POSTER ABSTRACTS

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Prague & Bohemia, 10.–13.6. 2025

POSTER NUMBER: 1

Exploring Germline-Dependent Inheritance Bias

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Nearly 160 years ago, Gregor Mendel's studies of trait segregation established the foundational principles of inheritance, positing random and independent segregation of alleles during gamete formation. Here, we report a skew in the sex-biased inheritance of synthetic heterozygous loci in Caenorhabditis elegans that violates Mendelian expectations. Extending previous observations, we show that crosses between wild-type hermaphrodites and males heterozygous for an integrated transgenic array consistently yield F1 progeny with significant deviations from the expected 1:1 inheritance ratio. Male progeny inherit the transgenic array more frequently than hermaphrodites, which preferentially inherit the non-transgenic homolog. We are currently elucidating the mechanisms of this skew combining genetic tools, different array compositions, and cytological approaches. As most known parent-of-origin effects are maternal, these results highlight an underappreciated role of paternal inheritance in shaping genetic transmission.

POSTER NUMBER: 2

Evolution of cytoplasmic intermediate filaments and invertebrate epithelial morphogenesis

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Proper development requires careful coordination of mechanical and biochemical processes. Alterations to either processes could impact the final form of an organism, but we understand relatively little on how changes in the molecular factors relate to altered mechanical responses. We hypothesize that cytoplasmic intermediate filaments (cIFs), a diverse cytoskeletal protein family, could link sequence evolution to morphogenic evolution. cIFs have greatly diversified across the animal kingdom, with different cIFs affecting epithelial spreading, an important process in animal morphogenesis, in varying manners. As a part of the HSFP team to bridge cIF sequence evolution with evolution of tissue morphogenesis, my project aims to understand the effects of individual cIF networks on tissue mechanics. To achieve this, I will be ectopically expressing individual invertebrate cIFs in Drosophila melanogaster, a cIF-free system, and disrupting endogenous cIFs in selected invertebrate species. By collecting data on the effects of varying cIF composition on tissue stiffness, tension, and migratory capabilities, I could understand the effects of the cIF networks on tissue mechanics and possibly animal form.

POSTER NUMBER: 3

A systematic approach uncovers new regulators of peroxisome abundance

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Peroxisomes are dynamic organelles essential for lipid metabolism, reactive oxygen species detoxification, and the synthesis of key cellular compounds, making them central in maintaining cellular balance. Given their broad functional impact, peroxisome abundance is highly regulated and deviations from optimal numbers can be detrimental to cells. Peroxisome levels can be controlled through a coordinated interplay of biogenesis, proliferation via fission, and selective degradation by pexophagy. Disruption of this balance can result in peroxisomal loss and lead to severe clinical disorders, yet the mechanisms that maintain this balance are still not clear. Here, we applied a high-throughput, microscopy-based whole-genome screening approach in baker's yeast (*Saccharomyces cerevisiae*), to identify new proteins involved in peroxisome abundance regulation. Follow-up work allowed us to differentiate between candidates affecting general autophagy and those specifically impacting peroxisome abundance. We focused on one such hit, identifying it as a novel peroxisomal protein with broad effects on peroxisome physiology and dynamics. Our work supports the effort of elucidating the regulatory mechanisms that govern peroxisome number and function in the cell.

POSTER NUMBER: 4

Extrathymic Aire-expressing cells control mucosal antifungal immunity by direct antigen presentation

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The autoimmune regulator (AIRE) has a fundamental role in the reinforcement of the central tolerance. AIRE deficiency leads to Autoimmune Polyendocrine Syndrome type-1 (APS-1), a severe autoimmune disorder that is often accompanied by chronic mucocutaneous *Candida* infections. While the mechanisms behind autoimmunity stemming from AIRE deficiency are well established, the susceptibility of APS-1 patients to fungal infections remains incompletely understood. In recent years a rare population of lymph node-resident extrathymic Aire-expressing cells (eTACs) has been identified. This population is characterized by the expression of Roryt and high levels of MHCII, costimulatory and fungal sensing molecules. Focusing on *Candida* infections associated with AIRE deficiency, we have previously demonstrated that Roryt+ eTAC-intrinsic Aire expression is critical for the induction of an effective *Candida*-specific Th17 response during gastrointestinal *Candida* colonization. Here we show that direct antigen presentation by eTACs is required for this process. Mice with MHCII-deficiency on Roryt+ cells exhibit significantly reduced numbers of *Candida*-specific T cells. Additionally, we present scRNAseq analysis of early innate interactions upon *Candida* colonization in both immunocompetent and Aire-deficient contexts. Our preliminary data suggest that upon *Candida* stimulation Roryt+ eTACs secrete molecules with the capacity to activate or attract mononuclear phagocytes (MNPs). We hypothesize that Roryt+ eTACs may cooperate with MNPs in lymph nodes, forming signalling hubs that support antifungal Th17 cell polarization.

POSTER NUMBER: 5

Identification and characterization of novel interacting partners of RNA polymerase in Mycobacterium smegmatis

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Abstract:

Transcription regulation in bacteria requires coordinated activity of DNA-dependent RNA polymerase (RNAP) and associated transcription factors. While our understanding of bacterial transcription largely derives from model organisms such as *Escherichia coli* and *Bacillus subtilis*, species-specific transcriptional adaptations remain poorly characterized. Mycobacteria, for instance, employ unique transcription factors such as CarD and RbpA- absent in *E. coli* - to modulate their gene expression. Here, using *Mycobacterium smegmatis* as a non-pathogenic model for *M. tuberculosis*, we aimed to identify novel mycobacterial transcriptional regulators. FLAG-tagged RNAP pull-down assays in *M. smegmatis* revealed interactions with a diverse set of proteins, including ribosomal subunits, providing direct evidence of transcription-translation coupling in mycobacteria. In addition, we identified several previously uncharacterized RNAP-associated proteins (UCPs). Among these; UCP1, which coexists with the nucleic acid clearance factor HelD, is essential for viability; and UCP7 associates with membrane architecture, suggesting a role in cell envelope biogenesis or maintenance. These findings expand the known mycobacterial transcriptional network and uncover novel factors critical for bacterial gene expression and survival.

Keywords: Transcription factor; Uncharacterized protein; RNA polymerase; Mycobacteria

This project was supported by the Grant Agency of the Czech Republic, GAČR 210379; and the Grant Agency of Charles University in Prague; GA UK 236823.

POSTER NUMBER: 6

Are You Stressed, ER? Host Membrane Remodeling and Lipid Droplets Accumulation During Human Polyomavirus BK Infection

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BK polyomavirus (BKPyV) is a small, nonenveloped human DNA virus that establishes lifelong, asymptomatic infections in the urinary tract of 80% of adults worldwide. However, it can reactivate under immunosuppression, leading to severe diseases such as BKPyV-associated nephropathy and hemorrhagic cystitis. Moreover, it can contribute to the development of urothelial tumors, particularly bladder cancer. During virus reactivation, renal proximal tubular epithelial cells (RPTECs) become the primary target; however, human bladder microvascular endothelial cells (HBMVECs) have been identified as viral reservoirs. Recently, we used HBMVECs model to investigate the molecular mechanisms of BKPyV replication and cell architecture remodeling. Notably, we have described the BKPyV-induced presence of cytoplasmic vacuoles, virus-loaded tubular reticular structures, neutral lipid droplet accumulation and endoplasmic reticulum (ER) remodeling. These phenomena are often associated with the induction of ER stress, which can be triggered by various conditions, such as the accumulation of misfolded proteins, calcium imbalance or membrane remodeling often induced as a response to viral infections. To restore ER homeostasis and enable cell survival, the cell activates the unfolded protein response (UPR). However, the severe and prolonged UPR results in programmed cell death. Numerous studies have demonstrated the pivotal role of ER stress and UPR pathways in the tumor development and uncontrolled cell proliferation in various types of malignancies, including urothelial carcinoma. We hypothesize that BKPyV manipulates UPR pathways to create a microenvironment that supports the development of BKPyV-associated malignancies. Our ongoing studies aim to determine the activity of individual UPR pathway branches during BKPyV infection of RPTE and HBMVE cells. This research could pave the way for targeted antiviral strategies.

POSTER NUMBER: 7

The Biomarkers of Senescence and Infertility in Aging Canines.

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Male infertility is a rising crisis across many species and populations around the world, with potentially catastrophic consequences for animal conservation and husbandry. Understanding the various phenotypes that can occur within ejaculates from a range of individuals of the same species may help develop strategies to combat this issue.

One area of infertility that has garnered recent research interest is the competition between sperm within the same ejaculate—specifically, the impact of sperm cell lifespan on both the fertility of the ejaculate and the fitness and health of potential offspring.

Male age is a significant factor associated with increased infertility and adverse effects on offspring across various species, including humans, bovines, canines, and others. Many hallmarks of cellular senescence observed in somatic cells—such as elevated levels of the γH2AX histone, increased histone methylation, and higher rates of DNA fragmentation—have also been correlated with negative impacts on sperm fertility.

This project focuses on the levels of three specific biomarkers in the sperm of canines of varying ages at the time of cryopreservation. The biomarkers include γH2AX histone as an indicator of senescence, H3K27Me3 to assess histone methylation, and DAPI to evaluate DNA fragmentation. The study examines how these markers are co-expressed in sperm populations from differently aged canines and their relationship to the longevity of these populations.

