UK Redox Network

‘UK Redox Network’ Meeting | Wednesday 17th April, 2024

Gordon Museum in Hodgkin Building
Guy’s Campus, King’s College London
London SE1 1UL

PROGRAMME

12:00-12:50 **Arrival - Registration, Refreshments & Networking**

12:50-13:00 **Welcome & Introduction** (chair: Giovanni Mann)

13:00-13:30 **Plenary Talk** (chair: Giovanni Mann)

Juan SASTRE (University of Valencia & President of SFRR-Europe)
‘TRP14 regulates intracellular cystine reduction and protein cysteinylation’
+ ‘Update on SFRR-Europe’

13:30-14:45 **Session 1 Talks** (chair: Ivan Gout)

13:30-13:50 **Paraskevi KRITSILIGKOU** (University of Liverpool)
‘Proteome-wide tagging with genetically encoded biosensors to identify highly localised oxidation events’

13:50-14:05 **Paul MIDDLETON** (Imperial College London)
‘Mitochondrial dysfunction underlies monocyte immunoparesis in severe alcohol-related hepatitis’

14:05-14:25 **Jérôme GOUGE** (University College London)
‘New trick for an old dog, redox signalling in the RNA Poly 3 system’

14:25-14:45 **Helena COCHEMÉ** (MRC Laboratory of Medical Sciences)
‘Redox signalling in ageing’

14:45-15:30 **Posters - Refreshments & Networking**

15:30-17:05 **Session 2 Talks** (chair: Helena Cochemé)

15:30-15:50 **Ivan GOUT** (University College London)
‘Coenzyme A biology, but not as we know it’

15:50-16:10 **Hayley SHARPE** (Babraham Institute)
‘Redox regulation of phosphotyrosine signalling’

16:10-16:25 **Andrew JAMES** (MRC Mitochondrial Biology Unit, University of Cambridge)
‘Acyl-CoA and thiol-dependent acyl transfer reactions’

16:25-16:45 **Olena RUDYK** (King’s College London)
‘Dissecting the role of a novel redox-switch in Cyclin D-CDK4 in pulmonary vascular disease’

16:45-17:05 **Jörg MANSFELD** (Institute of Cancer Research)
‘Cell cycle control by reactive oxygen species’

17:05-17:15 **Concluding Remarks & Future Directions**

17:15-18:00 **Reception & Networking**

UK Redox Network

INFORMATION

Event: '**UK Redox Network' Meeting 2024**

Date: Wednesday 17th April, 2024

Time: 12 - 6 pm

Venue: Gordon Museum
Hodgkin Building, Guy's Campus
King's College London
London SE1 1UL

London Bridge station (Jubilee & Northern underground lines & network rail)

https://www.kcl.ac.uk/visit/hodgkin-building

SUMMARY

This meeting will bring together UK-based groups interested in redox biology. Following on from an inaugural event in 2018, we hope to continue strengthening ties within the UK redox community, as well as developing further links with SFRR-Europe. Join us for opportunities to present your redox research (there will be a poster session and elevated talks for ECRs), interact with leaders in the redox field, and discuss strategies to support further 'UK Redox Network' initiatives.

Format:	**in person**	Talks, poster session & networking. FREE to attend - please register on Eventbrite (select 'In Person') To present a **poster**, add this option at registration.
	online	Talks will also be streamed online. FREE to join - please register on Eventbrite (select 'Online') Participants will receive a link prior to the meeting.
Registration:		https://UKRedoxNetwork2024.eventbrite.com

PROGRAMME

Plenary:	Juan SASTRE	University of Valencia & President of SFRR-Europe
Speakers:	Helena COCHEMÉ	MRC Laboratory of Medical Sciences
	Jérôme GOUGE	University College London
	Ivan GOUT	University College London
	Paraskevi KRITSILIGKOU	University of Liverpool
	Jörg MANSFELD	Institute of Cancer Research
	Olena RUDYK	King's College London
	Hayley SHARPE	Babraham Institute
Elevated talks:	Andrew JAMES	MRC Mitochondrial Biology Unit
	Paul MIDDLETON	Imperial College London

