Scottish Cell Biology Meeting 2026 Programme – 4th September 2026
Abstract booklet

Posters

1. **Tamara Tubbeh, University of Edinburgh**
   Title: Investigating lysosomal targeting as a therapeutic strategy in glioblastoma
   Glioblastoma (GBM) is the most common and lethal brain tumour in adults, with a median survival of 12-15 months, highlighting the urgent need for new therapies. Previous work from our lab has shown that autophagy, a lysosome-dependent degradative process, contributes to GBM progression, while its inhibition reduces tumour formation. Building on these findings, our group has identified a subset of antipsychotic compounds that block lysosomal degradation, inducing cell death in glioma stem cells. My project aims to investigate the cellular pathways underlying this response, by analysing lysosomal content and genome-wide CRISPR/Cas9 screening. Here, I will present current approaches used to optimise these assays in order to study the role of lysosomes in GBM survival. Identifying critical vulnerabilities may reveal new targets for GBM therapy.

2. **Bhavika Choukhani, University of Glasgow**
   Title: Targetting Mitochondrial Pyruvate Carrier 1/2 (MPC 1/2) as a therapeutic metabolic vulnerability in Chronic Myeloid Leukemia
   Chronic myeloid leukaemia (CML) is driven by the BCR::ABL1 fusion oncogene and is effectively treated with tyrosine kinase inhibitors (TKIs); however, metabolically resilient leukaemic cells can persist despite treatment. These cells rely on oxidative phosphorylation (OXPHOS), making mitochondrial metabolism an attractive therapeutic target. My study aimed to evaluate whether inhibition of the mitochondrial pyruvate carrier (MPC) using the novel inhibitor Mito66 could enhance the therapeutic efficacy of Imatinib in CML cell lines (K562 and KCL22) by restricting mitochondrial pyruvate uptake and limiting OXPHOS. Cells were treated with Mito66, alone or in combination with Imatinib, for 72 hours, followed by assessment of growth kinetics, apoptosis, and cell cycle progression. CRISPR-Cas9-mediated MPC1/2 knockout cell lines were used to validate the target specificity of Mito66. Preliminary LC-MS stable isotope tracing demonstrated effective inhibition of MPC activity at higher Mito66 concentrations, supporting ongoing functional studies evaluating the therapeutic potential of MPC inhibition in CML.

3. **Jaijeet Singh, University of Dundee**
   Title: LRRK2-Mediated Rab8A Phosphorylation Drives TBC1D2 Recruitment
   Parkinson's disease is a progressive neurodegenerative disorder affecting approximately 1.5 per 1,000 people worldwide. While most cases are sporadic, 10–15% are familial, with mutations in the LRRK2 kinase gene representing the most common known genetic cause. Pathogenic LRRK2 mutations drive disease through hyperphosphorylation of a subset of Rab GTPases, key regulators of vesicle trafficking and endolysosomal homeostasis, yet the downstream effectors that transduce these aberrant phospho-Rab signals into cellular dysfunction remain poorly characterised. Here, we identify TBC1D2, a proposed Rab7 GTPase-activating protein, as a novel and selective phosphorylation-dependent interactor of LRRK2-phosphorylated Rab8A (pRab8A). Using immunoprecipitation–mass spectrometry, co-immunoprecipitation, and AlphaFold3 structural modelling, we demonstrate that TBC1D2 engages pRab8A through coiled-coil lysine residues K410 and K414, establishing a direct phospho-dependent binding interface. Proteomic characterisation of the endogenous pRab8A–TBC1D2 complex reveals selective co-recruitment of functionally distinct protein classes, including lysosomal components (V-ATPase subunits), lipid transfer proteins (BLTP3A and VPS13 family members), and 14-3-3 adaptor proteins.

Focusing on the 14-3-3 interaction, phosphoproteomic analysis identified threonine 290 (T290) as a critical regulatory site; mutational analysis confirms that T290 phosphorylation is required for 14-3-3 association. Ongoing work aims to determine how pRab8A binding and 14-3-3 recruitment alter TBC1D2 GAP activity and downstream lysosomal function. This study provides mechanistic insight into a previously uncharacterised node of LRRK2-dependent signalling and identifies TBC1D2 as a novel effector regulated by pathogenic LRRK2 activity.

