QUEEN'S UNIVERSITY BELFAST

WELLCOME-WOLFSON INSTITUTE FOR EXPERIMENTAL MEDICINE

Virtual Postdoctoral Research Symposium 2021

slack

28th-29th January 2021

Microsoft Teams

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Symposium Timetable 28th Jan

13:00 – 13:15	**Welcome and Introduction** **Prof Jose Bengoechea**, Centre Director, Wellcome-Wolfson Institute for Experimental Medicine **Dr Claire Tonry**, Co-organiser	
13:15 – 14:15	**Keynote Speaker: Dr Annie Curtis, RCSI** *""The Time of our Lives, Learnings from Body Clocks, Immunity and our own Adventures"* (Chair: Dr Claire Tonry)	
Session 1: Selected Oral Presentations	Chair: Dr Amy Dumigan	
14:15 – 14:30	Dr Ryan Brown	Genetic deletion of SLPI promotes inflammatory cell recruitment in a model of chronic lung disease
14:30 – 14:45	Dr Keren Turton	Modelling *Achromobacter* infection of macrophages
14:45 – 15:00	Dr Lauren Kerrigan	Using high-fat diet/high-dose streptozotocin hybrid in vivo model for the study of epigenetic changes in diabetic cardiomyopathy
15:00 – 15:45	**Poster Presentations Session 1 (Slack)**	
15:45 – 16:00	**Leica mini-talk**	
16:00 – 17:00	**Keynote Speaker: Prof Tony Wyss-Coray, Stanford University CA** *"Young Blood for Old Brains"* (Chair: Dr Anna-Claire Devlin)	

Symposium Timetable 29th Jan

09:00 – 10:00	**Keynote Speaker: Dr Luke Garratt, Telethon Kids Institute, Au** (Chair: Dr Keren Turton)	
10:00 – 10:15	**ThermoFischer mini-talk**	
Session 2: Selected Oral Presentations	Chair: Dr Aditi Singh	
10:15 – 10:30	Dr Alerie Guzman de la Fuente	Oligodendrocyte Progenitor Cells: New Players in CNS inflammation
10:30 – 10:45	Dr Mohammed Inayatullah	Advancing cancer discovery through single-cell genomics and artificial intelligence
10:45 – 11:00	Dr Lindsay Broadbent	Differential infectivity of SARS-CoV-2 in primary airway epithelium derived from different donors
11:00 – 11:15	**Coffee break**	
Session 3: Selected Oral Presentations	Chair: Dr Ryan Brown	
11:15 – 11:30	Dr Flávia Viana	Dissecting the molecular pathogenesis of *Legionella* spp. in human lung models
11:30 – 11:45	Dr Deborah Lavin	MNT is a critical regulator of cell-fate changes during EMT
11:45 – 12:00	Dr Michael C. McKelvey	The role of the extracellular immunoproteasome in the acute respiratory distress syndrome
15:00 – 15:45	**Poster Presentations Session 2 (Slack)**	
13:00	**Update from Postdoctoral Development Centre** **Dr Alice Dubois**, Head of the Postdoctoral Development Centre, Faculty of Medicine, Health and Life Sciences	
13:15	**Closing Remarks and Awards** **Prof Vijay Tiwari**, Co-Chair of WWIEM Postdoctoral Career Development Committee	

Poster Speed Dating and Imaging Competition

Poster Speed Dating

Postdocs are encouraged to **visit all Slack poster channels** over the course of the symposium. However, we will also be running a virtual postdoc speed dating activity.

All postdocs will be provided with **10 poster numbers**. These numbers will be assigned to postdocs via Slack (***check your Slack channel prior to symposium***) Postdocs are encouraged to visit their assigned poster channels on Slack and leave a comment or any sort of feedback using the chat function

Postdocs who have engaged with **all 8** of their assigned poster presenters will be entered into a prize draw. Winners will be announced on Workplace on **Monday 1st Feb 2021**.

Imaging Competition

During the Poster Sessions images will be displayed on the screens in the inner atrium. Please vote for your favourite image using one of the voting slips beside the ballot box below the display screen.

