# The 28th Annual Conference of Chinese Life Scientists Society in the UK

全英华人生命科学学会
Chinese Life Scientists Society in the UK

The Cruciform Building lecture theatre 1 (B304)
London WC1E 6AE

5th August 2023
Queen Mary University of London
University College London

## Chinese Life Scientists Society in the UK (CLSS-UK) Committee (2022-2023)

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**Committee Secretary**: Prof Shouqing Luo, University of Plymouth

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Dr Haotian Gu, King's College London

Mr Qizi Ju, King's College London

Miss Minxue Tang, University of Reading

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Mr Yong Ji, Imperial College London

**Local Organisers (in the order of alphabets)**

Coordinator: Dr Guifen Chen, Prof Huiliang Li

Members: (in the order of alphabets)

Dr Jinke Chang, Miss Yiting Feng, Mr. Qizi Ju, Mr. Yong Ji, Dr. Wenqianglong Li, Miss Minxue Tang, Mr. Jian Wang, Miss Xiuting Yang, Dr Jingkun Zeng

**Sponsors**

## Agenda

### CLSS-UK 2023 programme

### Morning session

| Time | Speaker and title | Chaired by |
|---|---|---|
| 8:50 | Registration | |
| 9:20 | Greetings 驻英使馆教育参赞匡建江老师等 | Guifen Chen |
| 9:40 | **Keynote speech I. Prof Ziheng Yang FRS**, **UCL**<br>*Inferring the history of species divergence and gene flow using genomic data* | |
| 10:30 | **Prof Huiling Tan, University of Oxford**<br>*Adaptive Neuromodulation for Movement Disorders* | Li Su |
| 11:00 | **Dr Chao Li, University of Cambridge/Dundee**<br>*Intelligent Neuroimaging for Precision Neuro-oncology* | |
| 11:20 | **Dr Yu Ye, Imperial College London**<br>*Reversing protein aggregation with proteasomes* | Jinke Chang |
| 11:40 | **Dr Binzhi Qian, University of Edinburgh**<br>*Macrophage heterogeneity in cancer metastasis and therapy resistance* | |
| 12:00 - 12:10 | Sponsor's talk 1 (Abclonal) | Jian Wang |
| 12:10 - 12:20 | Sponsor's talk 2 (Sino Biological) | |
| 12:20 - 13:50 | Lunch, Posters & Networking | |

**Afternoon session**

| Time | Speaker and title | Chaired by |
|---|---|---|
| 13:50 | **Keynote speech II Prof Yang Shi FMedSci, University of Oxford**<br>*Epigenetic regulation and human diseases* | Guifen Chen |
| 14:40 | **Prof Yong-Jie Lu, Queen Mary University of London**<br>*Circulating tumour biomarkers for prostate cancer diagnosis and precision medicine* | Zhidao Xia |
| 15:10 | **Prof Yaohe Wang,** Queen Mary University of London<br>*How can we use viruses to conquer human cancer?* | |
| 15:40 | **O1. Zihao Wang, Oxford,** *Stoichiometry and function studies of human cardiac sHsps*<br>**O2. Pingfan Song, Cambridge,** *Interpretable deep learning for light-field microscopy neuroimaging*<br>**O3. Yan Liang, QMUL,** *Annelid functional genomics reveal the origins of bilaterian life cycles*<br>**O4. Qiukai Qi, Bristol***, Shape Memory Oleogel Composites with Optical Modulation*<br>**O5. Xu Shen, UCL,** *Understanding CDK4/6 Inhibitor Resistance: The Cell Cycle Position Matters*<br>**O6. Yichen Wang, Sanger,** *Mutational processes in normal kidneys across countries with varying cancer incidence rates*<br>**O7. Jingnu Xia, KCL,** *Optogenetic Miro cleavage reveals direct consequences of real-time loss of function in Drosophila*<br>**O8. Mingzhe Tang, Crick,** *Mitotic chromosome formation by DNA entrapment inside the condensin ring* | Jingkun Zeng |
| 16:20 | Break | |
| 16:30 | Academic job application – **Dr Guifen Chen**, **QMUL** | Wenqianglong Li<br>Huiliang Li |
| 16:45 | fellowship application – **Dr Matthew Swire, UCL,** (MRC career development) | |
| 17:00 | funding applications – **Prof Huiliang Li**, **UCL** | |
| 17:15 | NHS career progression – **Dr Zhangjie Su, Cambridge University Hospitals NHS** | |
| 17:30 | Industrial job application – **Dr Yue Du AstraZeneca** | |
| 17:45 | Q and A | |
| 18:10 | Closing | |

**19:00 – 21:00 Dinner**
**Chang's Noodle, 35-37 New Oxford St, Holborn, London WC1A 1BH**

# Keynotes

**K1: Inferring the history of species divergence and gene flow using genomic data**

**Prof Ziheng Yang FRS**
University College London, UK

**RESEARCH SUMMARY**

"The history of the Earth is recorded in its crust; the history of species is written in their genomes". Genomic sequences from modern species allows us to infer species phylogenies, date species divergences and test for between-species gene flow. However, the evolutionary process of genes and genomes is highly stochastic, so that inference using genomic sequence data requires powerful statistical inference methods and computational algorithms. In the past two decades, the multispecies coalescent (MSC) model has emerged as the natural framework for analysis of DNA sequences sampled from multiple species, to address a number of interesting biological problems, such as estimation of species relationships and divergence times, inference of between-species introgression, and delineation of species boundaries. Implementations of the MSC have mostly taken the Bayesian approach, using Markov chain Monte Carlo (MCMC) to average over the genealogical histories at different genomic regions. In this talk, I will provide an overview of the MSC model and discuss our efforts to implement efficient MCMC algorithms under the model.

**BIO**

Ziheng Yang holds the RA Fisher Chair in Statistical Genetics at University College London (UCL), and is Director of UCL Centre for Computational Biology. He received a PhD in agronomy in 1992 from Beijing Agricultural University, and has been a faculty member in UCL since 1997, initially as a lecturer (1997), then reader (2000) and professor (2001). He works in the areas of molecular phylogenetics, population genomics, and computational statistics. He develops statistical methods and computer software for comparative analysis of sequence data from different species, to infer the history of species divergence and gene flow and to understand the forces and mechanisms of gene sequence evolution. He has published ~200 papers and two graduate-level textbooks (in 2006 and 2014, both by Oxford University Press). He maintains two computer programs called PAML and BPP. Yang has won several awards, including Presidents' Award for Lifetime Achievement from SSB (2008), Frink Medal for British Zoologists from The Zoological Society of London (2010), and Darwin-Wallace Medal from Linnean Society of London (2023). He was elected a Fellow of the Royal Society in 2006. Further information about his research can be found at http://abacus.gene.ucl.ac.uk/.

**K2: Epigenetic regulation and human diseases**

**Prof. Yang Shi, FMedSci**
University of Oxford, UK

**BIO**

Yang Shi received his PhD from NYU Medical Center and postdoctoral training with Dr. Tom Shenk at Princeton University where he discovered the transcription factor YY1. He began his independent research career at Harvard Medical School as a tenure track assistant professor in 1991 and received tenure and full professorship in the Department of Pathology at Harvard Medical School in 2004. Yang is widely known for the seminal discovery of the first histone

demethylase (LSD1), which overturned the 40-year-old dogma that histone methylation is static and irreversible. This discovery completely changed our view of how histone methylation is regulated, demonstrating that methylation can provide both stability and flexibility in controlling epigenetic information. In 2009 he joined Boston Children's Hospital where he held a Merton Bernfield Professorship in the Department of Medicine and was also professor of Cell Biology of Harvard Medical School, where he was honored with the inaugural C. H. Waddington Professorship of Pediatrics in 2018. He joined Oxford University in 2020 and is currently Professor of Epigenetics of Oxford University and member of the Ludwig Cancer Research. His honors include election to the American Association for the Advancement of Science (2011), The Ellison Medical Foundation Senior Scholar in Aging (2012), American Cancer Society Research Professor (2012), election to the American Academy of Arts and Sciences (2016), National Cancer Institute Outstanding Investigator Award (2017), election to EMBO (2022), to AACR Academy (2022), the National Academy of Medicine (2022) and UK Academy of Medical Sciences (2023).

# Invited Talks

**I1: Adaptive Neuromodulation for Movement Disorders**
**Prof Huiling Tan**
University of Oxford

**RESEARCH SUMMARY**
Although electrical brain stimulation is already affording major therapeutic benefits, there is vast scope for improving and extending this to provide adaptive and tailored interventions, and for harnessing recent advances in non-invasive stimulation techniques to deliver multisite manipulation of brain circuits. Therefore, we are at the beginning of a therapeutic revolution whereby we can interact with neural dynamics from moment-to-moment as necessary to reverse or ameliorate dysfunctional brain activity. To this end, we must record and interpret brain signals in real-time with sufficient temporal and spatial resolution to give nuanced control.
The Tan Group takes a multidisciplinary approach, combining experimental manipulations in healthy subjects and patients with sophisticated signals analysis and modelling. Our experimental manipulations include non-invasive brain stimulation, and often involve patients who have had deep brain stimulation electrodes implanted as treatment for problems with movement. Over the years we have made major advances in understanding how abnormal interactions between brain cells cause slowness of movement, tremor and stiffness in people with Parkinson's disease. At the same time we have leveraged these insights to pioneer closed-loop approaches to therapeutic brain stimulation.
We will share a few projects demonstrating how we work with patients with movement disorders who received the surgery for Deep Brain Stimulation to: 1) better understand the role of the subthalamic nucleus in gait control and to drive forward adaptive DBS for gait difficulties; 2) explore the use of machine learning based approach to detect specific brain states to drive closed-loop DBS for essential tremor; 3) test and compare different signal processing and control algorithms for adaptive DBS for Parkinson's disease while using beta amplitude as the feedback signal; 4) design and test a new translational neuroscience research tool with improved performance on sensing during stimulation.

**BIO**
Huiling is a MRC Investigator and Professor of Human Electrophysiology and Neuromodulation by the University of Oxford. Before that, Huiling studied Control Engineering (B.Sc.) at Bei Hang University China, where she was awarded an Undergraduate Academic Excellence Scholarship for four successive years (1996-2000). In 2003, Huiling came to the University of Oxford with an Oxford Overseas Research Scholarship and China Oxford Scholarship. After completing her D.Phil. in Engineering Sciences at the University of Oxford in 2006, Huiling studied Psychology with The Open University and was awarded B.Sc. in Psychology with Honours (1st class). Huiling started her career in

neuroscience research in 2010 when she started as a postdoctoral research associate, focusing on the role of the basal ganglia in purposeful movement, and how information important for motor control and motor learning is represented, processed and transmitted in different networks of the brain, including the motor cortex and the basal ganglia.
Huiling's current research focuses on defining how activity in large populations of neurons is coordinated in healthy movement, how such coordination may go awry in diseases, and translating this information in to improved treatment for Parkinson's Disease, Essential Tremor and other disorders of movement. The ultimate goal of her research is to develop recurrent Brain Computer Interfaces that selectively and strategically target circuit malfunctions for the correction of identified pathological neuronal network dynamics in neurological and psychiatric disorders.