4. **Vivianna James Richards, University of Glasgow**
   Title: Targeting Mitochondrial Metabolism in Chronic Myeloid Leukaemia by Serine-Glycine Restriction
   Chronic Myeloid Leukaemia (CML) stem cells evade standard therapies by using mitochondrial one-carbon (1-C) metabolism for proliferation, heavily depending on exogenous serine and glycine. Therefore, serine/glycine (-SG) restriction represents a potent therapeutic strategy, but translating this diet into in vivo CML models revealed a survival paradox: -SG monotherapy unexpectedly accelerated host mortality compared to a standard diet. We hypothesise that this accelerated decline is driven by immune failure in host macrophages, which rely on 1-C metabolism. This research project aims to identify metabolic vulnerabilities of macrophages under SG restriction. Using primary murine bone marrow-derived macrophages (BMDMs), we quantify the effect of -SG deprivation on viability via cellular assays, functional polarisation via flow cytometry and LC-MS metabolomics to evaluate compensatory de novo serine synthesis. Characterising these mechanisms will optimise precision dietary therapies to safely eradicate CML without compromising essential immune response

5. **Zofia Pukalo, University of Edinburgh**
   Title: Perturbation of Cdk-PLK1 Axis Causes Chromosome Hypercondensation Leading to Microcephaly
   Chromosome condensation is crucial for maintaining genome integrity during mitosis and is carefully controlled by the condensin complexes. Condensin II begins early chromosome compaction through a CDK1–PLK1 phosphorylation pathway. A novel heterozygous gain-of-function mutation of condensin II near its PLK1 docking site was discovered in microcephalic dwarfism, causing excessive chromosome condensation. While hypercondensed chromosomes do not display mitotic defects, they appear to negatively impact cell fitness. The project reveals a novel perturbation of CDK1-PLK1 axis as a critical regulator of condensin II and suggests a new pathogenic mechanism, highlighting the requirement of regulation of chromosome condensation for proper development.

6. **Ben McKinnel, University of Edinburgh**
   Title: Cell-matrix Adhesion Proteins Suppress the Unfolded Protein Response and Maintain Glioblastoma Stem Cell Fitness
   Glioblastoma (GBM) is the most prevalent and deadly primary brain cancer, with a median survival of 15 months after diagnosis. Poor survival is largely driven by glioblastoma stem cells (GSCs), which promote the emergence of heterogeneous, treatment-resistant GBM cells and tumour recurrence. In the present work, we investigate the role of the matrix-adhesion proteins ILK and FAK in GSC transcriptional heterogeneity and plasticity using genetic depletion of ILK or FAK followed by single-cell RNA sequencing. We show that depletion of ILK or FAK reduces GSC fitness and induces a lineage shift characterised by progressive enrichment of early-, middle-, and late-stage unfolded protein response (UPR) markers, impaired transition into differentiated-like glial states, and accumulation of hybrid progenitor-like states. Consequently, ILK and FAK emerge as promising targets to decelerate GSCs self-renewal, promote apoptotic-inducing UPR, and hinder transition into heterogenous cell-states.

7. **Ridvan Kucuk, University of Strathclyde**
   Title: Human iPSC pluripotency validation and neuronal differentiation to develop a stroke-on-a-chip model
   Despite its substantial global burden, therapies for stroke remain limited, in part due to species differences in stroke models, highlighting the need for more predictive human relevant models. Our aim is to validate the pluripotency of human induced pluripotent stem cell (hiPSC) lines that will be differentiated into neurons and utilised in a stroke-on-a-chip model. The pluripotency of three independent hiPSC lines was validated using flow cytometry and quantitative PCR (qPCR), prior to differentiation via dual SMAD inhibition. All three cell lines showed >90% expression of SSEA-4, TRA-1-60 and TRA-1-81 by flow cytometry, and the qPCR detected the expression of OCT4, SOX2 and NANOG. Qualitative data indicated differentiation into neural progenitor cells (NPCs) and terminal differentiation into neurons. Overall, our experiments verified pluripotency through the expression of surface markers and key transcription factors and initial results suggest differentiation into NPCs and neurons, laying foundations for the stroke-on-a-chip model.

8. **Lucía Puchades, University of Edinburgh**
   Title: Investigating the mechanisms underlying low-grade and high-grade endometrial stromal sarcoma (ESS)
   Endometrial stromal sarcomas (ESS) are a group of rare, aggressive mesenchymal cancers driven by oncofusions, where a chromosomal translocation fuses two genes together, resulting in a chimeric protein that drives tumorigenesis. ESS predominantly involves the fusion of NuA4/TIP60 and Polycomb subunits, wherein PRC2.1 subunits are associated with low-grade pathology, and PRC1.1 subunits with high-grade pathology. During my PhD project, I am characterising the transcriptomic mechanism of different ESS fusions, by exogenously expressing them in endometrial stromal cells. I have performed RNA-Seq on a panel of fusions and identified very distinct transcriptomic profiles, with low-grade being Polycomb-independent whereas high-grade may be Polycomb-independent. Additionally, I am performing CUT&RUN and CUT&Tag on a subset of fusions, to provide more detail into how the epigenetic landscape is being altered to drive malignancy. Furthermore, I will perform IP mass spec on a selection of fusions to obtain an insight into their malignant downstream interactors.