Symposium Information

Thank you

We would like to express our gratitude to the WWIEM Clerical Support Team for all their help and guidance in organising this Symposium and liaising with sponsors.

Thank you to the PIs and postdocs who have volunteered their time to act as the abstract selection panel, poster judges and chairs.

We would also like to thank the members of the WWIEM Postdoctoral Development Committee for their support.

Poster Judges

Dr Reinhold Medina, Dr Yvonne Dombrowski , Dr Rebecca Coll

Oral Session Judges

Dr David Courtney, Dr Eric Campbell, Dr Derek Brazil

Abstract Selection Panel

Dr Amy Dumigan, Dr Claire Tonry, Dr Keren Turton
Dr Rebecca Coll, Dr Vijay Tiwari

Keynote and Oral Presentation Session Chairs

Dr Claire Tonry, Dr Amy Dumigan, Dr Anna Claire Devlin, Dr Keren Turton, Dr Aditi Singh, Dr Ryan Brown

Symposium Organising Committee

Dr Claire Tonry, Dr Keren Turton, Dr Aditi Singh, Dr Amy Dumigan
Dr Rebecca Coll, Dr Vijay Tiwari

Keynote Speakers

Dr. Annie Curtis

Royal College of Surgeons Ireland (RCSI), Dublin

Dr Annie Curtis and her talented team of PhD students and postdocs investigate the influence of our **natural body clock and circadian rhythms on immunity**. Circadian rhythms are 24-hour rhythms in behaviours and physiologies allowing us to align to the predictable daily cycle of day and night. The body clock is the molecular timing system within our cells that drives these circadian rhythms. However, modern life, with exposure to light at night, erratic work, activity and eating patterns is disrupting our circadian rhythms and epidemiology data indicates that body clock disruption is a significant contributing factor to the rise of chronic inflammatory disease such as arthritis and cardiovascular disease.

Blending the fields of **immunology** and **circadian biology** together, her teams work may have far reaching impact on our understanding and treatment of **chronic inflammatory diseases**. There are also implications for when medications – including vaccinations – should be dispensed, aligning treatment with the daily changes in our immune system. The CurtisClockLab is funded by Science Foundation Ireland, Irish Research Council and Annie is a L'Oreal Women in Science Fellow.

In this talk, Annie will also discuss **her own career path and experiences**. This includes moving in and out of academics, motherhood, gender equality and some learnings which she has embraced along the way.

Keynote Speakers

Prof. Tony Wyss-Coray

Stanford University, CA

Prof. Wyss-Coray initially studied at the Institute of Clinical Immunology at the University of Bern in Switzerland, but has been at Stanford University since 2002. The Wyss-Coray research team studies **brain aging and neurodegeneration** with a focus on **age-related cognitive decline and Alzheimer's disease**. The lab is following up on earlier discoveries which showed circulatory blood factors can modulate brain structure and function and factors from young organisms can rejuvenate old brains. Current studies focus on the molecular basis of this systemic communication with the brain by employing a combination of **genetic, cell biology, and proteomics approaches** in model organisms and humans.

Prof. Wyss-Coray serves on the scientific advisory board for the Alzheimer Research Consortium and on the international advisory board for Advances in Clinical and Experimental Medicine. In 2013, he was given a Transformative Research Award by the director of the National Institutes of Health

In this talk, entitled **"Young Blood for Old Brains",** Prof. Wyss-Coray will describe the rejuvenating effects of young blood and the 'Parabiosis' model, as well as discussing his own career journey to date. Prof. Wyss-Coray has previously spoken about the impact of his research at TEDGlobal (London 2015), which has had over 1 million views on YouTube!

Keynote Speakers

Dr. Luke Garratt

Telethon Kids Institute, Perth

Dr. Garratt is a cell biologist and biomedical scientist, who completed his PhD at the University of Western Australia. As an **NHMRC Early Career Fellow**, Dr. Garratt is currently leading an international collaborative study with **cystic fibrosis (CF)** researchers at Emory University, USA that closely investigates the behaviour of **the immune system in the lungs of babies** with CF. Dr. Garratt's research is focused on understanding why the **neutrophil**, a key immune cell for controlling infections, so quickly becomes more harmful than helpful to the CF lung.