POSTER NUMBER: 8

Protein modifications and ionic strength show the difference between protein-mediated and solvent-mediated regulation of biomolecular condensation

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A significant amount of biomolecules organize themselves in an aqueous environment by forming so-called biomolecular condensates. Condensates are structures created through transient, multivalent interactions between intrinsically disordered proteins. The formation of such condensates involves extensive overlapping of the hydration shells of individual molecules, expulsion of water from the intermolecular interfaces, and confinement of the interstitial water between the biomolecules. This work aims to find out how these three processes contributing to the dynamics of the hydrogen bond network within biomolecular condensates are modulated by co-solutes and amino acid level chemical modifications of the protein. The effects of addition of post-translational (added in the cell after the polypeptide chain is synthesized) modifications and salts on the hydrogen bond network dynamics inside condensates formed by the Fused in Sarcoma (FUS) protein have been studied using Terahertz (THz) spectroscopy. This spectroscopic method probes the intermolecular hydrogen bonding network of water and reports on the water hydrating the protein. A comparison of spectra of the FUS protein with and without PTMs at 100 mM and 2.5 M KCl. The phase behavior of FUS shows a non-monotonic trend with respect to salt concentration, where the protein undergoes a salt-dependent reentrant phase transition. Comparison of THz spectra between the droplets formed in the low ionic strength and high ionic strength regime show a significant change in the amount of hydrophobic hydration water and the stiffness of the water network. Post-translational modifications were found to decrease the propensity of the protein to phase separate, however without significant changes to the behavior of water in the condensates. These findings show, that the hydration network within biomolecular condensates is more strongly affected by the chemical composition of the solvent than by biologically introduced chemical modifications of the protein.

POSTER NUMBER: 9

Can RNAi responses be artificially transferred between individuals?

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In C. elegans, RNA interference (RNAi) can be triggered by double-stranded RNA (dsRNA) that is injected or fed to the nematodes. Within the worm, the RNAi response is amplified by RNA dependent RNA polymerases that produce abundant small RNAs using the target gene as template. The silencing can spread between tissues and gets inherited across generations. Still, the exact molecular identities of the heritable small RNA molecules are not fully characterized. We are trying to artificially transfer silencing-responses between worms by extracting RNA from the descendants of donor worms that were injected or fed with dsRNA and injecting it to naive recipient worms. Clues arising from our experimental system will hopefully facilitate the elucidation of the characteristics and limitations of the mediators which transfer the information between tissues and transgenerationally.

POSTER NUMBER: 10

Basement membrane lama3 times epithelial cell renewal in the gut

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Homeostasis in the intestine is kept by the constant renewal of the cells of the intestinal epithelium, which takes between 5 to 7 days. Cell renewal is orchestrated by a tightly regulated balance between epithelial cell proliferation within the intestinal crypt, cell death and extrusion at the villus tip and an active directional migration of differentiated cells from the crypt towards the villus tip. However, the mechanisms that regulate the rate of cell turnover and maintain homeostatic cell numbers along the crypt-villus axis remain poorly understood.

A key but often overlooked factor in this process is the basement membrane (BM), a specialized extracellular matrix that underlies the intestinal epithelium. Its molecular composition is not uniform across the crypt-villus axis and changes during intestinal development, homeostasis, and disease. Yet, whether these regional variations in the BM serve as extracellular regulators of epithelial cell behavior remains unclear.

We hypothesize that the regionalized expression of laminin-332, specifically restricted to the villus tip under homeostatic conditions, plays a regulatory role in epithelial cell renewal by controlling cell extrusion. Using an in vivo knockout mouse model and live imaging of tissue explants, we found that loss of *Lama3*, which encodes the α-chain of laminin-332, leads to increased epithelial cell extrusion and an overall acceleration of the renewal rate of the epithelium while maintaining cell differentiation and organization across the crypt-villus axis. Using both 2D and 3D organoid cultures, we further observed that cell-matrix adhesion dynamics and cell extrusion rates vary with laminin concentration, suggesting that alterations in cell adhesion at the villus tip disrupt normal extrusion dynamics and ultimatly influencing the overall rate of cell renewal.

Our results identify **lama3** as a key regulator of epithelial homeostasis, acting as a timer that coordinates cell turnover through adhesion-mediated control of cell extrusion.

POSTER NUMBER: 11

Strange RNAs in unexpected places - exploration of noncanonical small RNAs in neuroblastoma

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While constructing a unified atlas of transcriptomic states in neuroblastoma, a lethal childhood cancer, from available single cell data, we stumbled on a publication describing the existence and function of Piwi interacting RNA (piRNA) in chicken neural crest (Galton etl al.). Driven by curiosity, and the fact that neuroblastoma derives from an incomplete transformation of neural crest into the adrenal gland, we have looked at the expression of the PIWI proteins in the newly formed single cell atlas. To our surprise, PIWI proteins were not only expressed in neuroblastoma, but showed cancer subtype specific expression, and their expression correlated with the activity of MYCN - a known driver of oncogenesis. Due to us working in computational biology, and therefore being wet-constrained, this would have been the end of our explorative journey. However, we have found an amazing resource by Misiak et al., who have profiled small RNA from neuroblastoma tumor samples from 120 patients. The study by Misiak et al. however, focused only on describing the relationships of microRNAs (miRNAs) with patient phenotypes. We have thoroughly explored the small RNAs, and found that many loci in access of miRNAs, that contribute to the heterogeneity of the small RNA in neuroblastoma, with many detected loci overlapping with previously described piRNAs. We have explored the associations of the newly detected small RNA with patient subclassification and looked at the specificity of their expression across different cancers.

POSTER NUMBER: 12

Genetic adaptations to increasing aridity across plants and animals

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The ongoing intensification of global aridity, driven by climate change, poses major challenges to both natural ecosystems and agricultural productivity. Rising temperatures, longer droughts and declining water availability threaten the survival of many species and exacerbate water scarcity in crops, reducing yields. In this context, understanding the genetic basis of adaptation to arid environments is critical for both biodiversity conservation and the development of resilient agricultural systems. In this work, we first focus on the Barn Swallow (*Hirundo rustica*), an iconic long-distance migrant songbird with six described subspecies that differ in body size, ventral colouration, tail feather length and migratory behaviour. We conducted a resequencing project using individuals from two migratory European populations (*H. r. rustica*) and a sedentary Israeli population (*H. r. transitiva*). We found little genetic differentiation, with the two European populations behaving as a panmictic population. Interestingly, genome-wide selection and differentiation scans revealed candidate genes previously implicated in adaptation to hot and dry environments. Selective pressure may therefore already be acting on genomic regions involved in high temperature tolerance in these species.

In plants, we aim to develop resilient crop varieties that are better adapted to water scarcity, starting with tomato (*Solanum lycopersicum*). To this end, we have used Crispr-Cas9 technology to generate mutant lines of 4 clade A protein phosphatase 2C (PP2C-A) genes, which act as negative modulators of the ABA signalling pathway, on the assumption that combinatorial inactivation of PP2Cs could generate a more favourable trade-off between growth and drought resistance, adapted to current climate change conditions. We are now exploring the impact of this variability through high-throughput phenotyping of T2 plants to select advantageous combinations of pp2c alleles. Hopefully, our work will provide insight into evolutionary strategies for coping with aridity and highlight opportunities for sustainable agriculture under future climate scenarios.

POSTER NUMBER: 13

A tale about mind-bending parasites

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We all live with parasites – on us, in us, in our food and water. They come in all shapes and sizes, from microscopic to meters long. Parasites are the pinnacle of evolution. I know, we all think that we are the pinnacle of evolution, but evolution has selected the parasitic life over the free-living one. There are more species of parasites than there are free-living organisms on Earth. They have been living with us forever and they have shaped the way we have evolved. Our immune system exists, in large part, to deal with them, and has been shaped as we have been exposed to them. Parasites influence where we live and how we live. They changed our behaviour. I am going to challenge you change the way you think about them. Herein, I will present my “Big Five” mind-bending parasites I have encountered during my research and share my fascination for parasites and their importance for ecosystems, heath and conservation.

POSTER NUMBER: 14

Making Cancer Cells Visible: Enhancing Immune Recognition Through MHC-I Regulation

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Cancer immunotherapy's effectiveness depends on immune cells recognizing and eliminating tumor cells. This recognition relies on cancer cells presenting antigens via major histocompatibility complex I (MHC-I) molecules, yet cancer cells often evade immune detection by suppressing their antigen presentation machinery. Through genome-wide CRISPR/Cas9 screening, we identified a complex network of MHC-I regulators operating at multiple cellular levels - epigenetic, translational, and post-translational - including both known and previously uncharacterised factors. By targeting these newly discovered MHC-I repressors, we achieved significant upregulation of MHC-I surface levels, making cancer cells more sensitive to elimination by CD8 T cells.

While these findings reveal mechanisms controlling overall MHC-I expression, we now aim to understand how cancer cells selectively present specific antigens on MHC-I molecules. Using two complementary approaches - functional T cell-mediated killing assays and innovative sortase-mediated labeling (uLIPSTIC) - we aim to identify the mechanisms that allow cancer-specific antigens to be preferentially presented on MHC-I over other antigens. This comprehensive analysis of both global MHC-I regulation and selective antigen presentation will reveal new strategies to enhance the visibility of cancer-specific antigens to the immune system, potentially improving the precision and effectiveness of cancer immunotherapy.

POSTER NUMBER: 15

What even is an amyloid?

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Amyloids are best known for their role in diseases like Alzheimer's and Parkinson's, where they form sticky plaques in the brain. But not all amyloids are bad. Some are functional—forming highly organized and stable structures that certain organisms put to good use.

Bacteria, for instance, use amyloids to build resilient biofilms. *Pseudomonas* species produce the amyloid protein FapC, which acts as a scaffold in their biofilm matrix, while *Staphylococcus aureus* secretes phenol-soluble modulins (PSMs) that can form cytotoxic amyloid-like fibrils involved in infection and immune evasion. Functional amyloids also appear in hormone storage in higher organisms.

Antimicrobial peptides (AMPs) have a surprising twist: some can also self-assemble into amyloid fibrils. In this study, we investigated one such AMP—a 22-amino-acid peptide flagged by our in-house prediction tool as a likely fibril-former. And it didn't disappoint. The peptide assembled into well-defined fibrils in both pure water and physiological buffer.