9. **Nelson Ankamah, University of Glasgow**
   Title: Investigating ISGylation in Radiotherapy Resistant Colorectal Cancers
   Background Colorectal cancers (CRC) are the second cause of cancer related mortality globally, a third of which are rectal cancers (RC). Radiotherapy (RT) is a standard treatment for locally advanced rectal cancer (LARC), but resistance limits its effectiveness. Although type I interferons contribute to RT responses, the role of Interferon-Stimulated Gene 15 (ISG15), their downstream effector, remains unclear in RT resistance. Methods Mouse models, patient-derived CRC datasets, and an orthotopic LARC model were used to investigate ISG15 expression, signalling, and responses to RT and poly(I:C), assessed by RNA sequencing and western blotting. Results ISG15 expression strongly correlated with WNT/YAP-high tumour cell states in mouse models and CRC patients. RT and poly(I:C) significantly increased ISG15 protein levels in organoids, while in vivo RT induced progressive ISG15 upregulation. Conclusions Altogether, our findings identify ISG15 is an RT-responsive factor and highlights a potential mechanistic link between ISGylation and WNT/YAP pathways in CRC progression.

10. **Rosie Gallagher, University of Dundee**

Title: A High-throughput analysis of Hes-7 dynamics in hiPSC-derived Somitoids

Somitogenesis is the process by which the paraxial mesoderm forms somites, precursors of bones and skeleton. The segmentation clock coordinates this through the spatiotemporal timing of oscillations of different clock genes. Human characterisation has been enabled by human-induced pluripotent stem cell (hiPSC)- derived organoids (somitoids), though these have not been staged across the segmentation clock cycle at scale. A high-throughput system was developed in which hiPSC-derived somitoids were cultured in 384-well plates with a programmable feeding schedule to stagger the initiation of oscillations. An automated pipeline reconstructed Hes7-Achilles oscillations through image processing, segmentation, and analysis. Results showed that staged feeding distributes the Hes7-Achilles signal across the segmentation clock cycle. Media changes during oscillation cause phase resets, whilst plate perturbation induces partial phase resets. Taken together, this research shows that media change can serve as a periodic stimulus for studying the segmentation clock

11. **Rhea Couper, University of Strathclyde**

Title: Investigating Glucose Metabolism & Lipolysis in Cultured 3T3-L1 Adipocytes Using SRS & Zinc-alpha2-glycoprotein (ZAG)

Heterogeneity is a feature across many cells and tissue types, and adipose tissue is no exception, with adipocytes of different sizes within a single fat depot exhibiting distinct metabolic and functional properties. Clinically, the association between a greater number of large adipocytes and metabolic dysregulation contributes to the risk of developing insulin resistance and T2D. Hence an understanding of defective and heterogenous adipocyte metabolism at single-cell level is urgently required. I have utilised SRS to analyse individual cells, circumventing the barriers of population averaging in tissues, using Glucose-D7 to trace and investigate glucose metabolism by exploiting its unique spectral fingerprint in the cell silent region. I now aim to investigate the impact of ZAG protein on metabolic processes, namely lipolysis. So far, ZAG appears to be interacting with 3T3-L1 adipocytes in such a way consistent with the literature. I also believe effects may be species specific between mice & humans.

12. **Flavia Constantinescu, University of Edinburgh**

Title: Selective interaction of SMCHD1 with chromatin is governed by LRIF1 and SMCHD1 ATPase activity

The chromosomal protein SMCHD1 is a GHKL ATPase with important roles in epigenetic silencing, including on the inactive X chromosome (Xi). Mutations of SMCHD1 have been linked to facioscapulohumeral muscular dystrophy (FSHD) and Bosma arrhinia microphthalmia syndrome (BAMS). We use live-cell and single-molecule imaging to investigate SMCHD1 interactions with chromatin and its function in epigenetic silencing. We show that chromatin binding of SMCHD1 genome-wide, including on the Xi, is critically dependent on the protein LRIF1 that mediates interaction with H3K9me2/3-modified nucleosomes. Engineered mutations in the GHKL ATPase domain demonstrate that ATP hydrolysis is required for selective enrichment of SMCHD1 at specific chromatin regions, which is critical for X-linked gene silencing. Conversely, gain-of-function BAMS-associated mutations are linked to accelerated Xi recruitment and gene silencing, or increased Xi compaction. Together, our findings advance understanding of SMCHD1 recruitment and function on the Xi and at other target sites in the genome.