## I2: Intelligent Neuroimaging for Precision Neuro-oncology

**Dr Chao Li**
University of Cambridge/Dundee

**RESEARCH SUMMARY**

Brain tumours encompass a range of malignant and benign entities, presenting complex pathophysiology that poses challenges to effective clinical decision-making and treatment. Multi-modal neuroimaging offers a non-invasive technique for investigating brain tumours [3,5]. Artificial intelligence (AI), coupled with neuroimaging, provides an automated solution to optimise patient management, promising to accelerate precision neuro-oncology. Tailoring AI models to the critical challenges in neuro-oncology, informed by clinical domain knowledge, could advance our understanding of brain tumours, accelerate individualised and precise therapeutics.
Glioma, the most common malignant brain tumour in adults, exhibits remarkable heterogeneity and extensive invasion. To characterise tumour heterogeneity based on MRI, we have devised novel radiological features that capture tumour morphology and spatial heterogeneity [11]. When combined with machine learning methods, these features demonstrate robust performance in subtyping patients across diverse tissues and imaging modalities. The identified patient sub-groups exhibit distinct molecular characteristics and prognostics. Advanced MRI techniques, such as perfusion and diffusion MRIs [4,6], offer sensitive information for characterising tumour invasion beyond contrast-enhanced MRI. However, advanced MRI scans are often low-resolution, hindering the availability of full training labels for developing supervised models. To address this challenge, we developed weakly supervised deep learning models capable of identifying tumour invasion beyond contrast enhancement [2]. Moreover, glioma is considered a systematic disease as it frequently spreads along white matter tracts throughout the brain. To globally characterise tumour invasion, we have developed an iterative tract-based spatial statistics method to quantify brain structural connectivity and measure tract integrity in brain tumour patients [10]. By comparing patients to healthy controls, we identified regional disruptions in the connectome of glioblastoma patients, which demonstrate significance in predicting patient survival and indicating treatment targets [9]. Building on this study, we incorporated the brain connectome into the AI model to better characterise glioma. Specifically, we developed a multi-modal learning model that leverages three encoders to extract features from focal tumour images, tumour geometry, and global brain networks to predict the isocitrate dehydrogenase (IDH) mutation, achieving superior performance compared to other state-of-the-art models [8].
When translating AI models into real-world practice, we must address challenges posed by heterogeneous clinical datasets, such as missing scans and low image resolution. Consequently, we have developed AI approaches to enhance image quality and standardisation [1,7]. To ensure trustworthy AI solutions, we are developing biophysics-informed deep learning models that enhance model explainability and generalisability. We are testing AI prototypes in real-world clinical settings by integrating model development with clinical systems to obtain clinical and biological validations. We conduct multi-centre imaging trials to validate the efficacy of imaging tools, employing reproducible and transparent pipelines for processing MR images. In the next phase, we will test these imaging tools at scale by connecting them to large population datasets. Our vision is to revolutionise the healthcare of brain tumour patients through the utilisation of image-based AI models.

**BIO**

Dr Chao Li is a Reader in Biomedical Imaging and Artificial Intelligence at the University of Dundee and Principal Research Fellow at the University of Cambridge. Before he started his research adventure at Cambridge, Dr Li completed his clinical training in neurosurgery and neurology covering major neurological conditions. At Cambridge Mathematics Information in Healthcare and Cambridge Brain Tumour Laboratory, he acquired his expertise in modelling neurological diseases based on novel AI and multi-omics approaches. Dr Li is particularly interested in developing cost-effective AI models and translating these models into healthcare management to promote personalised medicine. His work has been published widely in clinical and technical venues, including *Nature Machine Intelligence*, *Brain, IEEE Transactions in Medical Imaging, MICCAI, etc*. He has been awarded a fellowship from the Guarantors of Brain, supporting him to develop novel translational AI approaches for clinical neurosciences. In 2021, due to his contribution to the neuro-oncology research, he is awarded as the Young Investigator Award by British Neuro-Oncology Society and Brain Tumour Research.

## I3: Reversing protein aggregation with proteasomes

**Dr Yu Ye**
Imperial College London

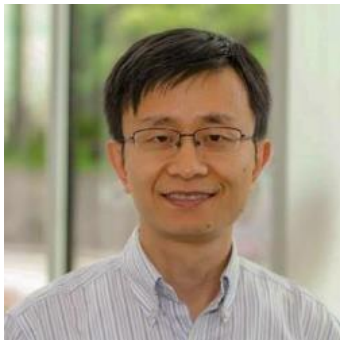
**RESEARCH SUMMARY**

My lab investigates the role of the ubiquitin-proteasome system (UPS) in proteostasis and cell stress. We are especially interested in the clearance mechanisms for pathological aggregates in neuronal models and how malfunctions of the proteasome system may lead to proteotoxic stress, inflammation and eventually degeneration of physiological functions. Over the past few years, we have provided the biochemical basis describing how proteasomes process filamentous aggregates (fibrils) and small aggregates (oligomers) (Cliffe et al., Cell Rep. 2019; Ye, Finley & Klenerman, JMB 2020). More recently, we demonstrated that toxic aggregates may be processed by proteasomes in cells and developed a super-resolution imaging approach to investigate the size and morphology of aggregates in live cells and neurons (Morten et al., PNAS 2022). This approach is also compatible with in situ imaging of aggregates in brain tissues. We postulated that proteasomes may accumulate into phase-separated droplets to facilitate cytosolic aggregate removal together with additional chaperones, co-proteins and enzymes involved in disaggregation (Mee Hayes, Sirvio and Ye, Front. Aging Neurosci. 2022). Our latest unpublished data suggest that proteasomes accumulate into a novel type of foci body that acts as a degradation centre specifically in response to toxic aggregates (Sirvio et al., in preparation). Such foci are assembled prior to co-localising with aggregates and their formation is dependent on a process called liquid-liquid phase separation (LLPS). Upon closer examination however, these foci are actually gel-like condensates that disperse slowly back into individual proteasomes only when aggregates have been cleared. We further determined that formation of the foci is cytoskeleton- ubiquitination-dependent, and that proteasome activity is not compromised within these foci. Our results indicate that such foci are a novel type of proteasome-dense degradation centre specific to proteotoxic stress.

**BIO**

I studied Biochemistry as an undergraduate in London and undertook PhD research at Cambridge. My training in David Komander's lab at MRC Laboratory of Molecular Biology combined structural biology with biophysical techniques to study molecular details underlying regulation of the ubiquitin-proteasome system (UPS). Following this, I was fortunate to secure funding first through a Henslow Junior Research Fellowship and then a Sir Henry Wellcome Research Fellowship to study the UPS and protein aggregation with super-resolution imaging techniques. Working between David Klenerman's group at Cambridge and Daniel Finley's group at Harvard, I benefited from my fellowship by carving out my own research, which focused on exploiting the UPS system to remove protein aggregates. In 2019, I was awarded a generous start-up fund from the UK Dementia Research Institute to build my own group and consolidate this research direction. My group at Imperial College London continues to study the interplay between UPS, protein aggregation, neurodegeneration and neuroinflammation.

## I4: Macrophage heterogeneity in cancer metastasis and therapy resistance



**Dr Binzhi Qian**
University of Edinburgh

**RESEARCH SUMMARY**

A concise and factual abstract, 11pt Times New Roman is required. The abstract should state briefly the purpose of the research, the key results and major conclusions. It must be able to stand alone, references should be avoided. Non-standard or uncommon abbreviations should be avoided.

Metastasis is the number one major challenge of cancer and accounts for over 90% of total cancer lethality. We have a long-term interest in the role of tumour associated stromal cells, known as the tumour microenvironment. Using multiple genetically engineered mouse models, my previous studies were the first to identify a distinct population of macrophages, a type of innate immune cell, promote tumour cell distal metastasis.

Bone is the major target organ for metastasis of several cancer types including breast and prostate cancer which accounts for over 70% of all metastasis cases. We have now developed several new models of breast and prostate cancer bone metastasis in syngeneic mouse background with complete immune system. Our recent studies illustrated a novel mechanism of macrophage sub-populations promotion of enzalutamide resistance of bone metastatic prostate cancer. Using a combination of wet and dry lab approaches, we aim to significantly improve the understanding of the pathogenesis of metastatic disease and therapy resistance, but also identify novel therapeutic targets and prognostic markers.

**BIO**

Dr. Bin-Zhi Qian is a Reader (tenured) holding a joint appointment in the Centre for Reproductive Health and the Edinburgh CRUK Centre at the University of Edinburgh (UoE), UK. Dr. Qian received his bachelor's degree in Biochemistry from Fudan University in Shanghai, China. He received a Ph.D. degree in Biomedical Sciences from Albert Einstein College of Medicine in New York, USA under the mentorship of Professor Jeffrey Pollard. Following postdoctoral training with Dr. Charles Sawyers at the Memorial Sloan Kettering Cancer Center in New York, he started his independent research group at UoE in 2013.

Dr. Qian is an expert in tumour associated macrophages with an established track record of high-quality research, including original articles in high-impact journals such as Nature, Cancer Discovery, and Journal of Experimental Medicine, as well as invited review articles in Cell, Nature Review Immunology, Trends in Immunology, which have received total more than 13,000 citations. Dr. Qian has received many distinguished awards, including the Prostate Cancer Foundation Challenge Awards, the Cancer Research UK CDF Award, and the European Research Council Starting Award, among many others. He is dedicated to understanding the mechanism of cancer metastasis and developing effective therapeutic approaches by focusing on the interactions among metastatic tumour cells and associated host cell types.

## I5: Circulating tumour biomarkers for prostate cancer diagnosis and precision medicine

**Prof Yong-Jie Lu**
Queen Mary University of London

**RESEARCH SUMMARY**

Both cancer screening for early diagnosis and precision medicine requires frequent repeated sampling for molecular analysis. However, the current gold standard diagnosis is pathological examination of tissues, which is very invasive, difficult for frequent sampling and cannot avoid intratumour heterogeneity caused issues. Liquid biopsy, in particular circulating biomarkers in the blood, will avoid these issues. Blood is a rich source for cancer biomarker development, including circulating cancer cell proteins, DNA

and RNA, exosomes, tumour cells and non-cancer cells and products. Circulating tumour cells (CTCs) has certain advantages over other circulating biomarker, so my team mainly focuses on CTCs. Taking advantage of the blood samples collected for CTC analysis, we also performed plasma exosome miRNA analysis. The cancer type that we work on is prostate cancer (PCa).