Using cryo-electron microscopy, we solved three fibril structures, including two formed under near-physiological conditions at resolutions of up to 2.2 Å. These fibrils displayed unexpectedly complex and diverse architectures, revealing just how structurally versatile short peptides can be.

Our findings offer a glimpse into the broader universe of functional amyloids and highlight how even tiny sequences can self-assemble into intricate nano-architectures. It's a beautiful reminder that biology is full of surprises—especially at the molecular level.

POSTER NUMBER: 16

Annelid methylomes reveal ancestral developmental and aging-associated epigenetic erosion across Bilateria

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DNA methylation in the form of 5-methylcytosine (5mC) is the most abundant base modification in animals. However, 5mC levels vary widely across taxa. While vertebrate genomes are hypermethylated, in most invertebrates, 5mC concentrates on constantly and highly transcribed genes (gene body methylation; GbM) and, in some species, on transposable elements (TEs), a pattern known as “mosaic”. Yet, the role and developmental dynamics of 5mC and how these explain interspecies differences in DNA methylation patterns remain poorly understood, especially in Spiralia, a large clade of invertebrates comprising nearly half of the animal phyla. Here, we generate base-resolution methylomes for three species with distinct genomic features and phylogenetic positions in Annelida, a major spiralian phylum. All possible 5mC patterns occur in annelids, from typical invertebrate intermediate levels in a mosaic distribution to hypermethylation and methylation loss. GbM is common to annelids with 5mC, and methylation differences across species are explained by taxon-specific transcriptional dynamics or the presence of intronic TEs. Notably, the link between GbM and transcription decays during development, alongside a gradual and global, age-dependent demethylation in adult stages. Additionally, reducing 5mC levels with cytidine analogs during early development impairs normal embryogenesis and reactivates TEs in the annelid *Owenia fusiformis*. Our study indicates that global epigenetic erosion during development and aging is an ancestral feature of bilateral animals. However, the tight link between transcription and gene body methylation is likely more important in early embryonic stages, and 5mC-mediated TE silencing probably emerged convergently across animal lineages.

POSTER NUMBER: 17

Wnt-Notch Crosstalk in Embryonic Epidermal Development: Insights from Xenopus Embryos

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Cells in multicellular organisms communicate through intricate signaling networks, including Wnt and Notch pathways, which regulate critical processes such as growth, metabolism, and response to stimuli. Dysregulation of these pathways can lead to diseases, including cancer. While Wnt and Notch signaling are known to interact, the precise molecular mechanisms remain unclear. Here, we investigated the role of key Wnt components - Dishevelled, Casein kinase 1 alpha (CK-1α), and β-catenin - in regulating Notch signaling through its intracellular domain (NICD). Using proteomic and microscopic approaches in *Xenopus laevis* epidermis, we identified novel interactions between these components, providing new insights into the crosstalk between Wnt and Notch pathways.

POSTER NUMBER: 18

Knocking out co-active plasticity rules in neural networks reveals synapse type-specific contributions for learning and memory

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Synaptic plasticity is thought to underlie learning and memory. Plasticity rules can be active in different synaptic connection types, such as excitatory-excitatory (EE) and inhibitory-excitatory (IE) connections, with evidence suggesting distinct mechanisms for each type. Studies typically probe only one type of plasticity at a time. Thus, the contributions of connection types and co-active rules to memory processes remain unclear. Here, we used a recent dataset of co-active Hebbian STDP and rate-dependent plasticity rules meta-learned to maintain homeostasis in large recurrent spiking networks. The baseline-configured spiking network comprises plastic EE, EI, IE, and II connections, each active with a different plasticity rule that produces stable activity. To investigate how sets of rules govern network computations, we simulated each set of co-active rules in a familiarity detection task. First, we present a specific stimulus multiple times. Following a delay period, we evaluate memory performance by presenting familiar and novel stimuli. Memory retention is quantified as the difference in population firing rate between stimuli. After assessing memory performance, we knock out plasticity in either one or three rules for the duration of the task, allowing us to evaluate each rule's contribution. We show that there are synapse type-specific contributions to stability and memory retention. Blocking plasticity in IE connections disrupts network stability in over one-third of knockout simulations, such that networks do not display cortical-like asynchronous-irregular firing dynamics. Blocking EE plasticity usually allows memory retention during replay but results in highly variable population firing rates in response to novel and familiar stimuli. Our results suggest that EE plasticity plays a crucial role in stabilizing pattern recognition and separation, linking specific synapse types and rules to network computation.

POSTER NUMBER: 19

Evolutionary trade-offs between intergenerational and transgenerational fitness effects

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Intergenerational and transgenerational fitness effects can shape evolutionary processes. Theoretically, however, intergenerational and transgenerational effects can trade-off with each other with profound consequences for population dynamics. Here we show that beneficial intergenerational effects that increase offspring fitness can result in detrimental transgenerational effects that decrease great-grand offspring fitness. We used theoretical and experimental techniques to investigate transgenerational fitness effects of larval starvation in Caenorhabditis elegans. We show that individuals who undergo larval starvation exhibit large decreases in multiple fitness estimates. However, regardless of offspring environment, their immediate descendants gain fitness benefits whilst great-grandchildren suffer fitness costs. Through simulating boom-and-bust population dynamics, we find an adaptive evolutionary strategy where beneficial intergenerational effects trade-off with detrimental transgenerational effects to increase fitness of a genotype across many generations. Our findings suggest that such multigenerational trade-offs will play an important role in evolution and challenge the view that transgenerational effects are necessarily adaptive.

POSTER NUMBER: 20

Exercise Training Reverses Age-Related Epigenetic Changes in Transcription Factors to Preserve Skeletal Muscle Health

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Age-related decline in skeletal muscle function represents a major biomedical challenge, with epigenetic modifications such as DNA methylation (DNAm) emerging as central players in this process. Transcription factors (TFs) are critical regulators of gene expression and muscle homeostasis, yet their activity and regulation are disrupted during ageing, leading to impaired mitochondrial function, metabolic dysregulation, and structural deterioration. Understanding the interplay between DNAm and TF regulation in ageing is essential for developing effective strategies to combat muscle decline and promote healthy ageing.

Using a combination of open-access and in-house DNAm datasets, we identified TFs with altered methylation patterns in skeletal muscle associated with both ageing and exercise. Bioinformatic analysis revealed significant hypo- and hyper-methylation events, with six key TFs (AR, FLI1, ETS1, ERG1, KLF9, and CEBPA) displaying opposing methylation patterns between ageing and exercise.

Our findings demonstrate that exercise training modulates the expression and epigenetic regulation of these TFs, potentially reversing age-related changes and preserving muscle function. These results underscore the therapeutic potential of exercise in attenuating muscle decline and extending health span. By elucidating the epigenetic mechanisms underlying the benefits of exercise, this work paves the way for innovative interventions to promote healthy ageing and improve quality of life.

POSTER NUMBER: 21

Cognitive biases in academic career decision-making: A self observational study with interactive peer contribution

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Cognitive biases are unconscious and systematic errors in thinking that occur when people process and interpret information. While cognitive bias is frequently discussed in research methodology, there is a notable gap in addressing how these biases affect academic career decisions, from lab selection and project management to career trajectory planning.

Through retrospective analysis of a 5-year PhD experience, I identified instances of authority bias, optimism bias, effort justification, sunk cost fallacy, and other biases that influenced critical career decisions. Each bias was analyzed for situational context, observable but ignored or overlooked warning signs, and potential intervention points where alternative actions could have been taken.

This analysis is compiled into a practical infotable, including red flag checklists and actionable decision-making strategies. Conference attendees are invited to participate by contributing their own experiences and alternative strategies directly on the poster or using sticky notes.

The goal is to spread awareness about cognitive biases and to help fellow scientists make more informed career decisions that can be summarized as: I effed up so you don´t have to.

POSTER NUMBER: 22

Retapamulin-assisted ribosome profiling uncovers small proteins and mechanistic aspects of translation initiation and regulation in Staphylococcus aureus

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Staphylococcus aureus is a major opportunistic human pathogen and represents a significant burden to healthcare systems worldwide. To adapt to stresses encountered in its host and environment, *S. aureus* employs a plethora of regulatory networks, acting both on the transcriptional and post-transcriptional level, involving trans acting small RNAs (sRNAs), mRNA elements, RNA-binding proteins and modifications on tRNAs and rRNAs. Indeed, many examples of such adaptive regulation have been characterized by our group, highlighting the importance of translational control in these processes.

Recent advances in antibiotic-assisted ribosome profiling have uncovered previously unnoticed translation events in bacteria, including upstream open reading frames (uORFs) and novel small ORFs. By improving retapamulin-assisted ribosome profiling (Ribo-Ret) in *S. aureus*, we achieved unprecedented resolution of initiation sites, revealing diverse regulatory mechanisms. This high-resolution approach allowed us to identify a new toxin-antitoxin (TA) system, uORFs that may modulate translation in response to environmental conditions, and many novel small proteins. Additionally, we could describe *S. aureus*-specific translation initiation features, revealing mechanistic differences in start codon recognition among bacterial ribosomes.

While our map of small proteins is likely to be of significant interest to the community studying the molecular biology and pathogenicity of *S. aureus*, our primary focus is on elucidating the mechanisms of translation regulation. Concentrating on regulatory initiation sites, uORFs and the study of a new TA system, we continue to study these intricate mechanisms as well as the species specificity of bacterial start codon selection.

POSTER NUMBER: 23

IAA modulates metabolism in liver cells

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Indole-3-acetic acid has been studied for many years as a key plant auxin hormone. However, this tryptophan derivative is also found in abundance in another significant area: the mammalian gut. Intestinal bacteria produce it, after which it travels to the liver via the portal vein. The levels of microbiota-derived IAA vary between health and disease. Its beneficial effects remain unclear, but rodent experiments suggest that it may alleviate negative symptoms in liver inflammation-related diseases caused by eq. excessive fat deposition (steatosis), ethanol consumption, or acetaminophen overdose.