13. **Nicole Promchai, University of Dundee**

Title: Elucidating stress signaling pathways in TDP-43 related disease

Stress granule formation is increasingly recognised as a key driver of amyotrophic lateral sclerosis (ALS), promoting cytoplasmic aggregation of the RNA-binding protein, TDP-43 as the defining hallmark of ALS. The integrated stress response (ISR) promotes stress granule assembly through phosphorylation of eIF2α. Preventing eIF2α phosphorylation emerged as a therapeutic strategy for ALS. However, the limited clinical success of an ISR-directed therapy suggests that additional mechanisms may contribute to stress granule formation in ALS. Using human neuroblastoma cells and human iPSC-derived motor neurons, I demonstrate that cellular stress pathways extending beyond the ISR influence TDP-43 disease phenotypes, and that there are fundamental differences in stress signaling between neuroblastoma cells and human iPSC-derived motor neurons. This work begins to delineate new biomarkers of disease pathway activation and opportunities for therapeutic intervention. It also highlights that a broader understanding of cell type specific stress responses is required to understand how TDP-43 is dysregulated in ALS.

14. **Matthew Walker, University of Glasgow**

Title: Developing a model of breast cancer dormancy in the bone marrow

Bone marrow is the most common secondary site for breast cancer metastasis and recurrence after dormancy. Within this niche, mesenchymal stem cells (MSCs) communicate with breast cancer cells via extracellular vesicles (EVs), regulating tumour dormancy. Our previous work showed MSC-derived EV metabolites induce a slow-growing MCF-7 phenotype. We aim to develop a bone marrow-mimetic dormancy model using synthetic EVs loaded with these metabolites, interacting with MCF-7 spheroids and MSCs in a 2.5D/3D system, integrated into an organ-on-chip platform for screening breast cancer recurrence. Methods include MCF-7 culture, immunostaining, confocal microscopy, nanoparticle tracking analysis, and qPCR, with statistical analysis using ANOVA-based approaches in GraphPad Prism. Results show synthetic EVs mimic natural EV size, remain stable at 4°C, and are efficiently taken up by MCF-7 cells. Metabolites increase dormancy markers (p27, COUP-TFI) and reduce Ki-67 expression. Overall, synthetic EVs act as effective delivery vehicles and support development of a dormancy-on-chip model.

15. **Faisa Omar, CRUK Scotland Institute**

Title: Understanding how mitochondrial ubiquitination impacts the anti-tumour immunogenicity of cell death

Mitochondria are key regulators of apoptotic cell death. During apoptosis, mitochondrial outer membrane permeabilization (MOMP) releases mitochondrial proteins, triggering caspase activation and cell death. However, when caspase activity is inhibited, cells undergo a distinct form of cell death called caspase-independent cell death (CICD). CICD is a pro-inflammatory, immunogenic form of cell death that can stimulate anti-tumor immunity. We have previously found that mitochondria become ubiquitinated during CICD, promoting recruitment of the NF-kB adaptor molecule NEMO, thereby initiating NF-kB inflammatory signalling. I investigated whether LUBAC regulates mitochondrial ubiquitination during CICD. Composed of three proteins, HOIP, HOIL and SHARPIN, LUBAC is a key mediator of M1-linked ubiquitination. I found that loss of HOIL, but not of HOIP or SHARPIN, significantly reduces mitochondrial M1-linked ubiquitination during CICD. These findings suggest a distinct role for HOIL in mitochondrial inflammatory signalling during CICD and point to a more nuanced role for LUBAC in this process.

16. **Rhona Christie, University of Glasgow**

Title: NADPH-sensing proteins coordinate lipid biosynthesis and govern ferroptosis susceptibility

Ferroptosis is a form of non-apoptotic cell death driven by catastrophic lipid peroxidation. The impact of metabolic configuration on ferroptosis susceptibility, however, remains poorly defined. Using a model of acute lymphoblastic leukemia (ALL) and diffuse large B cell lymphoma we observed profound metabolic and lipidomic alterations governed by cell culture density. Specifically, we found robust fluctuations in regulatory proteins involved in fatty acid, cholesterol and selenocysteine biosynthesis that altered ferroptosis sensitivity by cell state. Metabolite and flux analysis of low- and high-density cells revealed significant shifts in nicotinamide adenine dinucleotide phosphate (NADPH) production through the pentose phosphate pathway. Three orthogonal interventions to restrict NADPH production reversed the observed density-dependent phenomena and, to our surprise, evidenced that canonical NADPH-consumptive protective axes could not restore ferroptosis sensitivity. We thus identified the roles of two NADPH-sensors (FTO and MARCHF6) in coordinating fatty acid, cholesterol and selenocysteine biosynthesis by cellular context.