Organisers: Giovanni MANN (King's College London) giovanni.mann@kcl.ac.uk
Ivan GOUT (University College London) i.gout@ucl.ac.uk
Helena COCHEMÉ (MRC LMS) helena.cocheme@lms.mrc.ac.uk

CHAIR & SPEAKER INFORMATION

Giovanni MANN (King's College London)
giovanni.mann@kcl.ac.uk

Chair: Welcome & Introduction, Concluding Remarks & Future Directions

Biosketch: Giovanni Mann obtained his BSc in Zoology from George Washington University, Washington DC USA and MSc and PhD in Physiology from University College London and is Professor of Vascular Physiology at King's College London. He is currently President of the Society of Free Radical Research-International (SFRRI), and served as General Secretary of SFRRI, President of the Society for Free Radical Research-Europe, Chairman of The Physiological Society, President of British Microcirculation Society, President of European Microcirculation Society, and was elected a Fellow of The Physiological Society in 2018. He is Associate Editor for Physiological Reviews, Senior Editor for Reviews and Special Issues for Free Radical Biology & Medicine and Chair of the Ethics Committee for FRBM and an Editor for Redox Biology. He previously served on Editorial Boards of The Journal of Physiology, Microcirculation and as an Editorial Advisor for the Biochemical Journal. He is a member of the Board of External Referees for the BBSRC and College of Experts for the MRC - Physiological Systems & Clinical Sciences, and previously served as Chair of Translational Sciences Panel for Heart Research UK, Medical Panel of The Henry Smith Charity and on grant panels of the BHF, Guy's & St. Thomas' Hospital Charitable Foundation and Royal Society International Networks Panel. Prof Mann has published >200 research papers and coordinated >45 research symposia at international conferences. Prof Mann's Vascular Biology research group at King's College London is investigating signalling cascades involved the transcriptional activation of antioxidant defence genes in endothelial and smooth muscle cells in oxidative stress. We are interested in vascular dysfunction induced by oxidative stress in diseases including ischemic stroke, atherosclerosis and gestational diabetes, and have demonstrated the health benefits of dietary sulforaphane as an activator of Nrf2 targeted antioxidant enzymes. More recently, his group have established the importance of Nrf2 in ischaemia-reperfusion injury in a murine model of stroke and microvascular endothelial cells adapted to 'physiological' O_2 levels. In collaboration with the London Metallomics Facility, his team obtained the first metal fingerprints in a mouse model of stroke and brain and coronary vascular cells subjected to ischemia-reoxygenation injury.

Juan SASTRE (University of Valencia, Spain)
Juan.Sastre@uv.es

13:00-13:30

Plenary: 'TRP14 regulates intracellular cystine reduction and protein cysteinylation'

Biosketch: Juan Sastre is a Professor in Physiology at the University of Valencia, Spain. His PhD Thesis was on liver aging. During more than 10 years, he did research highlighting the important role of oxidative stress in aging, physical exercise, and perinatal development at the laboratory of Prof. José Viña. From the 90s, he leads a research group on experimental Gastroenterology. The most relevant scientific findings of his research group are related to oxidative stress and redox signaling in acute pancreatitis, alcoholic cirrhosis, and biliary cirrhosis. He is currently President of European Society for Free Radical Research since January 2023. He was General Secretary of the European Society for Free Radical Research from 2013 till 2020, President of the Spanish Group for Free Radical Research from 2010 till 2015, and Head of the Department of Physiology of the University of Valencia from 2017 till 2023. Juan Sastre has published > 150 articles, with currently > 11,000 citations and an H-index of 58.

Paraskevi (Pari) KRITSILIGKOU (University of Liverpool) 13:30-13:50
pari@liverpool.ac.uk

'Proteome-wide tagging with genetically encoded biosensors to identify highly localised oxidation events'

Abstract: Genetically encoded redox biosensors have revolusionised the field of redox biology. They provided avenues to understand the dynamics of glutathione and hydrogen peroxide and identify new components involved in their regulation. By targeting these probes to cellular compartments such as the mitochondria, we were able to understand further cross-organellar communication and redox signalling. However, as redox signalling perhaps requires proximity to occur, highly localised oxidation events are unseen by the conventional, freely diffusible probes. To overcome this limitation we generated tethered biosensors, fusing genetically encoded redox probes at the C-terminus of every single open reading frame in *S. cerevisiae*. This approach allows us to monitor the redox environment around each individual protein. By exposing our biosensor libraries to different metabolic conditions, we were able to show that redox heterogeneity even within one compartment exists and that this is specific to the protein the probe is fused to.