We decided to investigate the mechanism of IAA action in liver cells. First, we screened how this metabolite affects the transcriptome. We discovered changes in genes involved in mitochondrial metabolism, prompting us to conduct quantitative microscopic imaging of mitochondria. Our results indicate that IAA is a metabolism regulator that should not be overlooked when considering the impact of microbial metabolites on human health.

POSTER NUMBER: 24

Modifying the Code: RNA-Modifying Enzymes as Architects of Embryonic Translation

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The regulation of gene expression during development is governed by complex, multilayered mechanisms, among which chemical modifications of RNA have emerged as pivotal modulators. Advances in epitranscriptomics have revealed RNA modifications as dynamic regulators of gene expression, yet their direct impact on translational control during cell fate transitions remains poorly understood. Recent studies suggest that these epitranscriptomic marks may act as a dynamic code, enabling cells to rapidly and precisely adjust protein synthesis in response to developmental cues. We hypothesize that RNA modifications and their associated enzymes form an integrated regulatory network that modulates the efficiency and fidelity of translation, thereby critically influencing lineage specification and cellular identity. Our aim is to systematically dissect the contribution of RNA-modifying proteins to translational control during differentiation, with the goal of uncovering how the epitranscriptome shapes developmental trajectories. By elucidating these mechanisms, this research seeks to establish RNA modifications as a central regulatory layer in development and to provide new conceptual frameworks for understanding and manipulating cell fate decisions.

POSTER NUMBER: 25

Structural characterzation of JASPer protein and its complex with JIL-1 kinase

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The recognition of specific posttranslational modifications on histone tails is an evolutionarily conserved mechanism that plays a critical role in regulating transcription in eukaryotic cells. The coordinated deposition of these modifications helps safeguard gene coding regions from transitioning between the transcriptionally active euchromatin state and the repressed heterochromatin state. Many histone-modifying enzymes lack chromatin-binding domains and therefore depend on interacting partners that recognize and target them to specific chromatin regions.

One notable example is the phosphorylation of serine 10 on histone H3 (H3S10), which is essential for mitosis and also plays a crucial role in maintaining euchromatin during interphase. In *Drosophila*, this modification is catalyzed by the JIL-1 kinase, which lacks a chromatin reader domain. To localize to active chromatin and ensure its stability, JIL-1 relies on an interaction with the chromatin-associated factor JASPer. JASPer binds to H3K36 di- and trimethylated histones and is structurally similar to the human transcription elongation factor LEDGF.

Both JASPer and LEDGF feature an N-terminal PWWP domain responsible for chromatin binding, followed by a central intrinsically disordered region, and a C-terminal structured domain that serves as a binding platform for proteins containing the F-X-G-F interaction motif. AlphaFold structural predictions suggest that JASPer's C-terminal region harbors an additional putative domain and exhibits notable differences compared to the TND (TFIIS N-terminal Domain) fold found in LEDGF, which is characterized by a five-helix bundle. The most striking divergence is the presence of an extended helix within JASPer's TND domain, along with the partial integration of the TND-interacting motif (TIM) directly into JASPer's C-terminal domain. These structural differences suggest that the interaction interface between JIL-1 and JASPer is distinct from previously characterized TND-TIM module structures, which are coordinating transcription elongation in humans.

We employed a combination of structural biology and biochemical approaches to reveal important structural features of JASPer and its complex with histone kinase JIL-1 in the chromatin context.

POSTER NUMBER: 26

Do yeast cells dream of metabolic sheep?

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Everything oscillates, especially in biology. When yeast cells are grown to high cell density in liquid culture, they tend to synchronize their metabolic activity exposing an otherwise hidden layer of cellular dynamics. Known as respiratory oscillations, due to varying oxygen concentration as our major read-out, these dynamics are in line with an emerging picture of a fundamentally periodic nature of cell growth, where multiple distinct pulses of growth (translation!) can occur independent of and upstream of the cell division cycle. After these growth pulses, we observe a phase of little metabolic activity. The ATP/ADP ratio, pH and metabolic heat production reach minima, i.e., cells literally cool down. Both promoter and gene body chromatin undergo genome-wide reset points. K+ concentration peaks. The remaining low-level ATP synthesis is mostly used for build-up of storage material (glycogen or lipid droplets).

Here, I speculate that this phase may not only be analogous but homologous to human sleep. Newly synthesized proteins and other macromolecular structures can self-assemble, the cytoplasm becomes granular. In the nucleus, DNA torsional stress from the previous phase of strong transcription can be resolved, and loci of increased stress can be marked and adapted. Thus, this phase is akin not only to sleep but even involves 'dreaming' where the activities during the previous growth pulses are echoed in DNA structure, and integrated into a new chromatin state, i.e., epigenetic memory can be established. Transposons (mobile regulatory elements) may target such stressed loci and thereby buffer transcription-induced stress during subsequent growth pulses, and provide a substrate for the rewiring of transcriptional regulation.

POSTER NUMBER: 27

Regulation of mRNA Homeostasis by Poly(A) Binding Proteins

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Homeostatic mechanisms enable cells to adjust mRNA abundance to maintain a constant concentration. RNA polymerase II (Pol II) activity has been shown to directly respond to perturbations of mRNA homeostasis, yet growing evidence indicates a feedback mechanism that coordinates mRNA synthesis and degradation, allowing cells to buffer mRNA concentration.

For the coordination of these processes, I hypothesise the existence of a “sensor” that “measures” mRNA concentration and conveys this information to the transcription and/or degradation machinery.

Cytoplasmic poly(A)-binding proteins (PABPC1, PABPC4) are highly conserved across eukaryotes, and specifically bind to polyadenylated RNA, regulating stability, translation, and degradation. Several properties of PABPC1 and PABPC4, including strong affinity for mRNA, translational autoregulation, nuclear import dependent on cytoplasmic mRNA abundance, and inhibitory effect on Pol II activity, make them ideal sensor candidates that report information on mRNA concentration into the nucleus to regulate transcription. These properties have been demonstrated in response to viral infection, yet it is unclear whether similar mechanisms control mRNA homeostasis in physiological conditions.

My project aims to understand the role of PABPCs in regulating mRNA homeostasis in mammalian cells. I will combine multiplexed fluorescent imaging, RNA sequencing, and biochemistry with mathematical modelling to test this mechanism and determine its key properties. This research will provide new insights into fundamental homeostatic mechanisms used by eukaryotic cells to regulate gene expression.

POSTER NUMBER: 28

An alternative end of the story: how do CPEBs act on neuronal RNA metabolism?

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RNA, like all of us, has a life cycle. In the nucleus, it is transcribed, spliced, and modified with a 5' cap and a 3' poly(A) tail before being exported to the cytoplasm for translation. The poly(A) tail promotes translation and stability but is gradually shortened through deadenylation, a rate-limiting step for RNA decay. However, in specific contexts, this tail is elongated via *cytoplasmic polyadenylation*, a process mediated by CPEB proteins. CPEBs bind target RNAs and, upon activation, recruit factors that extend the poly(A) tail, enhancing translation. This mechanism has been proposed as essential for synaptic plasticity - an underlying process in learning and memory -where localized translation at synapses modifies neuronal connectivity. However, recent high-resolution sequencing studies challenge this model. While CPEB-mediated polyadenylation is clear in germ cells and development, its presence in neurons is less certain. To investigate, we applied multiple RNA-sequencing methods to analyze mRNA metabolism upon neuronal activation. Surprisingly, we found that a subset of rapidly induced genes showed early poly(A) tail elongation, but this elongation was transcription-dependent. Moreover, CPEB knockout increased RNA levels of the same genes, suggesting a regulatory role distinct from polyadenylation. So, what is the role of CPEBs in neurons? Revisiting this question may uncover key molecular mechanisms behind learning and memory. The synapse is the structure-in-motion *par excellence*, constantly adapting to stimuli, reshaping itself, and triggering precise molecular responses. It is only thanks to recent advances in sequencing technologies that we can now observe these spatio-temporal dynamic processes with unprecedented resolution.

Be the best worst Reviewer #2 of my poster and win the prize!

POSTER NUMBER: 29

Melanosomes role in melanoma immunity

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Melanoma, a neoplasm of melanocyte origin, is the most lethal of human skin cancers, with 100,000 new cases per year. Despite progress in melanoma therapy, including immunotherapy, 50% of patients are treatment-resistant, with most experiencing disease recurrence. Given that tumor cells often express altered or neo-antigens and are recognized by TILs, it remains unclear why immunotherapy approaches do not eliminate tumors in more patients. Melanosomes are large EVs (200-500 nm) specifically synthesized by melanocytes and are responsible for transporting melanin pigment to neighboring epidermal cells as protection against UV-induced DNA damage. Interestingly, melanoma retains the capacity to produce melanosomes, though the underlying reason remains largely unknown. Previous works showed that melanosomes are secreted in close proximity to melanoma, promoting metastatic niche generation. Further, these EVs contribute to melanoma drug resistance by exporting chemotherapeutic agents. However, whether there is a direct interaction between melanosomes and T cells has not yet been investigated. Our preliminary findings suggests that melanosomes inhibits tumor-infiltrating cytotoxic T cells activity and effectiveness. Our study reveals a novel immune-evasion mechanism of melanoma tumors utilizing melanosomes and proposes a therapeutic avenue to enhance tumor immunity.