17. **Chiara Cazzaniga, University of Dundee**

Title: UBE2Q1 cooperates with ZER1/ZYG11B in CUL2-dependent N-terminal degron targeting of JAK2

Ubiquitylation regulates protein stability through the coordinated action of E1, E2, and E3 enzymes. Non-canonical ubiquitylation extends the ubiquitin code beyond lysine residues to include serine, threonine, tyrosine, and other hydroxyl-containing biomolecules. This added layer of complexity defines an emerging and technically challenging frontier in ubiquitin biology. We have recently identified non-canonical activity within the UBE2Q family of ubiquitin-conjugating enzymes (E2s), demonstrating their ability to catalyse serine and threonine ubiquitylation (Rehman et al., 2024). Among these, UBE2Q1 is essential for early embryonic development and embryo implantation; however, the molecular mechanisms, substrates, and cognate ubiquitin ligases (E3s) that underpin its non-canonical activity remain unknown. Here, we identify UBE2Q1 as a previously unrecognised component of the CUL2-dependent N-degron pathway. Using integrated transcriptomic and proteomic profiling of UBE2Q1-knockout HeLa cells, as well as biochemical approaches, we find that UBE2Q1 cooperates with the CUL2 substrate adaptors ZER1 and ZYG11B to promote degradation of a variety of N-degron-containing proteins. Together, these findings identify UBE2Q1 as an E2 enzyme functioning with CUL2–ZER1/ZYG11B complexes to regulate N-degron-mediated protein turnover and provide mechanistic insight into how UBE2Q1 and non-canonical ubiquitination contribute to protein homeostasis.

18. **Billie Nuth, University of Dundee**

Title: Epithelial-derived factors and metabolites derived from Parasutterella excrementihominis promote neutrophil activation and NET formation

Neutrophil activation and extracellular trap (NET) formation contributes to tissue injury in gut inflammation, with Parasutterella excrementihominis driving pathological NETosis. Epithelial-injury signals and microbial metabolite contributions to human neutrophil activation remains unclear. Myeloperoxidase (MPO) release, NET formation, and inflammatory gene expression were assessed in human blood neutrophils (n=6 donors) stimulated with injured human colonic organoid-derived supernatant (freeze thaw + mechanical injury, HCOrgSN), or lipopolysaccharide (LPS; 100 ng/mL) ± P. excrementihominis metabolites (succinic acid, 6-hydroxyhexanoic acid). HCOrgSN increased MPO release ($p=0.0171$), NET formation ($p<0.0001$) and increased IL1B ($p=0.0476$) and CXCL8 ($p=0.0022$). LPS + P. excrementihominis metabolites increased MPO release ($p=0.0223$) and NET formation ($p<0.0001$) compared to unstimulated control. LPS + P.

excrementihominis metabolites increased TNF (p=0.0059) and IL1B (p=0.0027) and reduced IL10 expression (p=0.0198) compared to LPS alone. Epithelial-derived mediators and P. excrementihominis metabolites amplify neutrophil activation and NETosis, supporting a role for epithelial-immune-microbiota crosstalk in sustaining inflammation in gastrointestinal diseases

19. **Phimmada Hatthakarnkul, University of Glasgow**

Title: Investigating the Prognostic Value of NF-κB Signalling and Tumour-Immune Cell Interactions in Prostate Cancer

Background Prostate cancer (PC) is the second most diagnosed cancer and the fifth leading cause of cancer-related death among men globally. NF-κB activation are known to associate with the development of castration resistance and resistance against other androgen depletion strategies, which remains a major therapeutic challenge. Methods We employed immunohistochemistry (IHC) to identify the prognostic value of NF-κB markers and multiplex immunofluorescense (mIF) to understand the interaction between tumour and the surrounding immune cells. Results Patients with High IKKα experienced poorer survival compared to those with low IKKα expression. Using Lunaphore COMET platform, 20-plex staining demonstrated an association between NF-κB signalling related tumours and tumour microenvironment (TME) in PC cohort. Conclusion The effect of IKK-alpha driven NF-κB signalling confirm our hypothesis that inflammatory signalling is involved in disease progression in PC cohort. The relationship with TME warrants further study to understand how tumour and immune cells interactions lead to the poor prognosis in PC patients.

20. **Isra Abdi, CRUK Scotland Institute**

Title: Investigating the Role of ARF GTPases in Regulating Cell Death and Innate Immune Signalling in Cancer Cells