On his research, Dr. Garratt has said that, "*If we can better control neutrophils, we can reduce the amount of progressive lung damage. I am monitoring how lung conditions vary between babies with CF in the initial stages of disease and how neutrophils respond to these conditions. More importantly, we hope to establish this approach as a way to test treatments targeting neutrophils in early CF*".

Dr. Garratt has been a member of the Australian Respiratory Early Surveillance Team for Cystic Fibrosis (**AREST CF**) team since 2007. The AREST CF team run a unique program – The Early Surveillance Program - focused on the assessment, treatment and prevention of CF lung disease in young children

In his talk at this year's symposium Dr. Garratt will describe his research into CF in children and his work with the AREST CF consortium, as well as providing us with an insight into his career to date.

Genetic deletion of SLPI promotes inflammatory cell recruitment in a model of chronic lung disease

Ryan Brown, Peter Ferris, Rebecca Delaney, Caoifa Dougan, Marcus A. Mall[2,3,4], Sinéad Weldon, Clifford C. Taggart

[1]Airway Innate Immunity Research Group, Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast, Northern Ireland;
[2]Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast, Northern Ireland;
[3]Department of Pediatric Pulmonology and Immunology, Charité – Universitätsmedizin Berlin, Berlin, Germany;
[4]Berlin Institute of Health (BIH), Berlin, Germany

Secretory leukocyte proteinase inhibitor (SLPI) is a major airways antiprotease. Its primary function is considered to be the inhibition of damaging neutrophil elastase activity. The presence of a protease/antiprotease imbalance is a key feature of many chronic lung diseases. Therefore, we aim to elucidate the role of SLPI in the development of airway pathogenesis in chronic lung disease. In this study β-epithelial Na+ channel-overexpressing transgenic (ENaC) mice, a model of chronic lung disease, were crossed with SLPI null (SLPI-/-) mice to generate four genotypes WT, SLPI-/-, ENaC and ENaC/SLPI-/-. Juvenile (2-3 wks) and adult (8-14 wks) mice were assessed for inflammatory markers, lung damage and mucus plugging. Levels of SLPI were elevated in the ENaC mice. Genetic deletion of SLPI resulted in increased protease activity and immune cell infiltration in both WT and ENaC mice. However, there was no effect of SLPI knockout on lung damage, airway mucus plugging or lung function in these mice. These data suggest that SLPI may ameliorate the recruitment of inflammatory cells in chronic lung disease, however, it does not limit the development of lung damage or airway mucus plugging in this model.

Modelling *Achromobacter* infection of macrophages

Keren Turton, Hannah Parks*, Miguel Valvano

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast
*School of Pharmacy, Queen's University Belfast

Six feet. That's the recommended distance to keep apart for people with cystic fibrosis (PWCF), who frequently suffer from contagious and chronic respiratory infections. Opportunistic bacteria are able to thrive in the immunocompromised lung of PWCF, and many of these bacterial pathogens are poorly-characterised. Herein, we detail preliminary work in deciphering the interaction between *Achromobacter* species and macrophages, first-line defenders of the immune system. *Achromobacter* is a gram-negative bacterium, ubiquitous in soil but also found in nosocomial environments. The prevalence of this genus in respiratory infection in immunocompromised individuals has sharply increased in the last decade. Many *Achromobacter* spp. are multidrug-resistant, making infections challenging to treat. The two workhorse strains in our experimental work are AC047 (*A. insuavis*) and QV306 (*A. xylosoxidans*), respiratory clinical isolates from PWCF. We have shown that QV306 and other strains can survive intracellularly in THP1 macrophages for several hours following phagocytosis, and that the bacteria accumulate in a late endosomal compartment. AC047 and other strains can induce pyroptosis (a pro-inflammatory lytic cell death) in THP1 and primary human macrophages. By developing a markerless gene deletion system we have created mutant strains to demonstrate that the Type 3 Secretion System is necessary for inducing pyroptosis. Currently, we are deciphering which macrophage inflammasome components are required for mediating this interaction, and are trying to pinpoint the T3SS effectors responsible for triggering pyroptosis.