Biosketch: Pari obtained her degree in Biology from the University of Crete, working with Kostas Tokatlidis on oxidative protein folding in the mitochondria. For her masters, she studied Biochemistry at the University of Oxford, Merton College, working with Stuart Ferguson and Christina Redfield on the disulfide bond formation system in bacteria. She then joined the Wellcome Trust-funded PhD program "The Dynamics of Cellular Pathways" at the University of Manchester, where she explored redox signalling cascades using tadpoles, cell lines and yeast as model organisms. During her thesis work with Chris Grant, Pari investigated how redox regulates organelle homeostasis, including uncovering an unexpected role for a Gpx in the mitochondrial intermembrane space. Her continued passion to uncover new redox-regulated signaling cascades led her to join Tobias Dick's lab at the German Cancer Research Center in Heidelberg, Germany. In her postdoctoral work Pari has used tethered biosensors to reveal highly localized redox microenvironments. Pari has accepted a five year Tenure Track Fellowship position at the Institute of Systems, Molecular and Integrative Biology at the University of Liverpool to continue exploring intracellular redox heterogeneity.

Jérôme GOUGE (University College London) 14:05-14:25
j.gouge@ucl.ac.uk

'New trick for an old dog, redox signalling in the RNA Poly 3 system'

Abstract: The RNA Polymerase III is in charge of transcribing all short and untranslated RNA in eukaryotic cells, e.g. the entire pool of tRNAs, U6 spliceosomal RNA and 5S rRNA. All RNA polymerases rely on a set of general transcription factors to accurately initiate transcription. Brf2 recruits Pol III at the type III gene-external promoters. Found only in vertebrates, Brf2 has been linked to tumorigenesis but the underlying mechanisms remain elusive. We have solved crystal structures of a human Brf2-TBP complex bound to natural promoters. Surprisingly, our structural and functional studies unravel a Brf2 redox-sensing module capable of specifically regulating Pol III transcriptional output in living cells. Furthermore, we establish Brf2 as a central redox-sensing transcription factor involved in the oxidative stress pathway and provide a mechanistic model for Brf2 genetic activation in lung and breast cancer.

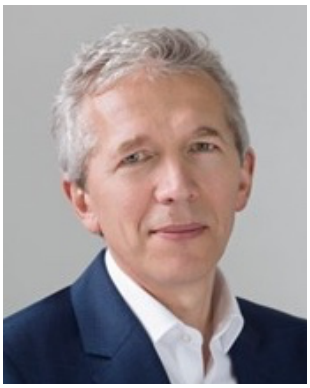
Biosketch: Jerome did his studies at the Ecole Normale Superieur in Paris where he studied at the interface between Physics, Chemistry and Biology. He did his PhD at Pasteur Institute, also in Paris, where he got interested in DNA polymerases involved in replication and DNA repair. Following on, he did his postdoc at the Institute of Cancer Research in London where he looked at the recruitment of the human RNA Polymerase III at a very specific promoter type. He uncovered an unexpected layer of regulation, linked to the oxidative stress. This is the basis of what he is looking at in his lab. Indeed, Jerome obtained a Sir Henry Dale from the Wellcome Trust to set up his lab at the ISMB in London a couple of years ago.

Helena COCHEMÉ (MRC Laboratory of Medical Sciences) 14:25-14:45
helena.cocheme@lms.mrc.ac.uk

'Redox signalling in ageing'

Abstract: Dysregulation of redox homeostasis has been implicated in the ageing process and in the pathophysiology of age-related diseases. Redox signalling operates through the specific post-translational modification of cysteine residues on target proteins. This cysteine oxidation acts as a reversible redox switch, resulting in changes to the biological function, subcellular localisation or binding partner interactions of the target. Many fundamental metabolic processes are under redox control, integrating cues from oxidative stress, nutritional status and mitochondrial function. We employ a combination of biochemical, molecular, proteomic and genetic knock-in approaches to gain mechanistic insight into the role of redox signalling *in vivo*. Here we will discuss how enhanced autophagy via redox regulation can lead to lifespan extension using *Drosophila* as a model system.