Introduction: ADP ribosylation factor (ARF) GTPases are key regulators of membrane trafficking and intracellular signalling pathways that maintain cellular homeostasis. Emerging evidence suggests that ARF proteins may also influence cancer cell survival and death pathways. In addition, cellular stress can activate innate immune signalling pathways such as the cGAS STING pathway, which is triggered by cytosolic DNA and can promote inflammatory cell death. However, the role of ARF GTPases in regulating these pathways in cancer cells remains poorly understood. This study aims to determine whether loss or inhibition of ARF activity alters apoptotic and innate immune signalling. Methods: CP2 prostate cancer cell lines containing CRISPR sgRNA ARF knockout were generated to examine the functional role of ARF. ARF activity was also disrupted via inhibition using Brefeldin A and NAV2729. Cells were treated with different drug concentrations and monitored over 48 hours using live cell imaging. Caspase 9 activity was assessed to evaluate mitochondrial apoptosis and markers of cGAS STING activation were analysed. Results: Both ARF knockout and ARF pharmacological inhibition altered cell death dynamics compared with control cells, with increased SYTOX Green positive cells over time. Conclusion: These findings suggest ARF GTPases may contribute to regulating apoptotic and innate immune signalling pathways in cancer cells. Understanding how ARF loss or inhibition influences the cGAS STING pathway may provide insight into mechanisms of cancer cell death and reveal potential therapeutic targets.

21. **Jacquie Mills, CRUK Scotland Institute**

Title: Characterisation of RNMT phosphorylation in glioblastoma

Glioblastoma (GBM) is an aggressive and incurable primary brain tumour. Dysregulation of gene expression in glioblastoma stem cells (GSCs) drives treatment resistance and tumour recurrence. RNMT catalyses the formation of the m7G RNA cap on RNA polymerase II transcripts, supporting transcript stability, maturation, nuclear export, and translation initiation. Experimental reduction of RNMT expression reduces tumour burden of the patient-derived GSC cell line G7. Therefore we are interested in how RNMT functions in GBM, including the configuration of the most active protein complex. The RNMT N-terminal region (NTR) regulates methyltransferase activity, gene expression, and proliferation. Here we show that the RNMT NTR is phosphorylated in G7 cells at a predicted α-helix within the first 30 amino acids of RNMT. Phosphorylation may be affected by FGF signalling but maintains RNMT interactions and cell phenotypes. Understanding RNMT NTR phosphorylation in GSCs will provide a novel opportunity to target gene expression regulation in GBM.

22. **Yinshan Liu, University of Dundee**

Title: Dynamics of chromatin loops and their molecular regulation in human genome organization

Chromatin loops are dynamic influenced by cohesin activity, cell-cycle progression and the surrounding nuclear environment. Here, we optimised CRISPR LiveFISH to visualise two endogenous chromatin loop anchors in live human cells. RAD21 depletion increased loop-anchor distances, whereas WAPL depletion produced no significant change. A loop-extrusion model incorporating cohesin loading, release and CTCF-dependent stabilisation reproduced the experimental distance distributions, and the corresponding chromatin contact Hi-C pattern. During the G2–M transition, loop-anchor distances transiently increased approximately four minutes before nuclear envelope breakdown and subsequently decreased during mitotic chromosome condensation. RAD21 depletion increased pre-mitotic distances but did not influence the post-NEBD reduction, consistent with a transition from cohesin-dependent loop organisation to condensin-dependent compaction. Finally, we established GFP–HP1, GFP–BRD4 and GFP–NPM1 cell lines for simultaneous visualisation of chromatin loops and distinct nuclear environments. Together, this work provides a live-cell framework for investigating chromatin loop regulation across cell-cycle and nuclear contexts in real time.

23. **Maha Khazi, The University of Manchester**

Title: Regulation of Antioxidant mRNA Translation by LaRP Protein Slf1 under Oxidative Stress in S. cerevisiae

Oxidative stress is a common feature of both cancer and neurodegenerative diseases. Elevated reactive oxygen species (ROS) produced during oxidative stress negatively impact protein synthesis, inducing global translational repression while selectively producing antioxidant proteins to maintain redox balance. The mechanisms underlying this selectivity remain unclear. In Saccharomyces cerevisiae, the La-related protein Slf1 has been identified as a positive regulator of translation under oxidative stress, sustaining antioxidant production despite this global repression. Previous studies suggest that Slf1 promotes antioxidant protein expression and may exhibit codon-bias effects. This study investigated how Slf1 affects the expression of five antioxidant proteins and whether synonymous codon substitutions affect translation in its absence. Cultures were subjected to oxidative stress, and protein expression was measured by western blotting. Slf1 effects appear to be protein-specific, with a bias for GGU codons in GRX2. These findings advance our understanding of selective antioxidant regulation during oxidative stress, with implications for ROS-associated diseases.

24. **Keira Hughes, University Of St Andrews**

Title: Understanding Metabolism for Tumour Treatment and Detection in Soft Tissue Sarcomas

Soft Tissue Sarcoma (STS) is a rare group of malignancies, encompassing around 80 different types of cancers with poor prognosis and a lack of biomarkers to distinguish them, with available therapeutic options also lacking specificity. Interferon-stimulated gene 15 (ISG15) is a ubiquitin-like modifier protein, found to be overexpressed in many different STS subtypes, even being considered a biomarker for solitary fibrous tumour. Metabolic reprogramming is a hallmark of cancer, due to its fundamental role supporting cancer cells proliferation. Thus, the study of metabolism offers the opportunity to uncover targetable vulnerabilities for STS. Of note, ISG15 is involved in metabolic processes such as oxidative phosphorylation and glycolysis. Thus, we hypothesize that high expression of ISG15 contributes to the metabolic alterations occurring in STS types. Using targeted and untargeted metabolomic (LC-MS/MS) approaches, we aim to define the role of ISG15 on metabolism and proliferation of STS cells and identify novel therapeutic approaches.