Starting our CTC study, we compared the cell size and deformability based Parsortix CTC isolation system with the FDA approved CellSearch and another antibody-based CTC isolation system, isoflux, and found that Parsortix has not disadvantage in capture epithelial CTCs that the other two system mainly capture, and captured many mesenchymal CTCs. Therefore, our further CTC studies used Parsortix. Firstly, we evaluated the prognostic value of CTCs and found that mesenchymal CTCs most significantly predicted poor overall survival. We then analysed PCa patients with and without metastasis and showed that CTCs in combination with prostate specific antigen predict metastasis with AUC of 0.92. We also detected CTCs in some localised PCa cases, indicating micro-metastasis. In our further analysis of CTCs in 155 localised PCa, we found that CTC positivity is associated with clinically significant cancer. In 87 pre-biopsy patients, the combination of CTC positivity, CTC gene expression and prostate specific antigen predicted the biopsy outcome of clinically significant PCa with AUC of 0.927. High number of CTCs before docetaxel treatment (the main chemotherapy of PCa) were found in stable or progression disease, which were further translated into significant poor overall survival. We also found that CTC KLK2, ZEB1, SNAL1 and ADAMTS1 expressions were associated with poor overall survival. We are currently also investigating if pre- and/or post-surgery CTCs can predict radical prostatectomy failure, consequently guide treatment stratification of 'localised PCa'. Surprisingly, during our CTC analysis, we also identified megakaryocyte, which usually located in the bone marrow, in periperial blood and they were strongly associated with good patient prognosis. Using plasma sample isolated during our CTC study, we also analysed plasma exosomal miRNAs for their potential of circulating biomarkers. Exosomal miRNA associated with hormone therapy resistance were first identified by total exosomal miRNA next generation sequencing, further confirmed by q-RT-PCR. The strongest associated miRNA, miR-423-3p, was confirmed in an independent research centre and had a treatment resistance prediction value of AUC=0.879.

In summary, circulating tumour biomarkers are valuable for minimal-invasion PCa diagnosis, metastasis and treatment prediction/monitoring. Analysing different subtypes of CTCs and CTC gene expression is beneficial. Plasma exosomal miRNA also has a high potential as PCa biomarker. We should always Look out for other undiscovered biomarkers in the blood.

**BIO**

Yong-Jie Lu, Professor in Molecular Oncology at Barts Cancer Institute, The Barts and London School of Medicine and Dentistry, Queen Mary University of London. His past work, since 1990 has been focused on identification of genetic alterations and genetic mechanisms in cancer development, progression and therapeutic response. He is a long-term member of prostate cancer International Cancer Genome Consortium and prostate cancer genetic risk study international consortium. In recent years, his research moved into liquid biopsy, in particular circulating biomarker studies, aiming to translate them into cancer diagnosis, prognosis and therapeutic stratification. He is currently the co-chair of the European Liquid Biopsy Society Circulating Tumor Cell Working Group. His findings in using circulating tumour cells as prostate cancer biomarkers have been highlighted in dozens of national and international media. Prof. Lu published nearly 200 peer reviewed papers, in journals including Lancet, Nature Genetics and PNAS and on the editorial board of a few scientific journals, such as Drug Resistance Update and American Journal of Cancer Research.

## I6: How can we use viruses to conquer human cancer?

**Prof Yaohe Wang**
Queen Mary University of London

**RESEARCH SUMMARY**

Following our better understanding of cancer biology and cancer immunity, vast array of immunotherapeutic agents for the treatment of cancer have been developed. Viruses that can selectively infect and destroy cancer cells, known as oncolytic viruses (OVs), are a promising new class of therapeutics for cancer, with having four OVs approved as new drugs for cancer treatment. However, current oncolytic virotherapy is unable to produce a long-term cure in patients, and the treatment has to be delivered directly into the tumour - a route that is not feasible for deeply embedded tumours, or tumours that have spread around the body. In this talk, the speaker will present

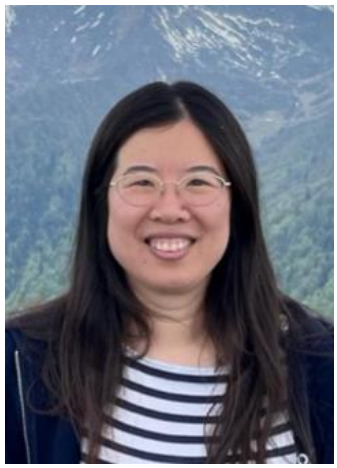
how to develop next generation of oncolytic viruses to cure cancer, in particular how to develop an intravenously deliverable oncolytic virus to conquer difficult to treat cancers. The speaker will also discuss some of the challenges associated with oncolytic virus therapies, and future perspectives in this evolving field.

**BIO**

Dr. Yaohe Wang is a Professor of Cancer Cell and Gene Therapy and the head of Cancer Viro-immunotherapy Laboratory at Barts Cancer Institute, Queen Mary University of London. He is also the chief founder and CSO of VacV Biotherapeutics Ltd, which is a recently established Queen Mary University of London- associated spinout Company. The major research interest in his Lab is to develop novel cancer therapeutic agents and regimens using their unique viral vector platform and novel animal models. Combination of oncolytic viruses with personalised cancer vaccine and CAR-T therapy is the focus of a major current programme. His lab has made several original contributions to the field of cancer virotherapy and has been granted six patents, with a substantial track record of publications in prestigious scientific journals such as “Nature Biotechnology ”etc.. He serves as Editorial Board Member for several pioneer-reviewed Journals and also as a referee for International Founding organizations and Journals. Professor Wang’s long-term research aim is to develop more effective cancer cell and gene therapies based on the genetically engineered oncolytic virus platform, for prevention, diagnosis and treatment of human cancer, and advance them into clinical testing.

### I7: Academic job application

**Dr Guifen Chen**
Queen Mary University of London



**BIO**

Dr Guifen Chen is a Lecturer in Neurobiology at QMUL. Her work focuses on the neuronal basis of multisensory integration, spatial cognition and memory. Her lab uses state-of-the-art techniques such as immersive virtual reality and in vivo electrophysiological/probe recording in mice. Her research is currently supported mainly by funding from BBRSC and the Royal Society.

Dr Chen completed her undergraduate studies in both biology and computer science at East China Normal University in China. She then pursued PhD in neuroscience, conducting research at both East China Normal University and Boston University in the USA. Following that, she undertook postdoctoral research at University College London in the UK. Her work has been published in high-impact journals such as Nature Communications, eLife, and Current biology.

### I8: Fellowship application

**Dr Mattew Swire**
UCL

**BIO**

I received my B.Sc. in Biological sciences, with honours in neuroscience, from the University of Edinburgh in 2011. Following this undertook a research technician position at University College London in the laboratory of Professor John Greenwood at the Institute of Ophthalmology (2011-2012). I then travelled back to the University of Edinburgh to work towards a Ph.D. at the Scottish Centre for Regenerative Medicine with Professor Charles ffrench-Constant and Professor David Lyons (2012-2017). After completing my Ph.D. I continued working with Charles ffrench-Constant and David Lyons as a post-doctoral researcher (2017-2019). I then undertook a research associate position back at University College London at the Institute for Biomedical Research with Professor William Richardson. In 2023 I was awarded an MRC career development award

to establish my own independent research team at University College London to investigate how oligodendrocyte linage cells shape neural circuits during learning.

**I9: Funding application**
**Prof Huiliang Li**
UCL

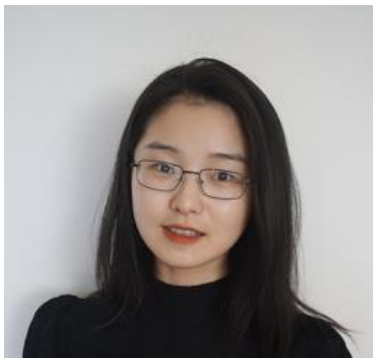
**BIO**
My lab is focused on deciphering glia functions in the healthy and diseased brain. I have extensive experience of studying myelin in neurological diseases with transgenic mouse models and a sustained track record of securing funding from research councils and charity funding bodies in the UK. We discovered a molecular mechanism underlying oligodendrocyte fate determination from neural stem cells and building on this work, developed a recipe of small molecules for generation of oligodendrocyte lineage cells for potential use in cell therapy. Through collaborative work, we revealed an essential role for adult oligodendrogenesis in motor learning and revealed myelin/cholesterol metabolism changes in mouse models of autism spectrum disorder and Alzheimer diseases. Over these years as an independent researcher, I have regularly appraised grant proposals for a dozen or so funding bodies and reviewed research papers for numerous journals, and have served as an associate editor of *Frontier in Cellular Neuroscience* and a member of the editorial board of *Glia*.
I have been in the Pool of Experts for Biotechnology and Biological Sciences Research Council (BBSRC), and served as a Core member to BBSRC Research Committee C since 2022. In addition, I am the 28th committee chair of the Chinese Life Scientists Society in the UK (CLSS-UK) and a vice president of Association of British Chinese Professors (ABCP) 2023-2025 committee.

**I10: NHS career progression**
**Dr Zhangjie Su**
Cambridge University Hospitals NHS

**BIO**
Bachelor degree (Clinical Medicine) from Sun Yet-sen University, Master degree (Surgery) from Nankai University, and PhD degree (Clinical Neurosciences) from University of Manchester.
After completing an NIHR sponsored post-doc research fellowship at the University of Birmingham, Dr Su returned to clinical practice within the NHS while maintaining research activities in neuro-oncology, neurotrauma, and neurodegenerative diseases. He underwent surgical residential training at Queen Elizabeth Hospital Birmingham and Salford Royal Hospital, and neurosurgery specialty training at the National Hospital for Neurology and Neurosurgery, and the Walton Centre. He is currently in higher fellowship training at Addenbrooke's Hospital, Cambridge.
Dr Su is a member of the Royal College of Surgeons of England, and the Society of British Neurological Surgeons, as well as the vice president of the Society of Chinese Medical Practitioners UK. He is also an honorary senior clinical lecturer at the University of Birmingham, visiting professor at Hunan Children's Hospital, grant application reviewer for the Medical Research Council (MRC), and reviewer for several academic journals (e.g. Journal of Nuclear Medicine, PLoS One, BMJ Open, etc.). His research work has been published in high impact journals such as the Lancet, New England Journal of Medicine, PLoS Medicine，Journal of Nuclear Medicine，Movement Disorders，Journal of Neurotrauma，British Journal of Sports Medicine, and JAMA Neurology, etc. He has also contributed to book chapters in the Oxford Textbook for Neurological Surgery, and Neuroanatomy Guidance for Successful Neurosurgical Interventions.