Biosketch: Dr Helena Cochemé is a UKRI Investigator at the MRC Laboratory of Medical Sciences (LMS), where she leads the Redox Metabolism group. She is also an Honorary Senior Lecturer at Imperial College London. She received her PhD in Biochemistry from the University of Cambridge in 2006, at the MRC Mitochondrial Biology Unit in the group of Prof. Mike Murphy, where she first developed her interest in mitochondrial oxidative stress and redox biology. She started working with *Drosophila* in 2007 as a post-doc at the Institute of Healthy Ageing, UCL in the laboratory of Prof. Linda Partridge. As part of her postdoctoral work, she characterised the mitochondria-targeted H_2O_2 probe, MitoB, and demonstrated that mitochondrial H_2O_2 levels increase *in vivo* with age. She established her own independent group at the LMS in 2013, and was promoted to Programme Leader Track in 2018. Research in the Cochemé lab is focused on the impact of redox signalling in ageing and metabolic disorders, mainly using *Drosophila* as an *in vivo* model system. She is particularly interested in dissecting the role of redox-responsive cysteines in regulating survival, by applying a combination of redox proteomic and genetic knock-in approaches. Ultimately, her work aims to uncover novel evolutionary conserved redox-sensitive targets and potential therapeutic strategies for health and longevity benefits



Ivan GOUT (University College London) 15:30-15:50
i.gout@ucl.ac.uk

'Coenzyme A biology, but not as we know it'

Abstract: Coenzyme A (CoA) is an essential cofactor in all living cells. CoA and its thioesters participate in diverse anabolic and catabolic pathways, allosteric interactions, biosynthesis of neurotransmitters and the regulation of gene expression. Deregulation of CoA biosynthesis in animal models and inborn mutations in human genes involved in the CoA biosynthetic pathway have been associated with human pathologies, including cancer, neurodegeneration and metabolic disorders. We have recently discovered the antioxidant function of CoA, involving covalent protein modification by this cofactor and termed it CoAlation. To discover and study protein CoAlation and the antioxidant function of CoA, we have developed several novel reagents and methodologies, including: (a) anti-CoA mAb, which specifically recognize CoA in ELISA, WB, IP and IHC; (b) a robust mass spectrometry-based methodology for the identification of CoAlated proteins; and (c) efficient *in vitro* CoAlation and deCoAlation assays. They have been employed to demonstrate that protein CoAlation is a reversible and widespread post-translational modification induced by oxidizing agents and metabolic stress in cells, tissues and model organisms. To date, we have identified more than 2100 CoAlated proteins and showed that CoAlation modulates the activity and subcellular localization of modified proteins. It can also protect oxidized cysteine residues from overoxidation and induce significant conformational changes. Based on these findings, we propose that under physiological conditions CoA functions as a key metabolic cofactor but acts as an antioxidant in cellular response to oxidative or metabolic stress. The pattern of protein CoAlation has been examined in human pathologies associated with oxidative stress, including neurodegeneration and cancer. Recent advances on defying molecular mechanisms of the CoAlation/deCoAlation cycle and investigating the antioxidant function of CoA in neurodegenerative pathologies will be presented.

Biosketch: Graduated as an MD with distinction at Lviv State Medical University (Ukraine) in 1983 with a great passion to become a surgeon in oncology. Thinking that a PhD in experimental oncology would help to realize his dream, he obtained a doctorate degree at the Institute of Experimental Oncology, National Academy of Sciences of Ukraine in 1987. A fellowship from the International Agency for Research on Cancer (IARC) took him even further from clinical oncology and also from Ukraine. He arrived in London on the first wave of perestroika and began post-doctoral studies in Mike Waterfield's laboratory at the Ludwig Institute for Cancer Research (UCL Branch), studying signal transduction via the PI3 kinase pathway. In 1996, he started his own group at the same Institute, focusing on the regulation of cell growth via the mTOR/S6K pathway in normal and cancer cells. Since 2003, he has been a Professor of Cancer Biochemistry at University College London, working on signal transduction, cellular metabolism and redox regulation in health and disease. He was the first to report molecular cloning and characterization of several signaling and metabolic proteins, including ribosomal S6 kinase 2 (S6K2), mammalian target of rapamycin (mTORb) and CoA synthase. A new field of research on protein CoAlation and antioxidant function of coenzyme A (CoA) in eukaryotes and prokaryotes has been recently pioneered in his laboratory. CoA, a key metabolic integrator in healthy cells, was shown to function as a major cellular antioxidant under oxidative or metabolic stress. The research is now focused on defining molecular mechanisms of the CoAlation/deCoAlation cycle and examining the pattern of protein CoAlation in health and pathologies. He firmly holds a world-leading position in this emerging and promising field of study.