25. **Mark Kelly, University Of St Andrews**

Title: Dysregulation of the de novo synthesis of phosphatidylethanolamine in β-catenin-driven Hepatocellular Carcinoma

Hepatocellular carcinoma (HCC) is a major cause of cancer-related mortality worldwide, consisting of multiple subtypes with varied therapeutic responses. Identifying metabolic alterations associated with these genetically distinct subtypes is important for improving therapeutic outcomes. Cancer cells undergo metabolic reprogramming to support tumour growth, and these metabolic alterations are often linked to specific genetic mutations. Therefore, investigating subtype-specific metabolic changes may reveal new biomarkers and therapeutic targets for HCC. In this study, plasma samples from genetically engineered mouse models (GEMMs) of HCC were analysed using LC-MS-based metabolomics through targeted and untargeted approaches. Untargeted profiling and structural elucidation identified ethanolamine phosphate as a metabolite potentially consumed by β-catenin-driven tumours. Ethanolamine phosphate is an important intermediate in the synthesis of phospholipids such as phosphatidylethanolamine. This study explores the biological impact of the dysregulation of the de novo synthesis of phosphatidylethanolamine using in vitro models to better understand its role in HCC metabolism.

26. **Maria Dolores Pejenaute-Ochoa, University of Dundee**

Title: Mechanistic basis of client recognition and cargo export by the Erv14 cargo receptor

COPII-coated vesicles mediate ER-to-Golgi transport of secretory and membrane proteins. Cargo selection often relies on receptors, such as Erv14, bridging clients to Sec24, the coat's cargo-recognition adaptor; how Erv14 recognizes clients remains unclear. Erv14 contains four transmembrane helices and two cytosolic loops, H1-H2 and H3-H4. AlphaFold3 modelling revealed a conserved H1/H4 contact surface across all clients tested, confirmed by Erv14-mutagenesis affecting Mid2 and Axl2 traffic. Modelling Erv14-client-Sec24 trimers revealed conserved contacts at H3/H4, harbouring the known IFRTL signal, and similarly conserved contacts at H1/H2, suggesting a secondary role in Sec24 binding, confirmed by effects on Axl2 and Mid2 traffic. H3/H4 likely engaged the Sec24 D-site, whereas acidic residues on the H1/H2-loop engage basic client residues, positioning client acidic residues near the Sec24 B-site. Combined mutagenesis of these residues impaired Mid2 export, supporting a cooperative mechanism in which client binding to Erv14 via H1/H4 positions both export signals for Sec24 engagement.

27. **Ann Wheeler, University of Edinburgh**

Title: The UK Node of Euro-BioImaging – Open Access to Advanced Biological Imaging

EuroBioImaging provides open access to basic and advanced microscopy. BioImagingUK administers the UK's biological Node for EuroBioImaging BioImagingUK, UKRI-BBSRC and UKRI-MRC are pleased to announce that the UK is now offering open access to state-of-the-art biological imaging technologies through Euro-BioImaging. Funding for accessing equipment is also available through the UK Bioimaging User access fund The pilot UK Node is a distributed infrastructure consisting of seven initial sites located across the UK: Edinburgh Super-Resolution Imaging Consortium (ESRIC), the Francis Crick Institute, King's College London, Liverpool University, Octopus at Harwell, Oxford Brookes University and York University. The UK Node offers open access to a wide range of advanced bioimaging techniques including correlative, multimodal, high-content and super-resolution microscopy. Users will have access imaging instruments, expertise, training and data management services to complement those at their own institutions or among their collaboration partners. For more information and details of how to apply, please visit https://www.eurobioimaging.eu/nodes/uk-node

28. **Nerea Caro, University of Strathclyde**

Title: Mechanically Defined Microenvironments Regulate Neurovascular Organisation in Blood–Brain Barrier Organoids

Extracellular matrix (ECM) remodelling contributes to blood–brain barrier (BBB) dysfunction in ageing and vascular dementia, where altered stiffness and viscoelasticity may disrupt neurovascular organisation. However, most in vitro BBB models lack defined mechanical microenvironments. Here, we developed tri-culture BBB organoids composed of human brain endothelial cells (hCMEC/D3), pericytes, and astrocytes within ECM-functionalised sodium alginate hydrogels with tunable mechanical properties. Alginate formulations were ionically crosslinked to model compliant brain-like and stiffness-elevated microenvironments associated with ageing-related ECM remodelling. Mechanical properties were characterised using oscillatory rheology and stress relaxation analyses. Multicellular spheroids were embedded within laminin- or fibronectin-functionalised matrices to investigate how biochemical and mechanical cues regulate neurovascular organisation. Preliminary observations indicate that increased matrix stiffness alters spheroid compactness and F-actin organisation. This work establishes a mechanically defined BBB organoid platform to investigate mechanobiological drivers of barrier dysfunction in vascular dementia.