**I11: Industrial job application**

**Dr Yue Du**
**AstraZeneca**



**BIO**
杜越博士现任阿斯利康制药公司（英国剑桥）高级研发科学家，牛津大学临床转化医学系博士后，爱丁堡大学罗斯林研究所博士。美国基因与细胞治疗协会 2021 年‘卓越研究奖’得主， 英国呼吸疾病基因治疗联盟成员， The Oxford Global 会议咨询委员会成员，SCI 杂志审稿人与特刊主编。研究领域从动物医学到体细胞克隆，到基因编辑与针对罕见病的基因递送疗法的开发与临床转化，新冠疫情期间带领团队开发了首个针对免疫力低下人群的抗新冠病毒感染免疫预防方案。 在创新创业方面带领团队获得过全英高层次人才创业大赛暨第三届硬科技创业大赛二等奖，‘创客未来’英国赛区二等奖，教育部‘春晖杯’创新创业大赛优胜奖等。

# Oral Presentations

## Abstracts

**O1: Stoichiometry and function studies of human cardiac sHsps**
**Zihao Wang, Justin Benesch**
Kavli Institute for Nanoscience Discovery, University of Oxford
**Abstract:**
Human small heat shock proteins (sHsps) are a family of proteins that work on the first-line to maintain the proteostasis. Within this family, cardiovascular heat shock protein (cvHsp, HspB7) and HspB8 (Hsp22) are the two members that perform their primary functions in cardiac tissues and skeletal muscles. Crystallography and native mass spectrometry are the main techniques we used to characterise the proteins. We obtained the first crystal structures of cvHsp and HspB8, and we found that unlike most of the well-studied human sHsps forming large oligomers based on dimeric subunits, both cvHsp and HspB8 are dominantly monomeric at dynamic conditions. Based on our results, we target the key residue contacts for human sHsp dimerisation and renew our knowledge towards the canonical sHsp dimer interface. The absence of cvHsp in heart is fatal and can cause the aggregation of the actin-linking protein filamin C (FLNC). We confirmed the interaction between cvHsp and FLNC in mice hearts, and we found that cvHsp is binding the dimerised domain 24 (d24) of FLNC and competes to its homo-dimerisation. We solved the complex crystal structure and noticed a vital residue on cvHsp to make this interaction specific. We observed that phosphorylation at a threonine residue or a tyrosine residue on FLNC under Covid-19 infection and cancer respectively has opposite and significant effects in mediating the FLNC dimerisation, and its interaction with cvHsp. We explained these effects with detailed structural analysis and the results supported the dual cellular roles of FLNC. On the other hand, HspB8 is to bridge the dysfunctional filamins into the chaperone-assisted selective autophagy (CASA) complex for degradation. We found a region which is buried in functional filamin proteins that can be permanently exposed when the filamins are overstretched. We extracted this region and noticed that the disease mutants on a hot spot residue of HspB8 had weaker binding capacities compared to the wild type, which explains the role of HspB8 in CASA as well as the mechanism of its disease mutants. Overall, our study provides new and deep insights into the nature on human cardiac sHsps using a variety of biophysical techniques.

**O2: Interpretable deep learning for light-field microscopy neuroimaging**
**Pingfan Song**
Department of Engineering at the University of Cambridge
**Abstract:**
Understanding how networks of neurons process information is one of the key challenges in modern neuroscience. A necessary step to achieve this goal is to be able to observe the dynamics of large populations of neurons over a large area of the brain. Light-field microscopy (LFM), a type of scanless microscope, is a particularly attractive candidate for high-speed three-dimensional (3D) imaging. It captures volumetric information in a single snapshot, allowing volumetric imaging at video frame-rates. Imaging neuronal activity using LFM calls for the development of novel computational approaches that fully exploit domain knowledge embedded in physics and optics models, as well as enabling high interpretability and transparency. Deep neural networks have demonstrated remarkable success in solving real-world problems in signal and image processing. However, their practical deployment and

development in biological imaging are hindered by the lack of interpretability and the need for large training sets. To this end, we propose a model-based explainable deep learning approach for LFM, which incorporates signal processing theory and wave-optics theory into light-field microscopy imaging to fill this gap. Different from purely data- driven methods, the proposed approach integrates wave-optics theory, sparse representation and non-linear optimization with the artificial neural network. In particular, the architecture of the proposed neural network is designed following precise signal and optimization models. Moreover, the network's parameters are learned from a training dataset using a novel training strategy that integrates layer-wise training with tailored knowledge distillation. Such design allows the network to take advantage of domain knowledge and learned new features. It combines the benefit of both model-based and learning-based methods, thereby contributing to superior interpretability, transparency and performance. By evaluating on both structural and functional LFM data obtained from scattering mammalian brain tissues, we demonstrate the capabilities of the proposed approach to achieve fast, robust 3D localization of neuron sources and accurate neural activity identification.

**O3: Annelid functional genomics reveal the origins of bilaterian life cycles**
**Yan Liang**
Queen Mary University of London
**Abstract:** Indirect development with an intermediate larva exists in all major animal lineages, which makes larvae central to most scenarios of animal evolution. Yet how larvae evolved remains disputed. Here we show that temporal shifts (that is, heterochronies) in trunk formation underpin the diversification of larvae and bilaterian life cycles. We performed chromosome-scale genome sequencing in the annelid Owenia fusiformis with transcriptomic and epigenomic profiling during the life cycles of this and two other annelids. We found that trunk development is deferred to pre-metamorphic stages in the feeding larva of O. fusiformis but starts after gastrulation in the non-feeding larva with gradual metamorphosis of Capitella teleta and the direct developing embryo of Dimorphilus gyrociliatus. Accordingly, the embryos of O. fusiformis develop first into an enlarged anterior domain that forms larval tissues and the adult head. Notably, this also occurs in the so-called 'head larvae' of other bilaterians, with which the O. fusiformis larva shows extensive transcriptomic similarities. Together, our findings suggest that the temporal decoupling of head and trunk formation, as maximally observed in head larvae, facilitated larval evolution in Bilateria. This diverges from prevailing scenarios that propose either co-optionor innovation of gene regulatory programmes to explain larva and adult origins.

**O4: Shape Memory Oleogel Composites with Optical Modulation**
**Qiukai Qi**
University of Bristol
**Abstract:** The increasing impact of technology disposal and e-waste on the environment has driven robotics scientists to develop more sustainable robots. Biodegradable and edible shape-memory polymer artificial muscles represent an important robotic component that enables environmentally modulated deformation and movement. The need for finer behavioral control of artificial muscles also requires proprioceptive functionality. Here we report the first edible shape-memory oleogel composites that monolithically couple shape-memory actuation and color-change sensing within one material. The shape recovery and color change capabilities were characterized and two sustainable soft robots, a robotic gripper and a biomimetic flower, were constructed to demonstrate their potential for the development of sustainable and edible robotics.

**O5: Understanding CDK4/6 Inhibitor Resistance: The Cell Cycle Position Matters**
**Xu Shen**
1UCL Cancer Institute, and 2UCL Institute of Prion Diseases, University College London, London, UK
**Abstract:** Cyclin-dependent kinase 4/6 inhibitors (CDK4/6i) used in combination with estrogen receptor (ER) targeting agents have emerged as a significant component in the current management of ER-positive (ER+) metastatic breast cancer. However, the prevalence of relapse during therapy seriously limits the long-term impact of this treatment. The underlying causes of this near-ubiquitous relapse are still poorly understood. In this study, we employed fluorescent ubiquitination-based cell cycle indicator (FUCCI) technology to analyse the response of breast cancer cells to CDK4/6i through single-cell resolved time-lapse microscopy imaging. Our findings reveal a cell cycle-linked nongenetic mechanism of drug tolerance in breast cancer cells. Specifically, cells exposed to CDK4/6i during the S, G2 or M phases were more prone to escape the G1 arrest compared to cells receiving CDK4/6i during the G1 phase. Furthermore, cells experiencing CDK4/6i in S/G2/M phases showed a higher capability to re-enter cell cycles after long-term continuous CDK4/6i treatment replicating the treatment schedule used in patients. Notably, cells

pre-treated with clinically used drugs that cause transient G1 accumulation drastically reduced the observed drug tolerance, presenting a potentially novel approach to enhance the clinical response to CDK4/6i. Collectively, our work points to the possibility that cell cycle-linked refractory response to CDK4/6i, as opposed to genetic resistance, underlies the ubiquitously observed disease progression under therapy. Importantly, it advocates for the use of scheduled treatment with agents that modulate cell cycle distribution in cancer tissues towards G1 as a thus far unexplored therapeutic strategy to prevent relapse in patients with ER+ metastatic breast cancer receiving CDK4/6i treatment.

**O6: Mutational processes in normal kidneys across countries with varying cancer incidence rates**

**Yichen Wang[1], Sarah Moody[1], Behnoush Abedi-Ardekani[2], Yvette Hooks[1], Jingwei Wang[1], Laura Humphreys[1], Tatsuhiro Shibata[3], Paul Brennan[2], and Michael R. Stratton[1]**

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**Abstract:** In recent years, large-scale whole genome sequencing of multiple types of cancer across multiple continents has been conducted as part of the “Mutographs” Cancer Grand Challenge to uncover unknown causes of cancer through detection of signatures of mutational processes operative during cancer development. Distinct mutational signatures have recently been detected by “Mutographs” in Renal Clear Cell Carcinomas (RCC) from different parts of the world. However, whether the detected mutational signatures are present in normal tissues or are initiated after tumorigenesis remains unknown. In general, there has been a lack of knowledge of somatic mutations in normal cells primarily due to technological barriers to detection of somatic mutations in highly polyclonal normal tissues. However, a recently developed duplex sequencing technology, NanoSeq, uses copies of both strands of each DNA molecule to reduce sequencing errors to 10-9. With NanoSeq, we are able to detect somatic mutations in polyclonal tissues including the normal kidney. In this study, we used NanoSeq to sequence 288 tumor-adjacent normal kidney samples from multiple countries with varying RCC incidence. Subsequently, we conducted agnostic signature extraction using a Hierarchical Dirichlet Process to investigate whether the region specific mutational signatures found in cancers can be extracted from normal kidney tissue. The normal kidney tissues we sequenced have paired RCC whole genome sequencing data from the same individual. Therefore, the mutational profiles of normal kidney can be compared to paired cancer samples to ascertain the timing of the mutational processes causing the mutational signatures found in the cancers. We confirmed that a predominantly T>C mutational signature that is highly enriched in Japanese RCC is present in normal kidney samples. A strong transcriptional strand bias in this signature provides circumstantial evidence that it is likely to have been caused by DNA damaging agents causing bulky DNA adducts which may be of environmental origin. A subset of RCC samples from Serbia and Romania had mutational signatures caused by aristolochic acid (AA). We found different dominant AA-related signatures in tumors compared to their matched normal tissues, potentially indicating different mutagenic or repair mechanisms between normal and cancer cells. Levels of SBS40, which is of unknown cause, were elevated in normal kidneys from the Czech Republic compared to other countries, and may contribute to the high RCC incidence in this country. In summary, this study provides the first systematic investigation of somatic mutations in normal kidney, revealing different mutational processes in different geographic regions and in cancer versus normal kidneys.