Using high-fat diet/high-dose streptozotocin hybrid in vivo model for the study of epigenetic changes in diabetic cardiomyopathy

Lauren Kerrigan, Kevin Edgar, Adam Russell-Hallinan, David Grieve, Chris Watson

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast

Heart failure (HF) is growing at alarming rate, largely due to increased prevalence of type-2 diabetes mellitus (T2DM). Diabetic cardiomyopathy (DCM) is defined as the presence of diabetic-associated diastolic dysfunction in the absence of typical cardiovascular risk factors. An effective strategy for prevention and treatment of DCM has yet to be established. Clear pathological mechanisms of DCM must be identified to determine therapeutic advancements.

Epigenetic regulation has been reported to have a role in development of structural alterations in HF. DNA methylation, the most common modification, occurs when a methyl group is added to a cytosine within a CpG site, commonly resulting in gene repression. Studies show that pro-fibrotic changes in cardiac cells were associated with global DNA hypermethylation, and inhibition of DNA methylation resulted in anti-fibrotic activity. The role of DNA methylation in the development of DCM has not been examined.

Our first study objective was to characterise an improved experimental model of T2DM with reliable development of DCM characteristics; our second objective was to then examine DNA methylation changes in cardiac tissue from this model.

Results of this study indicate that a single, high dose of streptozotocin in combination with high fat diet over a period of seven months is adequate to induce T2DM, providing evidence of cardiac fibrosis and diastolic dysfunction. Data also indicates that there are global DNA methylation changes in the cardiac tissue correlating with induction of cardiac fibrosis and hypertrophy, and that inhibition of DNA methylation via administration of 5azacytidine attenuates the development of cardiac structural and functional modifications.

Oligodendrocyte Progenitor Cells: New Players in CNS inflammation

Alerie Guzman de la Fuente, Andrew Young, Katie Mayne, Robin JM Franklin* and Denise C Fitzgerald

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast
*University of Cambridge

During CNS remyelination, adult oligodendrocyte progenitor cells (OPCs) are recruited to the demyelinated area and differentiate into new myelinating oligodendrocytes that ensheath axons. Like all regenerative processes in mammals, remyelination efficiency declines with ageing leading to neuronal loss and accrual of permanent disability.

Recent publications have highlighted the wide heterogeneity of oligodendrocyte lineage cells in demyelinating models as well as MS patient samples, including a previously unrecognised immune-like oligodendrocyte lineage cluster. Here we show that neonatal OPCs can induce the expression of inflammatory cytokines, such as IFN-γ, by activated CD4+ T cells without affecting expression of the anti-inflammatory cytokine, IL-10. We also show that this immune modulation is contact-dependent, as neonatal OPC-conditioned media do not influence IFN-γ expression in activated CD4+ T cells. Decreased IFN-γ expression is independent of MHCII expression by OPCs, suggesting a novel OPC-CD4 T cell contact-dependent mechanism. We also show that this OPC-mediated immune-modulatory process is impaired with ageing, even though aged OPCs have high levels of expression of proteins associated with immune processes.

These results reveal an OPC-mediated immune modulatory mechanism contributing to the regulation of T cell-mediated inflammation. The age-associated impairment of OPC immune-modulation may contribute to OPC insufficiency and remyelination failure leading to MS disease progression. Exploring this mechanism will provide new insights into MS pathogenesis as well as new therapeutic targets.

Advancing cancer discovery through single-cell genomics and artificial intelligence