Hayley SHARPE (Babraham Institute) 15:50-16:10
Hayley.Sharpe@babraham.ac.uk

'Redox regulation of phosphotyrosine signalling'

Abstract: Pervanadate is a widely used tool in cell biology and signalling research. It is a potent and irreversible inhibitor of the cysteine-based, classical protein tyrosine phosphatase (PTP) family. It acts via irreversible oxidation of PTP catalytic cysteines. Upon treatment of cells with pervanadate there is a rapid increase in protein tyrosine phosphorylation, previously attributed to PTP inactivation and subsequent derepression of Src family kinases. This has led to the assumption that basal PTP activity is high and extensively suppresses tyrosine phosphorylation in cells. However, several PTPs are positive regulators of growth and signalling in cells; they are not simply off switches. In addition, we, and others, have noted that inhibition of PTP function alone does not recapitulate the effects of pervanadate. Here, we find that pervanadate directly oxidises and activates Src kinase by promoting its open, active conformation. This has implications for how we interpret the data generated to date using pervanadate. Additionally, our study indicates that endogenous oxidation activates Src in the regulation of cell growth. Overall, our work shows oxidation-induced, specific conformational changes for both PTPs and Src, suggesting that oxidation is a key, reversible switch in phosphotyrosine signalling. We now aim to understand hydrogen peroxide spatiotemporal dynamics at cell membranes, where these proteins function, in homeostasis, disease and ageing.

Biosketch: Dr Hayley Sharpe is a group leader in the Signalling research programme at the Babraham Institute, Cambridge. Prior to joining the Institute in 2019, Dr Sharpe was a Principal Investigator at the Cambridge Institute for Medical Research where she established her lab after obtaining a Wellcome/Royal Society Sir Henry Dale fellowship. She undertook her postdoctoral work in the lab of Dr Fred de Sauvage at Genentech in California, USA. Dr Sharpe gained her PhD with Dr Sean Munro FRS at the MRC LMB and has a Masters in Biochemistry from the University of Bath. While at the Institute she became an EMBO Young Investigator and a Lister Prize Fellow. In 2023, she was awarded a 2023 ERC consolidator grant (now UKRI Frontier Research grant). Her lab focuses on understanding how protein tyrosine phosphatases and kinases cooperate to enable cell-to-cell communication, and how information about the extracellular environment is communicated internally to change cell behaviours such as growth, movement or attachment.

Olena RUDYK (King's College London) 16:25-16:45
olena.rudyk@kcl.ac.uk

'Dissecting the role of a novel redox-switch in Cyclin D-CDK4 in pulmonary vascular disease'

Abstract: Pulmonary hypertension (PH) is a devastating, progressive chronic vascular disease with unmet clinical needs and no cure. Identifying cancer-like growth of pulmonary vascular cells in PAH patients validated novel targets for disease-modifying therapies, with some already showing promising results in clinical trials. PH is characterised, among other abnormalities, by the hyperproliferative phenotype of all pulmonary vascular cell types, perturbed cellular redox and metabolic balance. By employing an unbiased gene chip and pathway analysis of differentially expressed genes in the lungs of mice exposed to chronic hypoxia for three days, we found an enrichment of numerous cell cycle pathways, suggesting cell cycle dysregulation occurs early in disease pathogenesis. Oxidants induce cell cycle arrest to halt proliferation; however, little is known about the redox-regulated cyclin-dependent kinases that mediate these processes. Here, we report a kinase-inhibitory disulfide bond in cyclin D-CDK4 (cyclin-dependent kinase 4) and investigate its role in cell proliferation and PH. We found that this novel redox-switch in cyclin D-CDK4 acts to inhibit kinase activity and halt cell proliferation; it forms at a critical cysteine residue, which is unique to CDK4, offering the potential for the design of a selective covalent inhibitor predicted to be beneficial in PH. We have also identified other cyclin-dependent kinases (e.g., CDK6, CDK2, CDK8) that have redox-reactive cysteines and are currently defining their functional role and cause and effect in vascular cell proliferation, with the aim of developing novel treatments for hyperproliferative vascular diseases.