**O7: Optogenetic Miro cleavage reveals direct consequences of real-time loss of function in Drosophila**

**Jingnu Xia**

King's College London, Department of Basic and Clinical Neuroscience

**Abstract:** Miro GTPases control mitochondrial morphology, calcium homeostasis and regulate mitochondrial distribution by mediating their attachment to the kinesin and dynein motor complex. It is not clear, however, how Miro proteins spatially and temporally integrate their function as acute disruption of protein function has not been performed. To address this issue, we have developed an optogenetic loss of function ‘Split-Miro’ allele for precise control of Miro-dependent mitochondrial functions in Drosophila. Rapid optogenetic cleavage of Split-Miro leads to a striking rearrangement of the mitochondrial network, which is mediated by mitochondrial interaction with the microtubules. Unexpectedly, this treatment did not impact the ability of mitochondria to buffer calcium. While Split-Miro overexpression is sufficient to augment mitochondrial motility, sustained photocleavage shows Split-Miro is surprisingly dispensable to maintain elevated mitochondrial processivity. Furthermore, functional replacement of

endogenous Miro with Split-Miro identifies its essential role in the regulation of locomotor activity in adult flies, demonstrating the feasibility of tuning animal behaviour by real-time loss of protein function.

**O8: Mitotic chromosome formation by DNA entrapment inside the condensin ring**
**Mingzhe Tang**
The Francis Crick Institute
**Abstract:** Condensin is the structural protein complex that builds and maintains the characteristic rod-shaped mitotic chromosome structures. Condensin mutations causes chromosome segregation defects and genomic instability, therefore are commonly found in many types of cancer cells. However, the exact mechanism employed by condensin to build mitotic chromosomes is still under debate. Here, we explore how condensin might build chromosome via topological entrapment of chromatin. Using bulk biochemical and single molecule experiments with purified fission yeast condensin, we observe that individual condensin complexes sequentially and topologically entrap two double stranded DNAs (dsDNAs). We then provide complementary in vivo evidence for such sequential DNA capture, in the form of condensin-associated chromatin contacts between chromosomes. Our results support a model in which condensin acts in mitotic chromosome formation by sequential dsDNA-dsDNA capture.

# Poster Presentations

## Abstracts

**P1:Rare Diseases in Developing Countries: Insights from China's Collaborative Network**
**Jishizhan Chen1, Yihan Li2, Jinke Chang1,***
1 UCL Centre for Biomaterials in Surgical Reconstruction and Regeneration, Division of Surgery & Interventional Science, University College London, London, NW3 2PF, UK; 2 Department of Politics and International Studies, Cambridge University, Cambridge, CB3 9DP, UK
Rare diseases (RDs) present significant challenges for healthcare systems in developing countries, where policymakers often lack appropriate templates and frameworks. This article explores a comprehensive approach to tackling RDs, using China as a case study. By combining top-down strategies and bottom-up interventions, the government takes the lead in formulating robust laws, policies, and guidance to coordinate national resources effectively. This ensures the establishment of necessary infrastructure and resource allocation for RD initiatives. Meanwhile, local authorities and non-governmental organizations (NGOs) play a pivotal role in policy localization, tailoring national policies to suit regional needs, and filling policy gaps through targeted interventions. This integrated approach creates a synergy between national and local stakeholders, fostering collaboration and knowledge-sharing. It empowers local authorities to adapt policies to the unique circumstances of their regions, while NGOs provide vital support services to affected individuals and families. Moreover, the government's top-down strategies facilitate the establishment of specialized centers of excellence for RD diagnosis, treatment, and research, further strengthening the healthcare system's response to these complex conditions. The experiences and lessons learned from China's efforts can inspire other developing countries in their journey to improve RD healthcare. By adopting similar strategies and tailoring them to their specific contexts, these nations can enhance their rare disease management and support systems. Collaboration between developing countries at similar stages of development can foster a global exchange of best practices, ultimately contributing to a collective effort in combating RDs.

**P2:Blocking CX3CR1+ Tumor-associated Macrophages Synergize with anti-PD-1 Therapy in Hepatocellular Carcinoma**
**Xiaonan Xiang1,2,3,4, Jianguo Wang5,6,7, , Hui Zhang7, , Haiyang Xie4,8, , Kourosh Saeb-Parsy2,9, , Xiao Xu1,3,4,7***
1.Department of Zhejiang University School of Medicine, Hangzhou, China; 2.Department of Surgery, University of Cambridge, and NIHR Cambridge Biomedical Research Centre, Cambridge, United Kingdom; 3.Key Laboratory of Integrated Oncology and Intelligent Medicine of Zhejiang Province, Hangzhou, China; 4.NHFPC Key Laboratory of Combined Multi-Organ Transplantation, Hangzhou, China; 5.Department of Hepatobiliary and Pancreatic Surgery, Hangzhou First People's Hospital, Hangzhou, China; 6.Radiation Oncology/Steele Laboratories, Massachusetts General Hospital, Boston, United State; 7.The Fourth Clinical Medical College of Zhejiang Chinese Medical University,

Hangzhou, China; 8.Department of Hepatobiliary and Pancreatic Surgery, First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China; 9.Transplant Unit, Addenbrooke's Hospital, Cambridge, United Kindom. **Background**: Tumor-associated macrophages (TAMs) are recognized as immune suppressor in tumor microenvironment (TME). However, due to their phenotypic and functional heterogeneity, their specific role in immune evasion and immunotherapy resistance remains unclear. Here, we aim to investigate the role and mechanism of CX3C motif chemokine receptor 1 (CX3CR1) positive TAMs, a special phenotype of TAMs, on HCC immunotherapy resistance and recurrence after surgery. **Methods**: Dynamic changes of tumor-infiltrating CD8+ T cells and myeloid cells were detected in mouse orthotopic HCC model during immunotherapy. In vitro and in vivo co-culture experiments were applied to explore the possible interaction between CX3CR1+ TAMs and CD8+ T cells. Metabolomics was used to explore changes in tumor metabolic profiles induced by immune challenge. Synergistic therapeutic effects of CX3CR1 neutralizing or COX2 blockade with anti-PD-1 mAbs were validated using different mouse models of HCC. The dynamic changes of peripheral blood PGE2 and the number of liver cancer tissue infiltrating CX3CR1+ TAMs were detected in HCC patients receiving anti-PD-1 therapy. **Results**: We detected high accumulation of CX3CR1+ TAMs during anti-PD-1 therapy of HCC (Fig. 1A-B). CX3CR1+ TAMs can induce T cell dysfunction by IL-27 secretion, thereby mediating immunotherapy resistance (Fig. 1C-D). Mechanically, prostaglandin E2 (PGE2) produced by immune-attacked HCC tumor cells induced increased expression of CX3CR1 inTAMs. Blocking tumor PGE2 releasing or targeting CX3CR1+ TAM can enhance the efficacy of anti-PD-1 mAb (Fig. 1E-F). Moreover, low infiltration of CX3CR1+ TAM or stable serum PGE2 levels can predict good responses of anti-PD-1 mAb-based immunotherapy for HCC patients (Fig. 1G). **Conclusions**: Targeting CX3CR1+ TAM may be a promising strategy to enhance the efficacy of anti-PD-1 therapy in HCC.

**P3: IN-VITRO MODELLING OF MITRAL VALVE THERAPIES**

**W. Zhou 1, S. Bozkurt 2,3, M.H. Cheang 4, S. Schievano 2,3, G. Burriesci 1,5**

1 UCL Mechanical Engineering, Cardiovascular Engineering Laboratory, University College London, UK. 2 UCL Great Ormond Street Institute of Child Health. 3 Great Ormond Street Hospital for Children. 4 Barts Health NHS Trust, University College London Hospital, London, UK. 5 Ri.MED Foundation, Bioengineering Group, Palermo, Italy.

**Introduction**: Mitral regurgitation (MR), the reverse blood flow through the mitral valve (MV), is the most common valvular heart disease. Various repair and replacement treatments are available to restore the function of the valve, but it is often difficult to evaluate and compare the risks and results of these alternative approaches, due to the great anatomical heterogeneity of mitral pathologies. Patient-specific in vitro simulation of MV function could provide an essential benchmark for potential treatments and to help predict post-surgical outcomes, thus assisting clinical pre-operation decision-making to increase the success rate of surgeries. However, this solution requires patient-specific anatomical heart chambers and the ability to finely control the anatomical parameters that monitor the MV functions, such as the position of the papillary muscle (PM) and the mitral annulus shape. **Methods**: In this study, a novel test rig is developed to incorporate and test MV functions into a cardiovascular hydrodynamic testing system (ViVitro System, ViVitro Labs, Inc., Canada), available at the Cardiovascular Engineering Laboratory, UCL Mechanical Engineering. The implemented system enables the accurate positioning of each PM to be adjusted in 3 dimensions, while replicating the ventricular shape with a silicone sack. **Results & Discussion**: The test rig has been successfully used to replicate the function of a porcine MV, evaluating the effect of the positions of the PMs on MV performances. Results were consistent with data from in vivo observations (1) from swine models. Further study investigated the impact of cardiac output and PM positions on MV performance. The system has been employed to test a novel prosthetic valve concept and a number of percutaneous repair implants. These in vitro implants have enlightened potential problems which are difficult to predict in numerical studies, providing a substantial support in the design optimisation of the therapeutic solutions and in their assessments. The test rig can also be employed as a useful tool for surgeons training on new treatments. **Conclusion**: The developed test rig has been used as a physiological model, demonstrating its ability not only to evaluate existing therapies but also to support the development of new solutions.

**P4: Visible Light-driven Ultra-stretchable Polyurethane Elastomer Bionic 'Photo-Fingers'**

**Lei Wu1, Xia Huang1, Meng Wang1, Jishizhan Chen1, Jinke Chang1, Han Zhang2, Xuetong Zhang3, Andrew Conn4, Jonathan Rossiter4, Martin Birchall5, Wenhui Song1 ***

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Robotics Laboratory, University of Bristol, Bristol BS8 1UB, UK; 5.UCL Ear Institute, Royal National Ear Nose and Throat and Eastman Dental Hospitals (UCLH NHS Foundation Trust) and University College London, London, United Kingdom.
Light-driven soft robotic actuators based on photo-responsive materials can be utilised to mimic the biological functions, such as swimming, walking, crawling without involving rigid and chunky electromechanical actuations. However, a robust photo-responsive material with desired mechanical and biofunctional performance, facile production and manufacuring process for constructing potential light-driven soft robotics for real-life applications, is yet to be developed. Herein, we report a new visible light-responsive elastomer synthesized by introducing photo-responsive moieties (i.e. azobenzene derivatives) into the main chain of poly(ε-carporlactone) based polyurethane urea (PAzo). PAzo elastomer shows controllable light-driven stiffness softening while maintaining excellent hyperelasticity (stretchability of 575.2% and strength of 44.0 MPa) due to its unique nanostructure and nanophase transition. A visible light-driven bilayer actuator consisting of PAzo and polyimide film has been developed, demonstrating two distinct bending modes under illumination with varying light intensities. The corresponding actuation mechanism in correlation to photothermal and photochemical coupling effects has been proven through experimental analyses and theoretical calculations. An exemplar application is demonstrated by visible-light-controlled soft robotic 'fingers' playing music through touching a piano on smartphone. The robustness of PAzo elastomer and scalable process plus its good biocompatibility render design and manufacture of reproducible light-driven wearable/implantable robots or assistive devices for potential medical rehabilitation and surgical reconstruction.