POSTER NUMBER: 30

Probing the duality of temperature and osmotic strength on developmental tempo using deep learning

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The tempo of embryonic development is highly sensitive to environmental conditions. Recent influential work has proposed a duality of temperature and osmotic strength on cellular function, and suggested that solvent thermodynamics driven by changes in water availability regulate protein activity and macromolecular interactions. However, this hypothesis has not been tested in the context of complex developing multicellular systems, and the relative contributions of temperature and osmotic strength on the regulation of developmental tempo remain unclear. To address this question, we developed zMorphoNet, a deep learning-based tool for detailed morphometric analyses across developmental stages. zMorphoNet combines semantic and instance segmentation for accurate tissue identification and was trained and validated using manually annotated zebrafish datasets. Our tool enables detailed morphometric analyses across developmental stages and precisely delineates embryonic structures, including the yolk, yolk extension, cells, embryonic body, and eyes. By applying zMorphoNet to embryos exposed to varying environmental conditions, we quantified tissue-specific growth rates and morphological changes with unprecedented precision. We found that temperature consistently dominated the regulation of developmental tempo in zebrafish embryos, while modulating osmotic strength had more subtle effects on tissue boundary integrity and morphogenetic processes. These findings suggest that temperature-driven solvent thermodynamics play a primary role in regulating the biochemical activity essential for developmental progression. By combining deep learning with experimental manipulations, our study underscores the utility of zMorphoNet for quantifying tissue dynamics and developmental tempo in complex biological systems.

POSTER NUMBER: 31

piRNA and the Case of the Missing Penis

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We study small RNAs—mostly piRNAs—in terrestrial slugs. These animals have a surprisingly simple RNA silencing machinery: one Dicer, two Argonautes, and two Piwi proteins. At the same time, their genomes are packed with repeats: in *Deroceras* around 48% of the genome is repetitive, and about 20% are LINEs. Slugs also show diverse reproductive strategies—some reproduce uniparentally, while others outcross—which makes them a good system to study how piRNA pathways adapt.

We assembled and annotated genomes of two species from the genus *Deroceras*—one with a penis and one without. We predicted piRNA clusters and found that most piRNAs come from around 20–30 clusters. Interestingly, slugs that outcross have a higher fraction of piRNAs, suggesting stronger piRNA activity. In ovotestis, piRNAs target LINEs and DNA transposons more or less equally, while in somatic tissues they mostly go after DNA transposons. Who knows—maybe DNA transposons are somehow involved in the loss of penis?

We also looked at the genome of *Arion vulgaris*—the big brown invasive slug you might know because it ate your garden. It has a penis and reproduces by outcrossing, but a bit more evolutionary distant.

Come to my poster if you want to hear a ukulele song about slugs and piRNAs!

POSTER NUMBER: 32

RPE Culture Model for Studying Dry AMD and Drusen Formation

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Dry age-related macular degeneration (AMD) is marked by RPE degeneration, Bruch's membrane thinning, and drusen formation containing hydroxyapatite (HAP). Despite progress in identifying risk factors, the mechanisms behind drusen formation remain unclear, and no effective treatment exists for dry AMD. In vitro models are needed to study its pathogenesis and screen potential therapies.

We developed a dry AMD cell model using primary porcine RPEs cultured under four conditions: porous polycarbonate (PC) membranes and plastic plates, each with or without vitronectin coating. RPE morphology was monitored, and cells were collected at weeks 1 and 8. Alizarin Red S (ARS) staining was used to detect HAP, followed by dye quantification and molecular analysis.

RPEs on PC membranes maintained healthy morphology, showed no HAP accumulation, and had high TEER (>500 $\Omega \cdot cm^2$). In contrast, plastic surfaces induced hypertrophy, hyperpigmentation, cell death, and significant HAP deposition (average 1.5 mM ARS), along with downregulation of RPE markers, including BEST1.

Our findings suggest that porous PC membranes support physiological RPE behavior, while plastic surfaces induce dry AMD-like changes. This model offers a promising platform for studying AMD mechanisms and therapeutic development.

POSTER NUMBER: 33

Classification of ligand-binding sites for Alphafold models of human proteins

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Proteins are flexible and can adopt different 3D conformations to react to a change in conditions of an environment or to carry out their function. Each of their binding sites often has at least two distinct states - one binding ligand (holo state) and one ligand-free (apo state) . Recent advances in protein structure prediction allow the generation of a 3D structure prediction for any protein sequence – they do not, however, describe what conformational state of protein is predicted. Here, we took advantage of the recently developed Ahoj-DB to identify all human proteins with the experimentally solved 3D structures for both apo and holo states. As proteins often contain multiple binding sites, these apo-holo relationships were established in AHoJ-DB for each site. We compared the structures of those proteins with their models from the AlphaFold Protein Structure Database (AFDB) to investigate what are suitable metrics for annotation of apo/holo states and whether we can describe models from the AFDB as apo or holo with respect to a given binding site.

POSTER NUMBER: 34

Blinatumomab and Tyrosine Kinase Inhibitors in B-ALL: friends, or enemies?

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Background:
Blinatumomab (CD3/CD19) is a T cell engager used in relapsed/refractory B-ALL. It often pushes leukemia out of the marrow into tissues, hinting at changes in cell adhesion and homing. Tyrosine kinase inhibitors (TKIs)—mainly dasatinib and ponatinib—are standard for Ph+ B-ALL, targeting BCR-ABL and Src-family kinases. Now, TKIs should theoretically block T cell activity by hitting Src kinases, making their combo with blinatumomab counterintuitive. But we thought—is it really *that* simple?

What we did:
We treated Ph- B-ALL cell lines (RAJI, REH, ARH-77) and healthy donor T cells with blinatumomab ± TKIs. We looked at killing efficiency, cell adhesion molecule expression, and pro-survival pathway activation using flow cytometry and immunoblotting. To peek into metabolism, we also ran Seahorse assays on the leukemia cells.

What we found:
TKIs at high (but still clinical) doses dampen blinatumomab-driven T cell cytotoxicity (as expected). On their own, they don't kill leukemia cells but do hit key growth signaling (Erk, Akt). They also tweak CD62L (a cell adhesion molecule) levels, suggesting altered adhesion, and therefore migrating into tissues they should not really migrate into. TKIs also reduce oxygen consumption in B-ALL cells without majorly affecting glycolysis, which is weird, since inhibition of SFKs usually does the exact opposite.

Takeaway:
TKIs may indeed destroy blinatumomab's best work, but they clearly mess with leukemia cell behavior in interesting ways—affecting signaling, metabolism, and maybe even where these cells like to hang out. Theoretically, the combo may therefore be useful even in Ph- B-ALL. Work in progress. More digging needed.

Supported by the Ministry of Health of the Czech Republic, grant no. NU23J-03-00033.

POSTER NUMBER: 35

Novel Mouse Models of ALGS- and BA-Associated Jag1 Variants Exhibit Distinct Vascular Phenotypes Without Affecting Experimentally Induced Cholestasis

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Jagged1 (Jag1), a key ligand in the Notch signaling pathway, is essential for proper liver development. Loss-of-function mutations in *JAG1* underlie approximately 94% of Alagille syndrome (ALGS) cases, and interestingly, distinct *JAG1* variants have also been identified in patients with biliary atresia (BA). In particular, the ALGS-associated (R1097W) and BA-associated (R1213Q) missense mutations both localize to highly conserved regions within the intracellular domain of JAG1, suggesting potential functional significance. To assess their in vivo consequences, we generated C57BL/6 mice homozygous for each variant (*Jag1RW/RW* and *Jag1RQ/RQ*). Both mutants exhibited striking, divergent alterations in hepatic vascular architecture that were more pronounced than the effects on bile duct (BD) morphology—and neither developed overt cholestasis. Specifically, *Jag1RW/RW* neonates displayed a marked reduction of hepatic arteries (HAs) and disorganization of the morphology of BDs along the portal triads, whereas *Jag1RQ/RQ* animals showed an increased number of HAs with only minor BDs morphological changes. *In vitro*, neither variant impaired JAG1-NOTCH2 transactivation in cultured cells—even when co-expressed with the E3 ubiquitin ligase Mib1—indicating preserved canonical Notch signaling. Moreover, acute cholestatic injury induced by a single biliatresone dose was comparable across wild-type and mutant mice. Our data reveal that these two *Jag1*ICD variants selectively perturb liver vascularization, possibly via a mechanism distinct from canonical Notch activation and independent of susceptibility to biliatresone-driven cholestasis. We, therefore, hypothesize that these *Jag1*ICD variants uncover a previously unrecognized, Notch-independent function of Jag1 in hepatic vascular development, acting along a different axis than that engaged by biliatresone-induced injury.

POSTER NUMBER: 36

Regulating the Giants: Single-Molecule Insights into the Transcription and Processing of Large mRNAs in Muscle Cells

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The regulation of large mRNAs presents unique biological and energetic challenges, especially in muscle cells where some of the most extreme gene architectures exist. My PhD project aims to explore how mammalian cells manage the transcription, processing, and localization of exceptionally large RNA molecules—focusing on dystrophin (DMD) and titin (TTN). These two genes represent contrasting regulatory paradigms: Dystrophin, with one of the longest mRNAs in the genome and extremely large introns, versus Titin, encoding the largest known protein but with an unusually compact and exon-rich mRNA structure.

Using single-molecule imaging approaches in mouse Myod1-overexpression models and differentiating human iPSCs, I aim to dissect the temporal and spatial dynamics of these transcripts during myogenic differentiation. Key questions include: How are these long mRNAs transcribed over 18+ hours? Are splicing and nuclear export initiated co-transcriptionally? What mechanisms coordinate their energy-intensive expression, and how has evolution shaped their architecture for efficient regulation? Additional the role of tranlation of large proteins from hugh mRNAs could be a objective of interest.

Through live-cell RNA imaging, fixed-cell smFISH, and transcriptomic analyses, this project will shed light on fundamental principles of large mRNA biology, with potential implications for muscle development, disease, and RNA therapeutics.