Biosketch: Dr Olena Rudyk is a Principal Investigator/Group Leader and a Lecturer within the Southbank Section of the School of Cardiovascular and Metabolic Medicine & Sciences, based at Rayne Institute of St Thomas' Hospital. Olena completed her PhD in the Department of Blood Circulation at the Bogomoletz Institute of Physiology in Kyiv, Ukraine, in 2007, followed by postdoctoral training at the Division of Women's Health at King's College London. In 2015, Dr Rudyk was awarded a BHF Intermediate Basic Science Fellowship to carry on her research at the School of Cardiovascular Medicine & Sciences, during which she continued working on redox regulation of the kinases in the cardiovascular system and further developed an interest in experimental PH. In 2020, Dr Rudyk was employed as a Lecturer by the Faculty of Life Sciences & Medicine. Since then, she has been working on understanding the molecular mechanisms contributing to PH. Currently, Rudyk Lab is working to identify novel redox sensors and signalling pathways that can be targeted to reverse pulmonary vascular remodelling and test potential new PH therapies. They do so by employing integrative *in vitro*, *ex vivo*, and *in vivo* approaches, novel imaging techniques, and emerging collaboration with clinical scientists. Dr Olena Rudyk has been a Fellow of the Higher Education Academy (since 2018), a member of the Pulmonary Vascular Research Institute (since 2017), and a recipient of a Butrous Foundation Young Investigator Award (2018) for her work in PH.

Jörg MANSFELD (Institute of Cancer Research) 16:45-17:05
jorg.mansfeld@icr.ac.uk

'Cell cycle control by reactive oxygen species'

Abstract: Reactive Oxygen Species (ROS) are small oxygen-containing molecules than can be produced inside cells. By oxidizing proteins, ROS regulate protein activities, stability, localisations and thereby, control different cellular processes including proliferation. ROS have been shown to promote cell cycle entry by influencing mitogen signalling, and a gradual increase of ROS is essential for progression through the cell cycle, whereas ROS removal halts proliferation. Supporting direct links between redox and cell cycle regulation, ROS recently have been shown to regulate the activity of cell cycle kinases CDK2 and Aurora A. Whether ROS can impact other core cell cycle proteins and act as a fundamental cell cycle regulatory mechanism, however, remains elusive. To address these questions, we have performed cell cycle stage dependent redox proteomics identifying the cyclin dependent kinase inhibitor p21 (CDKN1A) as a redox-regulated core component of the cell cycle machinery. I will discuss the mechanistic consequences of p21 oxidation and how its redox state during the G2 phase defines the fate of cells.

Biosketch: Jörg Mansfeld studied biology at the University of Konstanz in Germany, followed by a PhD in biochemistry at the ETH Zurich, Switzerland, and a PostDoc in the laboratory of Jon Pines at the Wellcome Trust Gurdon Institute in Cambridge, UK. In 2013, he started his own laboratory at the Biotec Institute of the Technical University Dresden, Germany, investigating how the ubiquitin system controls the decision of cells to proliferate of not to proliferate. Since it became clear that ubiquitin enzymes and other components of the core cell cycle machinery can be targeted by reactive oxygen species his recent work aims to define the interplay between the cell cycle and redox systems, in particular how oxidation of cell cycle regulators affects cell proliferation and cell fate. He moved to the Institute of Cancer Research London (ICR) in 2020 and leads the Post-translational Modifications and Cell Proliferation team within the Cancer Biology Division.