**P5: Could transgenic microalga Chlamydomonas reinhardtii boost shrimp growth and protect shrimp from viral diseases?**

**Luyao Yang1, Patai Charoonnart2,3, Harry Jackson1, Brenda Parker4, Vanvimon Saksmerprome2,3, Saul Purton1***
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Growth rates and disease control are two major limiting factors in aquaculture, but both could be addressed through oral delivery of affordable therapeutics (Charoonnart et al., 2018). The edible microalga Chlamydomonas reinhardtii has emerged as a promising platform for producing recombinant therapeutics for the aquaculture industry (Jackson et al., 2021). Fish growth hormones (fGHs) have been shown to promote the growth of fish and shellfish (Cavari et al., 1993; H. L. Chen et al., 2008; Dita Puji Laksana; Siti Subaidah; Muhammad Zairin Junior; Alimuddin, 2013; Ng et al., 2016; Rothan et al., 2014; Sekar et al., 2015)and specific double-stranded RNA (dsRNA) molecules designed to key viral genes can serve as RNA-based vaccines (Charoonnart et al., 2019; Y. G. Chen & Hur, 2021; Posiri et al., 2013). When taken up by animals, the dsRNA can trigger the RNA interference (RNAi) mechanism and produce small interfering RNA (siRNA) that silence viral genes (Y. G. Chen & Hur, 2022). Traditional methods for delivering therapeutics in aquaculture involve purification, cold chain storage, and injection by hand, often making them technically challenging, prohibitively expensive and hindering widespread use (Charoonnart et al., 2018). Our study sets out to produce fGHs and dsRNAs in the chloroplast of C. reinhardtii to develop a system for whole-cell bio-encapsulation and oral delivery. Initial studies this summer using shrimp as model will focus on the optimisation of fGHs and dsRNA administration doses, shrimp growth and viral challenge performance, as well as optimisation of a low-cost 'hanging bag' photobioreactor system used for scale-up production of the algae to produce sufficient dried biomass for further shrimp feeding trials.

**P6: Distribution and microstructure of elastic fibre system in functionally distinct.Shan Lu1, Binghao Chai2, Helen Birch1.**

1 Research Department of Orthopaedics and Musculoskeletal Science, University College London, Royal National Othopaedic Hospital, Stanmore HA7 4LP, United Kingdom. 2Research Department of Computer Science, University College London, London, WC1E 6BT, United Kingdom.
**Introduction**: Tendon consists of collagen fascicles bound together by the interfascicular matrix (IFM). Recent work has shown that the IFM plays an important role in tendon elongation by allowing sliding of fascicles relative to each other. The IFM is more extensive in high-strain energy storing tendons and enriched with elastin. The elastic fibre system consists of varying amounts of elastin deposited on a scaffold of microfibrils (mainly fibrillin -1 and-2). The relative proportions of elastin and fibrillins differ between elastic fibre types, resulting in elastic fibres with different

morphologies and mechanical properties. The distribution and the microstructure of elastic fibres in tendon has not been fully examined. We hypothesize that the elastic fibre types differ in energy-storing and positional tendons and become more disordered with ageing. **Materials and Methods:** The energy-storing superficial digital flexor tendon (SDFT) (n=8) and positional common digital extensor tendon (CDET) (n=8) were harvested from young (4–7 years, n=4) and old (18–22 years, n=4) horses. Fascicle and IFM morphology were visualised using H&E stain. Elastic fibre type was investigated using a combination of Millers and Weigert's stains and immuno-staining for elastin, fibrillin-1, and fibrillin-2. Tendon sections were observed using second harmonic generation (SHG) confocal microscopy. **Results:** Thick, mature, elastin-containing elastic fibres were more numerous in the SDFT and predominantly seen in the IFM, where they had an oblique arrangement. The thinnest fibres, likely lacking elastin, were mostly seen in the fascicles and the CDET had significantly more than the SDFT. Fibrillin-2 staining was found in the fascicles and IFM whereas fibrillin-1 was mostly in the IFM. Both fibrillin types were more abundant in the positional CDET. In the IFM, the elastin formed a peri-cellular meshwork and amorphous collagen bridged between elastin fibres. In old tendons, the elastin appeared more disordered and fragmented, especially in the SDFT. **Discussion:** The results suggest that specialised mechanical properties are achieved, in part, by differences in the elastic fibre type and distribution. The thick, elastin-containing fibres in the SDFT IFM allow high strains whereas the positional CDET has more fascicle located, thin, stiffer, fibrillin fibres. Ageing is detrimental to both.

**P7: A Genomewide Association Study of Antipsychotic Induced Extrapyramidal Side Effects**
**Kai Yao, Israa Elbashir, Zain Ahmad, Abdullah Lodhi, Nick Bass, Andrew McQuillin**
Molecular Psychiatry Laboratory, University College London
**Background:** Antipsychotics are the cornerstone of the acute and ongoing schizophrenia treatment. However, more than 50% schizophrenia patients can develop at least one type of significant side effects after taking antipsychotics in the long term. Of these serious effects, around 40% patients would develop one type of extrapyramidal side effects (EPSE) which are movement abnormalities that can lead to severe impairment and reduction in the patient's quality of life. Being able to identify patients who are at higher risk of developing EPSE could help improve treatment outcomes. **Methods:** We performed a genome-wide association study (GWAS) of 1017 European schizophrenia patients who have been treated with antipsychotics. 587 (57.5%) of them were recorded to have at least one type of defined movement abnormalities including dyskinesias, Parkinsonism, Akathisia, or Dystonia. We then conducted a meta GWAS of 1755 samples with data from a previous GWAS which used the Abnormal Involuntary Movement Scale (738 additional schizophrenia patients) using METAL. We calculated the polygenic risk scores (PRS) for schizophrenia, major depression, Lewy body dementia and Parkinson's disease using the PRS-CS method with the latest available reference GWAS. The PRS were standardised using the z-score method then we applied logistic regressions to test whether risk of EPSE was associated with differences in these PRS. The first three principal components from the population stratification and the chip type were included as covariates to adjust for confounding in all analyses. **Results:** The SNP rs10892599 located in the GRIK4 gene was associated with the risk of developing EPSEs (p= 4.218e-08). GRIK4 encodes the kainic acid-type glutamate receptor 1 (KA1) subunit, which co-assembles with other glutamate receptor subunits to form cation-selective ion channels. Abnormal glutamate signalling has been implicated in the aetiology of schizophrenia by various lines of evidence. However, we could not find any evidence to suggest that patients who developed EPSE might have any different PRS according to our regression results. **Discussion:** Overall, this is the first GWAS study that observed associations between GRIK4 and EPSE symptoms indicating the potential linkage between glutamate channels and EPSE. It has been hypothesised that synergistic interactions between mitochondrial defects, oxidative stress and glutamatergic stimulation may create the conditions for the development of the nigrostriatal damage that characterizes PD. However, our analyses on PRS could not provide further evidence for such arguments. One major limitation of the study was that we did not separate the antipsychotic and EPSE types. The current study may also suffer from common limitations of GWAS and these results require further validation. Future meta-analyses with increased sample size and widened population coverage are needed.

**P8: Gene-environment correlation: The role of family environment in academic development**
**Quan Zhou1, Agnieszka Gidziela1, Ziye Wang1, Andrea Allegrini2,3, Rosa Cheesman2,4, Jasmin Wertz5, Robert Plomin2, Kaili Rimfeld2, & Margherita Malanchini1,2**
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Academic achievement is partly heritable and highly polygenic. However, the pathway from genotype to its phenotypic expression is likely to be mediated or moderated by environmental exposures. The environmental factors linking individuals' genetic propensities to manifest differences in academic achievement over development have not been systematically investigated. We systematically investigated the role of multiple aspects of the family environment in mediating the polygenic score (PGS) prediction of academic achievement throughout compulsory education. We included data on academic achievement (teaching ratings and exam scores) and family environments (including family socioeconomic status, parenting and home environments) collected from the twins and their parents at ages 7, 9, 12 and 16, as well as geocoded indices of neighbourhood quality. We constructed PGSs for educational attainment, cognitive and noncognitive skills. Three core findings emerged. First, we found that several aspects of the family environment, but not the wider neighbourhood context, consistently mediated the PGS prediction of academic achievement over development, indexing widespread gene-environment correlation. Second, we found that family environments were more robustly linked to noncognitive genetic effects on academic achievement than to cognitive PGS effects. This is particularly striking when considering the comparatively weaker predictive power of the noncognitive PGS. Third, we conducted 1-1-1 multi-level mediation analyses to separate between-family effects (also capturing passive gene-environment correlation) from within-family effects. We found that the mediating role of family environments was nearly exclusively observed for between-family PGS effects, which suggests that family environmental contexts shape academic development largely via passive gene-environment correlation processes.

**P9: Gestational age at birth and cognitive outcomes in term-born toddlers: a multi-context study**

**Zeyuan Sun1,2, Lu Zhang3,4, Qiaoyue Ge3,4, Chenghan Xiao3,4, Shona Falconer2, Andrew Chew2, Chuan Yu3,4, David Edwards2, Chiara Nosarti1,2, Zhenmi Liu3,4**

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Introduction: Previous studies have highlighted an association between gestational age (GA) at birth and early developmental outcomes, indicating that children born at a younger GA are more likely to experience long-term cognitive delays, poor school performance, and mental health problems. Recent evidence suggests that this association may not be confined to preterm births (<37 weeks' GA) and may also be noticed in term births (37-42 weeks' GA). However, the possible influence of clinical and environmental factors in understanding the association between GA and developmental outcomes is not fully understood. This study therefore aimed to explore the association between GA and early developmental outcomes in toddlers born at term in the United Kingdom (UK) and China, after accounting for individual- and area-level environmental variables. Methods: This was a multi-context longitudinal study. Participants were term-born toddlers drawn from two cohorts from UK and China, the Developing Human Connectome Project (dHCP) and the Sichuan Multi-stratified Infants and Early Life (SMILE) study. Early cognitive outcomes were assessed by the Bayley Scales of Infant and Toddler Development-Third Edition (Bayley-III) at 18 months of age in the dHCP, and at 6 months of age by the Second Edition (Bayley-II) in the SMILE study. T-tests, likelihood ratio F-tests and linear regression models were conducted to examine the association between GA at birth and early cognitive outcomes for each cohort separately. A number of clinical and environmental variables, including maternal depressive symptoms (measured by the Edinburgh Postnatal Depression Scale, EPDS), Index of Multiple Deprivation (IMD) and birth weight were modeled as possible confounders in the analyses. Results: In the dHCP, longer GA (unit: week) was associated with better cognitive development (B=1.35 [0.33, 2.37], p=0.010) and motor development (B=1.49 [0.59, 2.39], p=0.001), after controlling for gender, IMD score, maternal depression and birth weight; in the SMILE study, longer GA (unit: week) was also associated with better mental development (B=2.47 [1.60, 3.34], p<0.001) and psychomotor outcomes (B=2.91 [2.01, 3.82], p<0.001), after controlling for gender, parents' education, family yearly income, maternal age, maternal depression, and birth weight. Conclusions: Toddlers born at term can benefit from longer GA, which is associated with more favourable early cognitive and psychomotor developmental outcomes. This association could be explained, at least in part, by maturing development of thalamo-cortical and cortico-cortical connectivity, which is established during the third trimester of

gestation. These findings were similar in two birth cohorts from the United Kingdom and China, providing cross-cultural evidence of robust extrapolation.