POSTER NUMBER: 37

The mechanisms that lead to the polarization of microbiota-specific T cells into intraepithelial lymphocytes

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Segmented filamentous bacteria (SFB) are Gram-positive, clostridia-like commensal bacteria that have evolved a unique life strategy involving close interaction with the host epithelial cells of the small intestine. Under homeostatic conditions, SFB remain non-pathogenic and they are controlled by the immune system - primarily through Th17 cell response - which prevents them from penetrating the epithelial barrier and causing pathological inflammation. While the Th17-mediated immune response to SFB has been well characterized, other responses induced by SFB in the host intestine remain poorly understood. Our recent findings revealed the additional role of immune system in response to SFB, which involves the conversion of SFB-specific CD4+ T cells into granzyme-expressing intraepithelial lymphocytes (IELs). Although the molecular pathways behind IEL differentiation are increasingly understood, the specific cell types and signals that drive this process remain largely elusive. To clarify these mechanisms, we aim to investigate the origin and fate of SFB-specific $CD4^+$ T cells that are reprogrammed into cytotoxic IELs. Furthermore, we will dissect the roles of distinct antigen-presenting cell subsets and the molecular cues that mediate this tissue-specific adaptation. These findings will contribute to a deeper understanding of host gut microbiota-mediated cellular regulation.

POSTER NUMBER: 38

Exploring mechanical landscape of living embryonic tissues with magnetic droplets

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The mechanical properties in the cellular microenvironment have been shown to fundamentally influence cell behaviors, including cell proliferation, cell migration and cell differentiation, suggesting that material properties and their changes in space and time play an important role in embryonic tissue morphogenesis. In order to quantitatively measure material properties, our laboratory has developed a technique based on biocompatible ferrofluid droplets as local mechanical actuators that allows spatiotemporal investigation of mechanical properties directly within living embryos. Using this technique, we were able to demonstrate that zebrafish body elongation involves spatially varying tissue mechanics along the anteroposterior axis (1). Subsequently, we were able to demonstrate how mechanical transitions, such as fluid-to-solid phase behavior, underlie large-scale tissue flows and shape changes during embryonic development (2). Moreover, we were able to reveal an important role of the cell nucleus in tissue mechanics (3). Overall, magnetic droplet actuation provides a minimally invasive quantitative technique that offers unprecedented insight into the mechanics of living tissues thus creating a bridge between biology and physics.

(1) Serwane, F., Mongera, A., Rowghanian, P. *et al. In vivo* quantification of spatially varying mechanical properties in developing tissues. *Nat Methods* **14**, 181–186 (2017). https://doi.org/10.1038/nmeth.4101

(2) Mongera, A., Rowghanian, P., Gustafson, H.J. *et al.* A fluid-to-solid jamming transition underlies vertebrate body axis elongation. *Nature* **561**, 401–405 (2018). https://doi.org/10.1038/s41586-018-0479-2

(3) Kim, S., Amini, R., Yen, ST. *et al.* A nuclear jamming transition in vertebrate organogenesis. *Nat. Mater.* **23**, 1592–1599 (2024). https://doi.org/10.1038/s41563-024-01972-3

POSTER NUMBER: 39

The Plastid-Encoded RNA Polymerase: When and Why Did It Become So Complex?

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Plastid genomes have dramatically reduced in size compared to their cyanobacterial ancestors, yet paradoxically retain a complex Plastid-Encoded RNA Polymerase (PEP). In land plants, PEP comprises a bacterial-like core and 15 accessory subunits, known as PAP1–15, whose evolutionary origins and functions remain poorly understood. Here, we report that the PEP from *Chlamydomonas reinhardtii*, a simple unicellular photosynthetic eukaryote, forms a ~2 MDa complex that also includes 12 previously unknown nuclear-encoded accessory subunits, which we named PEPS1-12. A 2.7 Å cryo-EM structure reveals that most PEPS proteins occupy positions analogous to land plant PAPs and contribute to stabilizing the extended catalytic core. Remarkably, however, they lack detectable sequence or structural homology—with two notable exceptions. These findings demonstrate that the complexity of PEP originated before the evolution of multicellularity and terrestrial plants, challenging long-held assumptions. Moreover, the elaborate structural architecture of this essential enzyme supports a model of constructive neutral evolution, suggesting that its complexity may have arisen not from direct functional advantages, but rather from the gradual fixation of neutral genetic changes that created essential interdependencies—ultimately stabilizing a core transcriptional complex vital for the growth of green algae and plants, and more broadly, for sustaining oxygenic life on Earth.

POSTER NUMBER: 40

Adaptive Fragility: Evolution of Antibiotic Tolerance by Disruption of the Cellular Network

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The evolution of tolerance, which slows bacterial death in antibiotics, is not well understood. Tolerance may result from increased dormancy, which protects bacteria from many antibiotics. Indeed, in evolution experiments under antibiotic treatment mutants with dormancy-mediated tolerance evolve rapidly. Increased dormancy also occurs in bacteria recovering from non-adapted growth-arrest, where it has been attributed to overwhelmed regulatory mechanisms sending cells into a default, random, disrupted state. We e explore theoretically this unique evolutionary mode using a random-network model of the disrupted state and experimentally verify resulting predictions. One such prediction is increased susceptibility of tolerant strains to various bactericidal agents, suggesting means of thwarting this evolutionary trajectory.

POSTER NUMBER: 41

Visualising experience-induced gene expression at single-molecule resolution in the memory centre of fly brains

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Long-lasting memories are thought to be encoded by molecular changes in neurons that modulate the strength of specific synaptic connections. These neuronal changes are believed to be initiated through rapid transcriptional induction of immediate-early genes (IEGs) in response to neuronal activity. However, different IEGs have distinct expression patterns and dynamics, making it unclear how exactly they contribute to memory formation in the brain. Therefore, we set out to visualize these transcriptional changes in memory-relevant neurons, both in fixed brains and live flies, and understand how they contribute to the writing of a memory.

Visualizing several IEGs using single-molecule fluorescence in situ hybridization (smFISH) revealed that while some IEGs are broadly expressed in the fly brain, others are strongly and selectively induced in a small population of neurons after stimulation. The most responsive IEG in fly brains is *Hr38*, the *Drosophila* ortholog of the mammalian IEG NR4A. In addition to stimulus-dependent induction of transcription, we identified that *Hr38* undergoes an additional layer of co-transcriptional regulation. We find that a large fraction of transcription initiation events are terminated prematurely, and that this process involves the polymerase-associated multi-subunit Integrator complex. Knockdown of the catalytic subunits of the Integrator complex by RNAi increases the number of neurons expressing *Hr38* and the number of *Hr38* transcripts produced after stimulation.

In addition to visualizing induced changes in gene expression in fixed tissues, we have developed RNA stem loop tools to visualize endogenous gene expression in live fly brains. We aim to correlate transcription dynamics with stimulus-induced calcium transients in live animals for *Hr38*, as well as other IEGs similarly upregulated after experience. In the case of *Hr38*, dual-colour live imaging with two orthogonal sets of stem loops, one in the first intron, the other in the 3' UTR, will be crucial to understand the dynamics of its expression. The two sets of stem loops will allow us to visualize both transcription initiation and production of the full-length mRNA, allowing us to investigate its co-transcriptional attenuation.

POSTER NUMBER: 42

The hidden metabolism of food additives: microbial breakdown of azo dyes in the gut

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Synthetic azo dyes are widely used as food additives to provide colour and enhance the food's attractiveness to consumers. These food dyes were approved as safe for consumption by the European Food Safety Authority (EFSA) based on studies evaluating their direct toxicity and allergenicity (doi:10.2903/j.efsa.2010.1778). However, with the growing recognition of the gut microbiome's importance, concerns have emerged that food additives that were once considered harmless may interact with microbiota, potentially disrupting intestinal homeostasis. Although intact azo dyes are not easily absorbed into the blood stream, there is evidence suggesting that gut bacteria can reduce azo dyes to potentially toxic aromatic amines and sulfanilic acid (doi:10.1016/j.anaerobe.2023.102783). The rate at which these metabolites are then absorbed or how they affect the immune environment in the gut is currently unknown, however, there is some evidence linking Red 40 (E129) consumption with intestinal autoimmune disorders (doi:10.1016/j.cmet.2021.04.015). The goal of our research is to identify the bacterial species that can degrade azo dyes and asses the effect of their metabolites on other microbiome components and the intestinal immune environment. Through screening bacteria isolated from various regions of the murine intestine, we found that the efficiency of azo dye degradation varied among bacterial species. While some dyes, such as Amaranth (E123) and Brilliant Black BN (E151), were broadly degraded across multiple species, others, like Tartrazine (E102), were reduced by only a select few. Currently, we are working on identifying those bacterial species and the enzymes responsible for dye reduction. In the future we plan to investigate whether these metabolites can pass through intestinal epithelium and be absorbed into the blood stream, followed by evaluating their effect on the intestinal immune cells *in vivo.*

POSTER NUMBER: 43

Where transcription meets translation

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Transcription and translation are key steps in gene expression. Although these processes are functionally distinct, they occur in close coordination, ensuring efficient gene expression and rapid adaptation to environmental changes. In many species these processes are physically coupled. This transcription-translation coupling plays a crucial regulatory role and contributes to the efficient processing of genetic information, allowing bacteria to quickly respond to external stimuli and adapt to changing conditions. However, this coordination between transcription and translation is not present in all bacteria. *Bacillus subtilis* is an example of a bacterium in which RNA polymerase (RNAP, the key enzyme of transcription) is faster than the ribosome, and this interaction does not occur.

Elongation factor Tu (EF-Tu) belongs to the group of translational GTPases and plays key role in translation. It binds and transports aminoacyl-tRNA to the ribosome. Moreover, EF-Tu has additional functions. EF-Tu interacts with the MreB protein, helping maintain the cell shape. Phages Qβ and MS2 use EF-Tu as a subunit of their RNA replicases for their proper function. EF-Tu also functions as a molecular chaperone. Finally, EF-Tu was shown to increase resistance of the translational apparatus to antibiotics tetracycline, streptomycin, spectinomycin, and erythromycin.

During studies of *B. subtilis* transcription, we identified that EF-Tu possibly associates with RNAP.