29. **Mohab Elghobary, University of Dundee**

Title: Tankyrase Catalytic Function in TDP-43 Stabilization and Toxicity

Amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD) are related neurodegenerative disorders characterised by cytoplasmic aggregation of the RNA-binding protein TDP-43 in over 90% of ALS and approximately 45% of FTD cases. Our laboratory has identified the poly(ADP-ribose) polymerase Tankyrase as a regulator of TDP-43 toxicity, with Tankyrase downregulation reducing TDP-43-mediated neurodegeneration. However, whether this protection is mediated through loss of Tankyrase catalytic activity or disruption of its interaction with TDP-43 remains unclear. Using Drosophila melanogaster and mammalian cell models, we compared the effects of Tankyrase downregulation, catalytic inhibition and disruption of Tankyrase binding. We show that both Tankyrase downregulation and inhibition of its catalytic activity are involved in TDP-43 toxicity. In contrast, approaches designed to disrupt the canonical Tankyrase substrate-binding interface did not alter TDP-43 toxicity, supporting the notion that TDP-43 binds Tankyrase through a distinct, non-canonical interface that requires further investigation.

30. **Dylan Fowler, University of Strathclyde**

Title: An Electrospun Platform to Study Glioblastoma Mechanotransductive Events

Glioblastoma is a lethal primary brain tumour with poor patient survival outcomes of 12-14 months, despite intensive therapeutic intervention. Poor preclinical models have exaggerated the effectiveness of therapies that often fail at the clinical trial stage. The invasive nature of glioblastoma cells is key to poor survival outcomes. Migrating cells hijack the native brain anatomy such as white matter and blood vessels to invade into distal sites. In our work, we aim to create a topographical model of key glioblastoma invasion routes such as the aligned white matter/blood vessels and mesh-like parenchyma to better understand glioblastoma mechanotransductive-events in vitro. We have produced aligned and randomly orientated electrospun substrates that we functionalise using brain extracellular matrix mimicking peptides. In the fabrication of this platform, we have overcome common electrospun substrate limitations relating to bio-imaging and membrane thickness. Moreover, we have incorporated our substrates into a common microscope compatible well-plate format.

31. **Alison Roberts/Micheal Porter, The James Hutton Institute**

Title: Live super-resolution STED imaging in plants.

The James Hutton Institute has a unique, upright, STED imaging system that's been specifically built for live cell imaging in plants and has a variety of modular additions including lifetime imaging and refractive index compensation. This is a challenging technique but we are developing methods to allow live, gentle STED imaging in intact tissues. The microscope is available for use by academics who have a need to utilise our facility. This talk will outline the capabilities of the system and share some novel data.

32. **Taryn Wilson, University of Strathclyde**

Title: Sex- and Cell-Type-Specific Mitochondrial Responses to NFE2L2 Inhibition and Oestrogen in Human Cardiac Cells Under Hypoxia

Background: Pulmonary arterial hypertension (PAH) causes right ventricular (RV) failure, which determines mortality. Mitochondrial dysfunction, oxidative stress, and fibroblast activation drive RV maladaptation, while sex differences implicate oestrogen and NFE2L2 signalling.

Methods: Male and female cardiac fibroblasts and cardiomyocytes were exposed to normoxia or hypoxia and treated with vehicle, ML385, 17Î²-oestradiol (E2), or ML385+E2. Confocal imaging assessed mitochondrial superoxide, membrane potential, and network morphology using MitoSOX, TMRM, and MitoTracker Green. Immunofluorescence assessed stress, mitochondrial, proliferative, and fibrotic markers.

Results: ML385 increased superoxide and reduced membrane potential and TFAM, with hypoxia amplifying these effects. E2 partially preserved mitochondrial function, particularly under hypoxia. Cardiomyocytes showed greater membrane-potential loss, whereas fibroblasts displayed broader stress and remodelling responses. Male fibroblasts showed stronger superoxide and stress responses, while E2 protection was greater in female cells.

Conclusions: NFE2L2 inhibition exposes mitochondrial vulnerability, while E2 provides sex- and cell-dependent protection relevant to RV remodelling in PAH.