**P10: Extracellular matrix in bone metastatic prostate cancer**

**Cheng-Bin Zhang1 and Bin-Zhi Qian1,2**

1Centre for Reproductive Health, Queens Medical Research Institute 2Edinburgh Cancer Research UK Centre, University of Edinburgh

Prostate cancer is the most common male cancer worldwide. Although the clinical efficiency of androgen-deprivation therapy (ADT) in inhibiting cancer progression has been well-established, patients who develop metastatic castration-resistant prostate cancer (mCRPC) at the late stage of the disease will no longer respond to it. Nearly 90% of mCRPC cases occur in bones, making bone mCRPC a significant challenge in prostate cancer treatment. Enzalutamide (Enz) is a second-generation antiandrogen agent which suppresses mCRPC progression by inhibiting the androgen receptor signalling pathway. However, cancer cells eventually develop resistance whose mechanism remains elusive. Previous data from our lab confirmed that extracellular matrix (ECM) proteins and their downstream signalling pathways play a critical role in the development of Enz-resistance in bone mCRPC. My work so far has demonstrated that in both in vitro and in vivo conditions, the responsiveness to Enz of mCRPC cells depends on the expression level of ECM proteins and their integrin receptors. Following flow cytometry further suggested ECM-receptor pathways may affect the Enz response of cancer cells by altering the composition of immune cells within the tumour microenvironment, which may lead to a more cancer-promoting environment. This study illustrated ECM proteins and their receptor integrins can potentially serve as novel therapeutic targets in treating late-stage prostate cancer. Their function in Enz-resistance may also be applied into other cancer models, elucidating novel mechanisms of cancer therapeutic resistance.

**P11: Construction of a click chemistry-based HBEXO-chip to isolate circulating exosomes for breast cancer diagnosis**

**Deng Kun**

The third affiliated hospital of chongqing medical university， Royal Marsden Hospital (visitor)

Accurate diagnosis of breast cancer is critical to improving patient survival, and improving quality of life. However, challenges remain for accurate diagnosis and treatment monitoring of breast cancer, and there is an urgent need to develop new non-invasive tests to increase diagnostic efficacy and improve patient experience. Exosomes are a subpopulation of small extracellular vesicles 30-150 nm in diameter that contain protein and nucleic acid molecules loaded by primary source cells through a sorting mechanism. Exosomes are considered as a potential marker for cancer liquid biopsy diagnosis. However, there are still great challenges in isolating highly-purity, tumor-specific, and bio-active exosomes from peripheral blood, which hinders the application of exosome-based liquid biopsy techniques for clinical diagnosis. Microfluidics offers an opportunity to investigate exosome-based cancer diagnostic techniques. However, existing microfluidic chips are still not effective in capturing and releasing intact, high-purity and tumor-specific exosomes. Therefore, we developed a click chemistry-based immunoaffinity microfluidic device HBEXO-Chip for the rapid isolation and specific detection of breast cancer-derived exosomes in peripheral blood. We developed a click chemistry method capable of rapid cleavage of disulfide bonds and applied this method to construct a novel HBEXO-Chip to greatly improve the exosome isolation efficiency. Moreover, the lysis agent DTT used in this chip is less disruptive to the bilayer lipid membrane structure of exosomes, allowing access to biologically active and intact exosomes. HBEXO-Chip can capture and elute exosomes within 10 min with a capture efficiency of 82.54 ± 2.128% , an elution efficiency of 92 ± 2.22% , and the specificity four-fold higher than the gold standard method (ultracentrifugation), according to the validated optimal parameters. Furthermore, we demonstrated that HBEXO-Chip could isolate tumor-specific exosomes directly from 15 clinical plasma samples and successfully differentiate between breast cancer, benign breast tumors, and healthy controls by quantification epcam positive exosomes. The area under the receiver operating characteristic curve of HBEXO-Chip is up to 0.82, superior to CA199/CA125, a standard diagnostic index for breast cancer. HBEXO-Chip provides a rapid, highly sensitive, and specific technique for peripheral blood tumor exosome detection, providing clinicians with a platform for liquid biopsy technology for breast cancer diagnosis.

**P12: A machine learning pipeline for the inference, classification and interpretation of microbial correlation networks**

**Yuanwei Xu**

Centre for Health Data Science, University of Birmingham

Microbiome communities exist in diverse habitats, exhibiting complex host-microbe and microbe-microbe interactions. Increasing evidence suggest that various human diseases may be associated with dysbiosis of the gut microbiome. Microbiome-based predictive analyses seek to understand the associations between microbiome and certain phenotypic outcome, such as an individual's health status. Despite numerous microbes have been implicated as potential biomarkers, challenges remain due to not only the statistical nature of microbiome data but also the lack of understanding of microbial interactions which can be indicative of the disease. I present a machine learning pipeline that 1) infers correlation networks of microbes based on their relative abundance profile; 2) classifies each whole network as either resulting from a diseased microbiome or from a healthy microbiome, and 3) uses a heuristic algorithm to identify a collection of microbes jointly influencing the prediction of the label of a given network.

**P13: Neurodevelopmental defects caused by loss of Cyp27a1 reveal novel developmental roles for microglia and bile acids**

**Hao HU**
Wolfson Institute for Biomedical Research, University College London

Dysfunction of cholesterol hydroxylase CYP27A1 leads to altered bile acid and lipid metabolism in Cerebrotendinous Xanthomatosis(CTX) patients, starting to cause broad and diverse nervous system deficits during development. Chenodeoxychonic acid(CDCA), the downstream metabolite bile acid of Cyp27a1, is clinically used to relieve global symptoms and stabilize nervous system deficits but the mechanism underlying the pathogenesis remains unclear. Here, using Cyp27a1 germline knockout mouse, we identified defective interneuron development namely delayed and decreased Parvalbumin expressing interneuron. We also identified developmental delay of myelin sheath in both white matter and grey matter. With the Cre-lox recombinase system, we located the potential role of Cyp27a1 through microglia, not neurons nor oligodendrocytes or astrocytes， that support interneuron development. In addition, lack of Cyp27a1 also results in anxiety, defective motor function, and impacted sociability. By oral treatment with a concentration gradient, we identified the alleviative function of CDCA above abnormal interneuron development. In conclusion, we identified the crucial functional role of Cyp27a1 in developing normal brain functions and the rescue effect of CDCA. These data together show novel roles for bile acids and microglia in brain development.

**P14: Note on the correct name of *Stauranthera grandifolia* Benth**

**Zhangjie Huang, Peiliang Liu**
Key Laboratory of Resource Biology and Biotechnology in Western China, Ministry of Education, Northwest University, 710069, Xi'an. Corresponding author: E-mail: liupl@nwu.edu.cn

Correct name is the only name that can be used universally of a species, which can reduce ambiguity in conversation. However, it will cause chaos in research and production if names are used improperly. As a hotspot plant Family, there are some problems of changes in scientific names in Gesneriaceae. Since the publication of *Stauranthera grandifolia* Benth., different forms of the epithet, *grandifolia* and *grandiflora*, were being used in various literatures. By checking related literatures, this paper confirms that the correct epithet should be *grandifolia* according to the Articles of the International Code of Nomenclature for algae, fungi, and plants. Key words: *Stauranthera grandiflora* Benth.； Gesneriaceae； epithet； International Code of Nomenclature for algae, fungi, and plants

**P15: The brain structure, immunometabolic and genetic mechanisms underlying the association between lifestyle and depression**

**Bingxin Lu**
Section of Systems Biology, School of Biosciences, University of Surrey, Guildford, GU2 7XH

Phylogenetic trees based on copy number profiles from multiple samples of a patient are helpful to understand cancer evolution. Here, we develop a new maximum likelihood method, CNETML, to infer phylogenies from such data. CNETML is the first program to jointly infer the tree topology, node ages, and mutation rates from total copy numbers of longitudinal samples. Our extensive simulations suggest CNETML performs well on copy numbers relative to ploidy and under slight violation of model assumptions. The application of CNETML to real data generates results consistent with previous discoveries and provides novel early copy number events for further investigation.

**P16: Studies of Muscle stem cell ageing and potential anti-ageing interventions.**

**Gailing Ma**

It is well established that ageing is characterised by a gradual decline in organismal fitness that leads to the loss of cellular homeostasis and physiological functions across multiple tissues and organs, which makes the organism more prone to age-related diseases. While in adult organisms, stem cells are responsible for sustaining homeostatic renewal and regenerative capacity in different tissues; and their functional decline is closely linked to ageing. Here, we aims to study the mechanisms underlying muscle stem cell (satellite cell, SC) functional decline and organism ageing, by employing primary cellular models derived from donors of different age groups. Overall, our preliminary results present a promising trend among most phenotypes of primary muscle SCs from young and aged donors. Specifically, young SCs show more robust proliferative potential than aged cells, and the aged cells present a potential pre-senescent status. Moreover, young SCs retain their stemness and present distinct regenerative capacity. By contrast, the stemness was significantly reduced in aged SCs and accordingly, aged SCs failed to differentiate into functional myofibers. In addition, the young cells featured higher levels of autophagy flux and less ROS production, while aged cells are opposite. These results give a comprehensive insight into this exciting topic, which can be developed as a promising target for anti-ageing interventions.