Here, we will present results from co-immunoprecipitation and western blot showing the interaction between EF-Tu and RNAP. We monitored the interaction in different growth phases and in rich and defined/minimal media. Furthermore, we will present *in vitro* transcription results addressing the effects of EF-Tu on RNAP.

This novel interaction suggests a coordinated regulation of transcription and translation, providing new insights into the complexity of gene expression regulation in *B. subtilis*. Further research into this interaction could uncover additional regulatory mechanisms and contribute to a broader understanding of bacterial gene expression.

The work was supported by the Czech Science Foundation project No. 23-06295S.

POSTER NUMBER: 44

A Minimal Mechanochemical Model for De Novo Patterning in Hydra Aggregates

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The interplay of chemical and mechanical information is central to animal development. The freshwater polyp Hydra vulgaris, with its remarkable regenerative capabilities, exemplifies how these signals orchestrate tissue patterning. While prior studies often used pre-patterned tissue fragments, our approach is based on using Hydra aggregates. These lack any initial pre-patterned polarity, thus being an ideal model system for studying de-novo patterning and the intrinsic mechanochemical feedback underlying this.

Here, we present a minimal physical model coupling Wnt3 signaling—a pivotal morphogen in Hydra and other animals—with the nematic alignment of actin fibers. On a spherical geometry, we explore how local morphogen concentrations and actin influence one another to drive axis formation and emergent patterning. Crucially, these theoretical predictions are grounded in our experimental observations from non-prepatterned Hydra aggregates, ensuring that the feedback mechanisms remain biologically relevant and testable.

By focusing on mechanochemical interactions between concentration gradients and nematic fields, this modelling approach will help us to explain the self-organizing potential of Hydra tissues in the absence of pre-existing cues.

POSTER NUMBER: 45

The importance of the epithelium-egg shell interaction in Drosophila gastrulation: uncovering morphogenetic instability through phenotypic variability

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Gastrulation is a complex and well-coordinated process that, through a precise combination of tissue rearrangements and cellular movements, leads to the segregation of the germ layers. Both individual cells and tissues change their relative positions over time, strongly influenced by the interaction with their surroundings. Of particular interest is the role played by the vitelline envelope (VE), the innermost layer of the eggshell enclosing many developing embryos. It has been shown that a conserved Integrin-mediated attachment of the blastoderm to the VE is required for proper gastrulation. In *Drosophila*, disrupting such attachment results in a twisted gastrulation (TG) phenotype. To quantify this phenomenon, we imaged the TG phenotype by high-resolution imaging in different genetic backgrounds. Surprisingly, we found a high variability in germ-band extension dynamics, even in wild-type. This suggests a mechanical instability of the germ-band, compensated by the blastoderm-VE interaction. To corroborate this experimental data, we are establishing a theoretical model to study the forces at play during germ-band extension. Quantifying the TG phenotype, we also detected a bias in the handedness of the twist. Moreover, comparing tissue deformation on the two sides of the midline, we observed a consistent left-right asymmetry across the samples. Based on this data, we developed the idea that left-right asymmetry could be at least partially set up already in the blastoderm, while the earliest developmental process showing chirality reported so far, gut formation, occurs hours later. We hypothesize that Scab-mediated attachment could be necessary to keep the germ-band symmetric during its extension.

POSTER NUMBER: 46

Tell me Y-BX1 binds RNA? Ain't nothing but another phase separating protein…

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YBX1 is a fascinating cold shock protein. Its folded cold-shock domain has been conserved from bacteria to humans. In humans, this domain is flanked by an intrinsically disordered region, allowing it to undergo liquid-liquid-phase separation (LLPS) and form dynamic, droplet-like condensates. So, this protein must be doing something important, one could think ! But wait ! There's more! What makes it even more interesting…drum roll…its interaction with RNA. In this study, we perform a series of experiments showing how RNA affects droplet formation of these proteins and how it modulates their material properties. We use FRAP to understand and compare the mobility of YBX1 with and without RNA. We perform temperature-dependent assays to see the effect of RNA on the dissolution and formation of YBX1 droplets. And to tie it all together, we quantify RNA binding affinity using fluorescence anisotropy and FCS. Altogether, our results highlight the importance of RNA in regulating YBX1 condensates in humans and raise the question: Why has evolution held on to this protein?

POSTER NUMBER: 47

Combining CARs and BiKEs on the road to treatment of Acute myeloid leukemia

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CAR-T cell treatment has proven to be a viable alternative to standard-of-care chemotherapy. However, some leukemia types, such as acute myeloid leukemia (AML), remain undefeated. This is caused by multiple factors, including finding a suitable target antigen or suppressive tumor microenvironment, which leads the CAR-T cells to inhibition and anergy. One of the possible approaches is further modification of the CAR-T cells in a way that would enable the harnessing of endogenous immune cells for the fight against cancerous blasts.
We have decided to try and make the CAR-T cells produce a bispecific antibody that on one side binds CD33, a myeloid-associated antigen commonly found on AML blasts, and on the other activates endogenous NK cells - a BIspecific Killer-cell Engager (BiKE).
After choosing the most suitable antibody, we modified PBMCs from a healthy donor to express an anti-CD123 CAR and, upon stimulation, the BiKE. We have tested the manufactured cells under several conditions, and the preliminary data show that when battling cancer, the combination of CARs and BiKEs seems to work well, unlike in traffic in Prague. NK cells derived from the same donor as the PBMCs have been shown to be more prone to activation, degranulation, and a longer-lasting effect when killing target cancer cells in the presence of CAR-produced BiKE than NK cells assisting only CAR-modified T cells. Moreover, the best-faring BiKE that we chose for our work is derived from a camelid scFv, giving our DNA construct the title of "alpaca CAR-BiKE," making it more patient-friendly in the case of a clinical trial.

POSTER NUMBER: 48

Syncytin-1: not a stromatolite, but still cool

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Before there were animals, plants, or even proper cells with nuclei, stromatolites were already here. Built by layers of cyanobacteria, they slowly grew in shallow seas, capturing sunlight and releasing oxygen into an atmosphere that was not ready for it. Around 2.4 billion years ago, this started what we now call the Great Oxidation Event (1), permanently changing the planet's atmosphere and surface chemistry. In a way, stromatolites made life possible.

Even though they are over 3.5 billion years old (2), stromatolites are still around. You can find them in a few places on Earth, like Shark Bay in Australia, quietly doing their thing. I find it fascinating that something so old, so simple, could leave such a huge mark on the planet. They didn't need to be complex or fast - they just kept going, layer after layer, for billions of years.

My poster was almost going to be about stromatolites. But unfortunately, I do not study them. I work on Syncytin-1 - a former retroviral envelope protein that mammals stole from a virus (3,4), and now use to build the placenta. It is not as old as stromatolites, but it's another strange story about how life reuses whatever it finds useful. In this poster, I will show how Syncytin-1 interacts with ASCT receptors (ASCT1 and ASCT2) to mediate cell fusion - a small example of how ancient viral genes are still shaping us today.

(1) Lyons et al., 2014, https://doi.org/10.1038/nature13068
(2) Nutman et al., 2016, https://doi.org/10.1038/nature19355
(3) Blond et al., 2000, https://doi.org/10.1128/jvi.74.7.3321-3329.2000
(4) Mi et al., 2000, https://doi.org/10.1038/35001608

The work was supported by the project National Institute of Virology and Bacteriology (Programme EXCELES, ID Project No. LX22NPO5103) – Funded by the European Union – NextGenerationEU and by Praemium Academiae Grant awarded by the Czech Academy of Sciences to JH.

POSTER NUMBER: 49

A comprehensive interrogation of transcription factors in mouse embryonic stem cells

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Mammalian genomes encode over 1,500 putative DNA-binding proteins, collectively known as transcription factors (TFs), to designate genes for coordinated expression. Despite the critical role of TFs in cell biology, the genomic locations of their binding sites and their interaction partners remain largely enigmatic. This is due to the limitations of state-of-the-art methods, which rely on the availability of specific, validated antibodies—a resource that is often scarce. To address this challenge, the Bühler lab has endogenously tagged all TFs (~ 800) expressed in mouse embryonic stem cells (mESCs) with the same V5-3xFLAG epitope. Leveraging this collection of cell lines, we are systematically mapping the interactomes and genomic binding sites of hundreds of TFs. This resource can be capitalised for functional interrogation and further characterisation of TFs. Here, I propose a strategy to utilize a degron fused to a nanobody that binds the V5 tag in the cell line collection of the Bühler lab. Depletion of any given TF in the mTFome is conceivable by this method for unveiling primary targets and dose-dependent action of TFs. The emergence of *de novo* protein design algorithms expands our repertoire of functionalised binders to allow the mapping of all protein and RNA interactors of a given TF with minimised background. Together, this work harnesses cellular engineering to investigate unresolved problems in gene regulation at a massive scale.

POSTER NUMBER: 50

HelD is a Transcription Factor with a Relationship to Antibiotic Resistance in Rapidly Growing Nontuberculous Mycobacteria

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The infections caused by nontuberculous mycobacteria have been on a rise in prevalence recently. Nevertheless, the information about the antibiotic sensitivity of different mycobacterial species remains limited. Here, we demonstrate that nontuberculous mycobacteria differ significantly in their sensitivity to rifampicin, the antibiotic which is predominantly used to treat their infections. Generally, slow-growing mycobacteria were more susceptible to rifampicin than fast-growing species. Vast majority of fast-growing mycobacteria encode HelD protein (also called HelR), which is a binding partner of RNA polymerase. On the other hand, most of the slow-growing mycobacterial species lack the HelD protein. We show that HelD is abundantly expressed in the fast-growing *Mycobacterium smegmatis*, even without any exposure to rifampicin, and that HelD has a global effect on transcription in this species. We propose a model in which HelD clears promoters blocked by stalled RNA polymerases during the exponential growth phase. This function enables mycobacteria not only to overcome rifampicin treatment but also to maintain rapid growth during the exponential phase, when the demands on RNA production are high and there is a busy traffic of RNA polymerases on the promoters. Consistent with this model, HelD is more prevalent in fast-growing mycobacterial species, which correlates with their higher growth rates and higher transcription rates, while in slow-growing mycobacteria, the HelD homologs are absent. HelD is an example of a gene with dual functions. We propose that its role in rifampicin resistance is a secondary effect of its primary function in transcription regulation. Additionally, we suggest that other genes similar to HelD are present in mycobacteria and may delay the effects of antibiotics, giving mycobacteria more time to develop antibiotic resistance.