Mohammed Inayatullah, Vijay Tiwari

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast

Breast cancer (BC) is the leading cause of cancer-related death globally and the cell populations underlying disease aetiology are poorly understood, hence hampering diagnostics and therapeutics strategies in cancer management.
Single-cell RNA-sequencing (scRNA-seq) emerged as a novel technique in uncovering tumor heterogeneity in variety of cancers including BC. However, such techniques could only provide detailed clustering signatures, and hence identification of subpopulations involved in cancer pathogenesis remains difficult. In recent years, many studies have used artificial intelligence to overcome such challenges, but most of them lacks in building predictive models for identifying subpopulations involved in tumor metastasis in BC. Here we used BC scRNA-seq data and artificial intelligence (AI) method and identified subpopulations showing migratory characteristics such as Epithelial–mesenchymal transition (EMT) in BC. We achieved high performance of the AI model with accuracy of more than 96%. The model has helped in discovery of new biomarkers that drives progression of the disease. Further validation of the identified markers on 3300 breast cancer transcriptomes, strongly suggests its clinical potential in BC.
In conclusion, our study have demonstrated the potential of single-cell RNA-seq and AI methods for detecting cell populations involved in tumor progression in BC. Additionally, our approach has identified new biomarkers associated with tumor metastasis in BC, hence to be further validated for their clinical utility in BC. Our model will help in the discovery of new cell populations and novel biomarkers for early diagnostics, precise therapeutics and prognosis of BC as well as other cancers.

Differential infectivity of SARS-CoV-2 in primary airway epithelium derived from different donors

Lindsay Broadbent, Connor Bamford, David Courtney, Ahlam Ali, Olivier Touzelet, Grace Roberts, Ken Mills*, Ultan Power

Wellcome-Wolfson Institute of Experimental Medicine, Queen's University Belfast
*PGJCCR, Queen's University Belfast

SARS-CoV-2, the virus that causes COVID-19, was identified in late 2019 and went on to cause over 1.5 million deaths in a year. The spectrum of COVID-19 disease ranges from asymptomatic or sub-clinical to severe long term damage to multiple organ systems or death. The reasons for varying responses to SARS-CoV-2 infection are yet to be elucidated.
Using our well-differentiated primary airway epithelial cell (WD-PAEC) model, which authentically replicates the morphology and physiology of the human airway epithelium, we investigated the host response to two different isolates of SARS-CoV-2. Interestingly, we discovered that susceptibility to SARS-CoV-2 infection, but not RSV infection, varied in WD-PAECs from different donors. This phenomenon was observed in nasal epithelial cells (WD-PNECs) from healthy young adults and bronchial cells (WD-PBECs) from >60 year olds. ACE2 and TMPRSS2, needed for attachment and entry of SARS-CoV-2, respectively, were expressed in all WD-PAECs. Western blotting showed that only WD-PBECs susceptible to infection expressed a second TMPRSS2 band, which is suggested to be indicative of efficient TMPRSS2 protease activity. However, this was not observed in WD-PNECs. The host factor limiting SARS-CoV-2 infection/replication remains to be determined. Proteomic and transcriptomic data are currently being generated.
These results indicate that susceptibility of airway epithelium to SARS-CoV-2 infection varies between donors. Correlation of such restriction to disease outcomes remains to be elucidated.

Dissecting the molecular pathogenesis of *Legionella* spp. in human lung models

Flávia Viana, Oisín Cappa, John Stegmayr*, David Simpson, Darcy Wagner*, Cecilia O'Kane, Gunnar N. Schroeder

Wellcome-Wolfson Institute for Experimental Medicine, SMDBS, QUB, Belfast,
*Lung Bioengineering and Regeneration, Department of Experimental Medical Sciences, Lund University, Lund, Sweden

A decrease in the angiopoietin-1 (Ang1) to angiopoietin-2 ratio, as well as abnormal blood flow is well documented in patients with diabetic retinopathy. We used a gene therapeutic approach to improve retinal Ang1 signalling in STZ-diabetic mice and studied the effect on capillary blood flow speed. Using a novel, fluorescent microbead-based approach, we measured blood flow speed in the capillary beds of anaesthetized non-diabetic and diabetic mice, injected intravitreally with vehicle control (PBS), control vector (AAV.CMV-GFP) or a vector carrying a gene for an engineered, stabilized version of Ang1 (AAV.CMV-COMPAng1). Speed measurements were accompanied by fundus imaging, fluorescence angiography and ex vivo immunohistochemistry to assess retinal status and spatial gene expression profiles. Flow maps showed changes in capillary blood flow decreasing with retinal eccentricity. We noted local decreases in blood flow speed on the nasal side of the retina at 10 weeks after diabetes induction. While intravitreal injection of the control vector caused an exacerbation of the effects of diabetes, AAV.CMV-COMPAng1 returned blood flow speed to normal levels. Our preclinical findings suggest that focal functional changes of the capillary bed occur in the STZ mouse model and that the Ang1/Ang2-signaling pathway may be a viable alternative or adjunctive therapeutic target to the current anti-VEGF regimes effective in supporting capillary functionality.