**P17: The function of CH25H in neuroinflammatory conditions**

**Tianhao Gao**
Division of Medicine, University College London

The brain is a cholesterol-rich organ, which contains nearly a quarter of the cholesterol content in the body. Around 70% brain cholesterol is found in myelin sheaths. Damage or loss of myelin, also known as demyelination, is associated with a range of neurodegenerative diseases, such as multiple sclerosis. In demyelinating diseases, microglia are critical for post-demyelination repair and support remyelination through a series of processes: phagocytosis of myelin debris, secretion of regenerative factors and regulation of the extracellular matrix. CH25H, a multi-transmembrane endoplasmic reticulum oxidoreductase, is the essential enzyme to catalyse cholesterol to 25-hydroxycholesterol (25-OHC). Interesting, according to the RNA sequencing database, CH25H is specifically expressed in activated microglia and macrophages in neuroinflammatory conditions, such as lipopolysaccharide (LPS; endotoxin) treatment. However, it is not clear whether CH25H plays an essential role in demyelinating diseases. We hypothesize that CH25H could be upregulated in the demyelinating mouse models and contribute to remyelination. First, Ch25h mRNA probes were successfully synthesized and high levels of Ch25h expression were found in brain tissues of LPS-treated mice. And then, histological expression results confirm that Ch25h mRNA is not expressed in astrocytes, oligodendrocytes or neurons during LPS stimulation, but is specifically expressed in microglia. The next step is to establish models (cuprizone model and LPC [lysophosphatidylcholine] model) of demyelinating disease and assess the role of Ch25h in the myelination process. The results shows that high number of Ch25h+ cells were detected at week 3 of cuprizone treatment. In addition, In the LPC-induced acute demyelination model, Ch25h mRNA level is highly and consistently increased in microglia around lesion area after injection (from 1dpi to 8dpi, days post-injection). After 8 days of LPC injection, larger demyelinated lesion were observed in the Ch25h KO group compared to the control group, and the number of newborn mature oligodendrocytes (ASPA+EdU+ cells) in Ch25h control group (204.55 ± 30.12, n=4) is 3 fold than Ch25h KO group (77.58 ± 5.10, n=3; p-value= 0.0096). These suggest that CH25H play a role in de/re-myelination.

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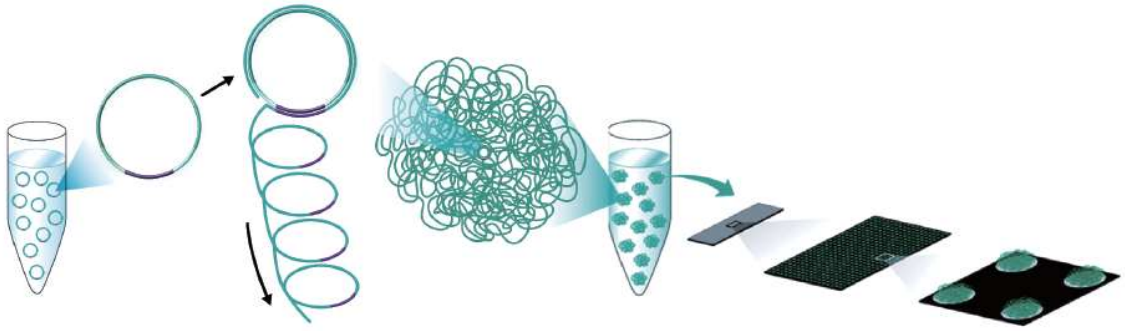

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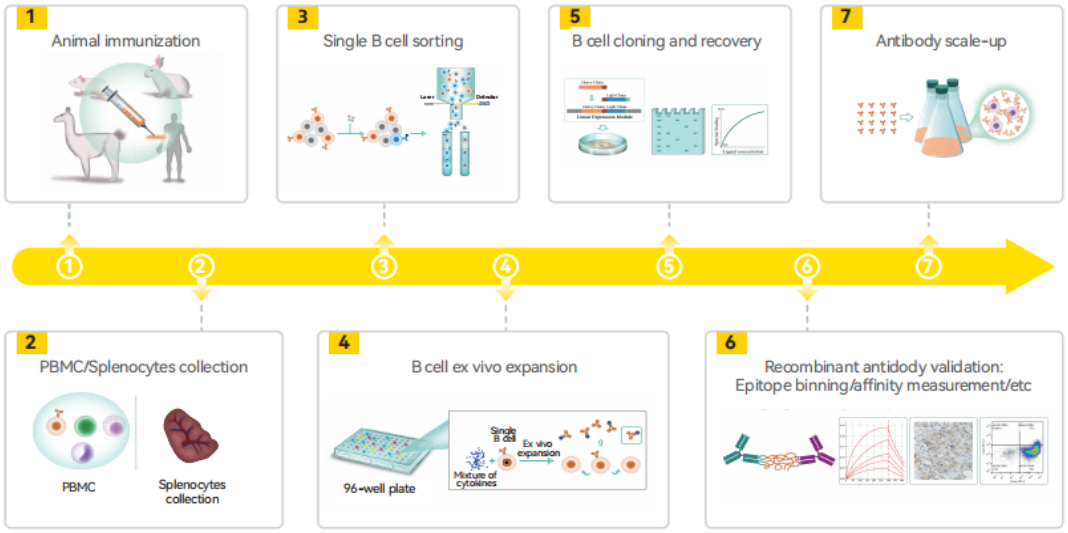

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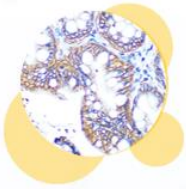

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### Consumable

**Lab consumables & Instruments**

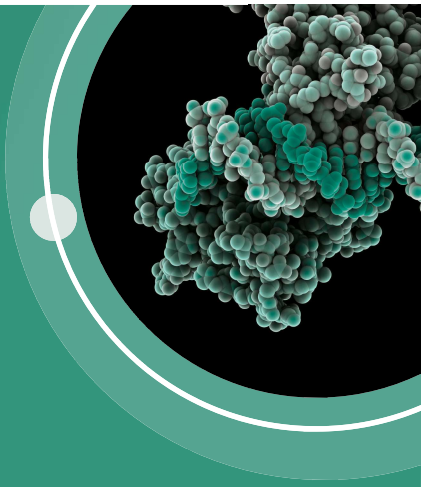
- *PCR related consumables*
- *Centrifuge tubes*
- *Pipette tips*
- *Vortex mixer*
- *Thermal cycler*
- *Tissue Cell-destroyer*

### CRO Service

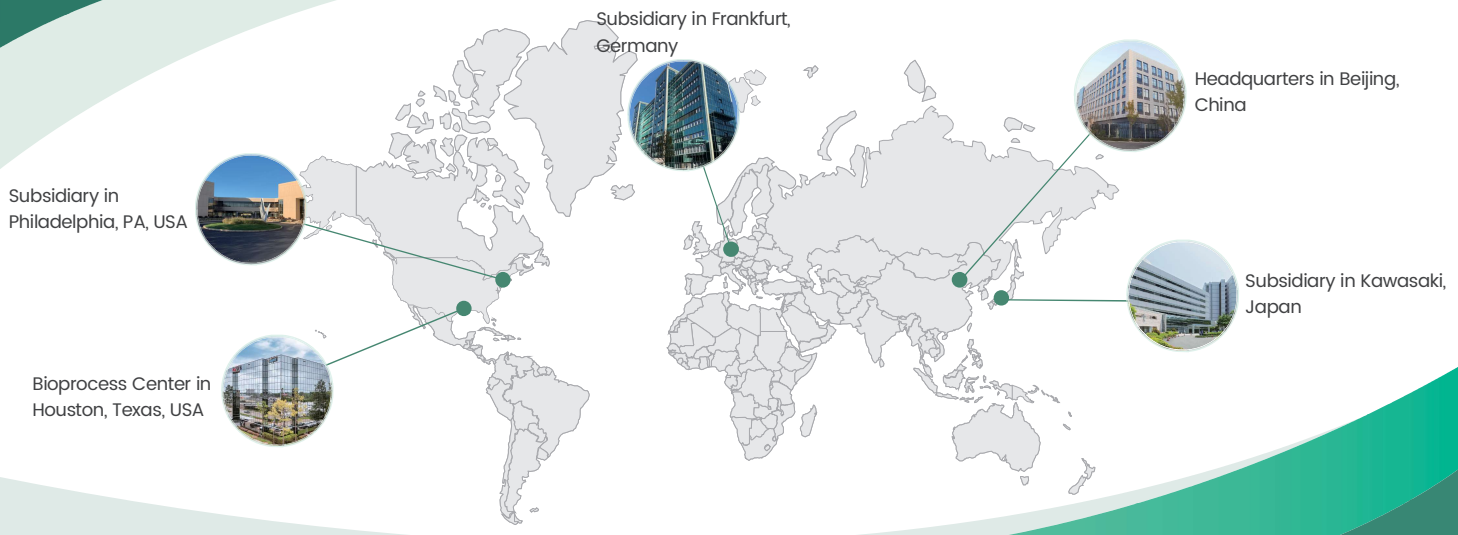

**Antibody & immunoassay development service**

- *Rabbit mAb discovery*
- *Camelid nanobody development*
- *Antibody pairs development*
- *Immunoassay development*
- *Multiplex assay development*
- *Antibody engineering*

# World's Leading Bioreagents Supplier of Recombinant Proteins and Antibodies

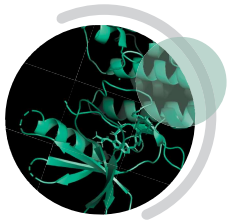


Sino Biological Europe GmbH is located in Frankfurt, Germany. With a local sales and service team as well as our comprehensive protein inventory, Sino Biological is able to deliver high-quality recombinant protein and antibody products with advanced technology platforms and services with competitive lead times to our European customers.



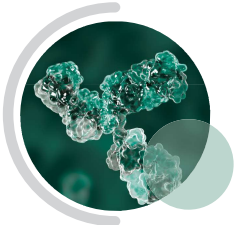

## 32000+ cDNA

## 200+ ELISA Kits



## 6500+ Recombinant proteins

- Drug Target Proteins
- Cytokines and Growth factors
- Biomarker Proteins
- CAR-T Cell Therapy Targets
- Immune Checkpoints
- CD Molecule Proteins
- Fc Receptors
- Viral Proteins
- Others: Allergens, Enzyme, Neurobiology, Stem Cell, Epigenetic Proteins



## 14,000+ Antibodies

- Primary and Secondary Antibodies
- Tagged Antibodies
- Loading Control Antibodies
- Control Antibodies
- EliteRmab
- Neutralizing Abs
- Antibody Pairs (IVD)

## Customized CRO Services

- Recombinant Protein Production
- HTP Recombinant Antibody Production
- Therapeutic Antibody Development Platforms
- Anti-idiotype Antibody
- Nanobody Production
- Antibody Humanization
- AI-powered Antibody Affinity Maturation
- VLP-based Membrane Protein Expression

# Location and Transportation

**Conference Venue**

The Cruciform Building lecture theatre 1 (B304)
University College London, London WC1E 6AE

**Dinner**

Chang's Noodle
35-37 New Oxford St
Holborn, London WC1A 1BH

**Local transportation (information taken from https://www.ucl.ac.uk/maps/public-transport/#tube )**

**Tube –** The closest tube stations to UCL's Gower Street site are Euston Square (Hammersmith and City, Metropolitan and Circle lines), Warren Street (Northern and Victoria lines), Euston (Northern and Victoria lines) and Russell Square (Piccadilly line). More information and a journey planner can be found at www.tfl.gov.uk/tube

**Bus –** UCL's Gower Street site is served by many Transport for London bus routes. Buses stop outside the Warren Street station, about five minutes' walk from The Cruciform Building. Services include route numbers: 24, 29, 73, 134, 390. More information and a journey planner can be found at www.tfl.gov.uk/buses