POSTER NUMBER: 51

Two tales of using deep learning models for the study of motif-mediated interactions

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We present two proof-of-concept approaches exploring how deep learning models can study motif-mediated interactions in intrinsically disordered regions (IDRs).

First, we investigate whether AlphaFold2's (AF2) apparent "failures" when modeling flexible interactions might contain biologically meaningful information. By examining cases where AF2 produces problematic outputs—such as clashing structures or low-confidence models—we explore whether these results could reflect the inherent conformational heterogeneity of dynamic protein-protein interactions. Using selected examples, we examine how AF2's modeling of multiple binding sites within IDRs might be reinterpreted to provide insights into allovalent binding patterns.

Second, we explore the potential for protein language models (PLMs) to discover novel short linear motifs (SLiMs). As an alternative to traditional regex or PSSM approaches used in databases like ELM, we investigate how embeddings from PLMs might identify new SLiMs based on existing binding peptides. This approach tests whether sequence representations learned by PLMs can capture motif recognition patterns that complement conventional methods.

Both projects represent early-stage explorations of how reframing our interpretation of deep learning outputs—from structure prediction and sequence analysis—might offer new perspectives on peptide-mediated binding interactions that retain conformational freedom.

POSTER NUMBER: 52

Role of the microbiome in liver fibrosis and gut health

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Liver fibrosis is the accumulation of collagen in the liver due to injury and chronic inflammation. This causes the liver to stiffen and overtime lose its functions. The gut microbiome has been implicated in various liver and intestinal diseases, but its precise role in liver fibrosis progression remains unclear. It is known, that the decline in intestinal health exacerbates liver disease. It was shown that feces in humans with fibrosis contained more calories when compared to healthy individuals, suggesting absorption is affected by liver disease.

In my project, I aim to understand how microbial disfunction in the intestine exacerbates liver disease. I explore the impact of microbiome on the function of small intestine in the context of liver disease using two distinct murine models of liver fibrosis: thioacetamide (TAA)-induced liver injury and high-fat diet (HFD)-induced hepatic steatosis.

To check if intestinal architecture is a culprit behind changes in gut absorption, I am measuring villi length. Moreover, I am doing single cell transcriptomics to check the expression of transporters of specific metabolites. Metabolomics of portal blood and intestinal contents will also be done to estimate gut absorption efficiencies. To evaluate the role of the microbiome in this process, it will be depleted using antibiotics.

I hypothesize that in both experimental models, the altered microbiome will exacerbate liver fibrosis and alter gene pathways related to fibrosis and inflammation.

This study will provide valuable insights how microbiome drives liver and intestinal pathologies, revealing therapeutic targets. It aims to identify microbiome-dependent mechanisms in hepatic injury and metabolic dysfunction.

POSTER NUMBER: 53

Engineering Human iPSCs to Study RNA Localization and Translation in iNeurons

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Single-molecule live-cell imaging in immortalized cell lines, like HeLa, has advanced our understanding of the complex life cycle of mRNAs, but their genetic abnormalities and cancerous origin limit physiological relevance. To address this, we are transitioning to the human induced pluripotent stem cell (iPSC) line KOLF2.1J, which more accurately resembles normal human cells and can differentiate into nearly any cell type. We aim to engineer KOLF2.1J cells by integrating two distinct landing pads into safe harbor loci using CRISPR-Cas9, followed by the specific insertion of live-cell imaging components via recombinase-mediated cassette exchange (RMCE). These components include the MS2 system for tracking individual mRNA molecules and the SunTag system for monitoring translation of nascent peptides in real time. This highly modular system will allow us to study RNA dynamics in a more physiological context. Later, we plan to differentiate these cells into neurons to investigate RNA behavior in a more functionally complex cell type.

POSTER NUMBER: 54

Virus-like transposons transfer host genes across the species barrier and drive the evolution of novel genetic incompatibilities

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Horizontal gene transfer (HGT)—the movement of genetic material between species—has been reported across all major eukaryotic lineages. However, the underlying mechanisms of transfer and the impact on genome evolution are still poorly understood. While studying the evolutionary origin of a selfish element in the nematode *C. briggsae*, we discovered that *Mavericks,* ancient virus-like transposons related to giant viruses and virophages, are one of the long-sought vectors of horizontal gene transfer. *Mavericks* (*Polintons*) are flanked by terminal inverted repeats and can readily jump and insert into genomes, like transposons. But like viruses, they code for a large number of proteins, including a type-B DNA polymerase, a retroviral-like integrase, and capsid proteins. Using phylogenetics, structural predictions and genetic crosses, we discovered that two novel nematode gene families are preferentially taken up as cargo genes by *Mavericks* and have been extensively transferred between different nematode species on a global scale. Remarkably, many of these transfers occurred between species that last shared a common ancestor likely hundreds of millions of years ago. We also found that nematode *Mavericks* captured a novel fusogen, which is structurally similar to the glycoprotein B from *Herpes simplex virus 1.* This event likely fueled their spread via the formation of enveloped infective particles, analogous to the inception of retroviruses from genomic retroelements. Lastly, we show how the union between a horizontally transferred *wosp* protease, *msft-1*, and a MULE transposon gave birth to a novel class of selfish gene in *C. briggsae*: a mobile toxin-antidote element that causes powerful genetic incompatibilities that drive in wild populations. Our results identify the first wide-spread vector of HGT in animals and highlight how the intertwined biology of viruses and transposons can ultimately impact gene flow between populations, shaping the evolution of the species that carry them. While there are likely many different vectors of HGT in nature, we predict that *Mavericks*—analogous selfish genes with viral properties—could mediate HGT in other eukaryotic lineages. Current and future work will focus on reconstituting the *Maverick* lifecycle, allowing us to study *Maverick* biology in unprecedented detail.

POSTER NUMBER: 55

Superpowers of terminators from the view of direct RNA sequencing

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Direct RNA sequencing via nanopore technology has revolutionised transcriptome and epitranscriptome analyses by enabling the detection of full-length RNA molecules without reverse transcription and amplification biases. The technology's unique capacity to capture modifications through characteristic current signal perturbations has opened new avenues for studying their roles in gene regulation and disease. However, significant challenges persist in modification detection accuracy.

Here we performed direct RNA sequencing of the bacterial RNA to investigate the RNA epitranscriptome of the model bacterium *Bacillus subtilis*. Surprisingly, we detected a strong false-positive signal interpreted as N6-methyladenosine upstream of hairpin structures – intrinsic transcription terminators. *In vivo*, these structures fold co-transcriptionally, causing rearrangement of the large RNA polymerase (RNAP)-nucleic acids complex, leading to its dissociation. As terminators are relatively small compared to RNAP and cause drastic rearrangements of the enzyme, they seem to be endowed with “superpowers”.

Our data show that bacterial transcription terminators and potentially similar secondary structures can fold after passing the sequencing nanopore, applying their superpowers to the nanopore. This results in altering translocation kinetics and ionic current signals, leading to artifacts that mimic modification signatures. Such false-positive signals cause misinterpretation of data in the young and promising field of epitranscriptomics. We discuss the data and propose that altering the space beneath the pore by immobilised RNases or denaturing agents could dramatically improve the accuracy of direct RNA sequencing and therefore the detection of real modifications.

POSTER NUMBER: 56

Predictive Genomics for Gene Regulation and Cell Fate Determination

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The vertebrate genome is hierarchically organized into three-dimensional (3D) conformation that regulates gene expression. While cohesin-mediated chromatin loop extrusion typically halts at convergently oriented CTCF binding sites to form TAD boundaries, a longstanding paradox remains: the number of CTCF binding sites far exceeds the observed TADs of a particular cell type, leaving critical questions about the mechanisms governing 3D genome organization. To address this, we developed a high-throughput in silico genetic screening approach, leveraging advanced machine-learning tools designed for predicting chromatin interactions, to identify novel regulators of genome organization. Through this approach, we identified several previously uncharacterized factors that bind at TAD boundaries, including chromatin-modifying enzyme and uncharacterized zinc-finger proteins. Subsequent experimental validation and mechanistic studies confirmed their roles in demarcating chromatin domains and provided new insights into how these factors collaborate with known 3D genome regulators, including CTCF and cohesin. Together, these results highlight the potential of applying predictive AI tools for in silico experimenting to accelerate fundamental discoveries in gene regulation and 3D genome organization.

POSTER NUMBER: 57

Ribosomes are decision hubs for embryonic cell fate and cell identity

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Embryonic development relies on precise and coordinated cell fate decisions, a complex process that orchestrates the formation of an entire organism from a single totipotent cell. The commitment to specific cellular lineages is governed by rapid and sequential steps, during which fate-regulatory proteins are produced with precise spatiotemporal control. While transcriptional and epigenetic mechanisms have been proposed to establish developmental trajectories, the role of selective translation in this context remains largely unexplored, particularly during human embryonic development.

In a proof-of-principle study, we have identified a protein factor known as RBPMS, which specialises mRNA translation. RBPMS binds to the 3' untranslated regions of mRNAs through its specific binding motif and promotes translation initiation. Loss of RBPMS disrupts translation initiation in human embryonic stem cells.

Building upon our previous research, we aim to expand the catalogue of fate-specific translation factors that contribute to selective translation. By employing combined proteomics and translatomics-based approaches, we will identify fate-specific translation regulatory mechanisms to understand the intricate establishment of embryonic cell identity.