ATTENDEES (in person)

Name	Organisation
Aldaz Casanova, Silvia	Babraham Institute
Angelova, Plamena	University College London
Arjun, Sapna	Queen Mary University of London
Buffonge, Stanley	Queen Mary University of London
Burgoyne, Joseph	King's College London
Cabiran, Helene	Institute of Cancer Research
Cochemé, Helena	MRC Laboratory of Medical Sciences & Imperial College London
Coleman, Pierre	King's College London
Costello, Joe	University of Exeter
Coupe, David	Institute of Cancer Research
Dutkiewicz, Roksana	Babraham Institute
Fraser, Paul	King's College London
Gouge, Jérôme	University College London
Gout, Ivan	University College London
Green, Hannah	King's College London
Holbrook, Lisa	University of Surrey
Ige, Esther Olufikayomi	Institute of Cancer Research
James, Andrew	MRC Mitochondrial Biology Unit, University of Cambridge
Lai, Tiffany	Babraham Institute
Leung, Jacky	Institute of Cancer Research
Lushchak, Oleh	MRC Laboratory of Medical Sciences
Malanchuk, Oksana	University College London
Mann, Giovanni	King's College London
Manna, Suman	University College London
Mansfeld, Jörg	Institute of Cancer Research
Marriott, Eloise	Queen Mary University of London
Middleton, Paul	Imperial College London
Mulholland, Katie	Babraham Institute
Naftalin, Richard	King's College London
Ng, Ezra	University College London
Rahman, Oishee	Babraham Institute
Rudyk, Olena	King's College London
Santos, Chloe	University College London
Sharpe, Hayley	Babraham Institute
Siow, Richard	King's College London
So, Po-Wah	King's College London
Su, Vivian	King's College London
Tolley, Isaac	University College London
Torley, Elizabeth	Institute of Cancer Research
Tossounian, Maria	MRC Laboratory of Medical Sciences
Tuncay, Ahmet	Babraham Institute
Veal, Elizabeth	Newcastle University
Vorhauser, Julia	Institute of Cancer Research
Yap, Vincent	MRC Laboratory of Medical Sciences
Yang, Fan	King's College London
Zhang, Dejun	University College London
Zhu, Aurelia	University College London

ATTENDEES (online)

Name	Organisation
Agrawal, Neha	University of Edinburgh
Alam, Sara	MRC Laboratory of Medical Sciences
Banerjee, Samrajni	University of Liverpool
Bellosta, Paola	University of Trento, Italy
Casas, Jose	University of Galway, Ireland
de Jesus, Daniel	Queen Mary University of London
Dwyer, Emilia	Newcastle University
Fernandez Mosquera, Lorena	Queen Mary University of London
Foy, Tom	Newcastle University
Goyal, Manisha	Institute for Stem Cell Science and Regenerative Medicine, India
Gupta, Simran	Indian Institute of Science Education and Research, India
Kritsiligkou, Paraskevi	University of Liverpool
Leks, Wiktoria	Plurigrid
Lennicke, Claudia	MRC Laboratory of Medical Sciences
Li, Penglin	University of Galway, Ireland
Lightfoot, Adam	Manchester Metropolitan University
McDonagh, Brian	University of Galway, Ireland
Murdoch, Colin	University of Dundee
Ortega-Prieto, Paula	MRC Laboratory of Medical Sciences
Patel, Parthive	University of Bristol
Pillay, Che	University of KwaZulu-Natal, South Africa
Povea-Cabello, Suleva	Veneto Institute of Molecular Medicine, Padova, Italy
Seternes, Ole-Morten	UiT The Arctic University of Norway, Norway
Singh, Savarna	University of KwaZulu-Natal, South Africa
Stinson, Jennifer	Bioscientifica
Xia, Qin	University of Galway, Ireland
Xirouchaki, Chrysovalantou	Monash University, Australia

POSTERS

Green, Hannah	King's College London *'Does oxidation of Protein Kinase A Regulatory Subunit PKARIalpha play a role in pulmonary hypertension?'*
Middleton, Paul	Imperial College London *'Mitochondrial Dysfunction underlies Monocyte Immunoparesis in Severe Alcohol-Related Hepatitis'*
Rahman, Oishee	Babraham Institute & University of Cambridge *'Studying the dynamics of Hydrogen Peroxide using the HyPer7 biosensor'*