemical Proteomics unit at SciLifeLab.

H. The SciLifeLab Bioinformatics Platform NBIS

Pär Engström (par.engstrom@scilifelab.se), NBIS, SciLifeLab & Stockholm university

I. Chemical Biology Consortium Sweden

Anna-Lena Gustavsson (anna-lena.gustavsson@ki.se), CBCS

J. OligoNova Biology - a Drug Discovery and Development Unit

Mario Ruiz (mario.ruiz@gu.se), OligoNova (SciLifeLab DDD / GU)

OligoNova offers state-of-the-art drug discovery infrastructure and expertise to researchers and supports the translation of fundamental research findings into patient-benefitting therapeutic molecules.

OligoNova Biology primarily provides bioinformatics and hands-on assays required to progress the oligonucleotide drug discovery and development programs, but can also provide services to academic and external researchers.

K. OligoNova Hub - Chemistry Unit

Dmitri Ossipov (dmitri.ossipov@gu.se), OligoNova Hub

OligoNova Hub - Chemistry Unit supports in design and synthesis of therapeutic oligonucleotides targeting RNA and proteins. We have expertise in development of oligonucleotide-based molecular tools for in vitro cellular assays and in vivo imaging as well as up-scale production of oligonucleotides for pilot in vivo studies.

L. SciLifeLab Genomics platform

Magnus Lundgren (magnus.lundgren@scilifelab.se), SciLifeLab Genomics platform

M. From assay design and validation to automation and scale - enabling identification and prioritization of new dryg candidates

Mathias Färnegårdh (mathias.farnegardh@scilifelab.se), Scilifelab, DDDP, BCA

N. Medicinal Chemistry, Hit2Lead, Solna, Drug Discovery and Development platform, Science for Life Laboratory

Ylva Gravenfors (ylva.gravenfors@scilifelab.se), Medicinal Chemistry, Hit2Lead, Solna, Drug Discovery and Development platform, Science for Life Laboratory

P. Integrated Structural Biology

Cecilia Persson (cecilia.persson@nmr.gu.se), University of Gothenburg

The Integrated Structural Biology (ISB) Platform offers insight at the atomic level by hands-on support within Structural Proteomics, Cryo-EM and Nuclear Magnetic Resonance. We work closely together with the bioinformatics platform (NBIS) and Chemical Biology Consortium Sweden (CBCS), as well as infrastructures outside SciLifeLab, such as Protein Production Sweden (PPS), Biophysical characterization (ProLinC), MAX IV and ESS. Together, we can guide you to the optimal and necessary technique/s for your scientific question within Structural Biology and support you throughout your project.

Q. Spatial Biology Platform

Katarina Tiklova (katarina.tiklova@scilifelab.se), SciLifeLab