MNT is a critical regulator of cell-fate changes during EMT

Deborah Lavin, Leila Abassi, Mohammed Inayatalluh, Vijay K. Tiwari

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast

The multi-step process of epithelial to mesenchymal transition (EMT), whereby static epithelial cells become migratory mesenchymal cells, is heavily involved in development, wound healing, and disease states. Despite the major involvement of basic helix-loop-helix (bHLH) transcription factors (TFs) in cell-fate determination, few have examined them for their involvement in fundamental processes that require EMT. Here, we have identified Max network transcription repressor (MNT) as a potent EMT promoting TF in mammary epithelium. We show that depletion of MNT blocked TGFβ-induced phenotypic changes, and transcriptomic analysis revealed that MNT is a transcription repressor of epithelial identity. We show that MNT mediates repression of epithelial identity via direct interaction with HDAC1. Lastly, we show that MNT and its target genes are heavily expressed in EMT-High breast cancer, with MNT required for breast cancer cell migration. Taken together, these findings establish MNT as a critical regulator of cell-fate determination and the EMT transcriptome.

The role of the extracellular immunoproteasome in the acute respiratory distress syndrome

Michael C. McKelvey, Sinéad Weldon, Cecilia M. O'Kane, Daniel F. McAuley, Clifford C. Taggart

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast

The acute respiratory distress syndrome (ARDS) is characterised by the flooding of the alveoli with protein- and leukocyte-rich oedema, exuberant pulmonary and systemic inflammation, and the development of refractory hypoxaemia. ARDS mortality is between 30-50% and no specific pharmacological treatments exist that can meaningfully affect outcomes. The constitutive proteasome (CP) performs important intracellular proteolytic functions including the degradation of ubiquitin-tagged proteins. However, CP and its derivative, the immunoproteasome (IP), are increasingly recognised as being present in the extracellular space during pathology.

In preliminary work, we show that the levels of CP, and especially IP, are elevated in bronchoalveolar lavage fluid (BALF) from patients with ARDS. Furthermore, levels and activity of LMP2 and LMP7, two of the catalytic IP subunits, are upregulated in BALF from mice receiving intratracheal lipopolysaccharide, indicating that IP induction is also present in this model. Future work in this model and other murine models of ARDS will assess the effect of a specific LMP7 inhibitor, ONX-0914 (Kezar Life Sciences) on pulmonary injury and inflammation. Early in vivo experiments indicate that the application of IP to A549 cells induces the release of proinflammatory cytokines, though further experiments are required to elucidate possible mechanisms by which IP causes inflammation is these and other cells.

In conclusion, early results indicate that extracellular IP is a feature in human ARDS and murine ARDS-like disease, though further work is required to establish whether it plays a pathogenic role in ARDS.

Poster Presentations

Number	Presenting Author	Title
1	Matthew G. Pilgrim	Characterization of mineral particles in the sub-retinal pigment epithelial space in aged human eyes
2	Claire Tonry	In-Depth Proteomic Characterisation of Different Aetiologies of Cardiomyopathy
3	María Llorián-Salvador	Complement C5a induced Epithelium to Mesenchymal Transition (EMT) in retinal pigment epithelial cells
4	Claudia Feriotti	The interplay between Klebsiella pneumonia and SARM in the modulation of host immune response
5	Hojjat Naderi-Meshkin	Human vascular blood vessel organoids derived from iPSCs is a powerful model to study diabetic vascular dysfunction
6	Hong Guo-Parke	Bronchial epithelial cells from severe COPD patients have dysregulated differentiation, senescence and secretory phenotype in air-liquid interface cultures
7	Karis Little	Patients with type-2 diabetes (T2D) have an increased risk of developing cognitive impairment and Alzheimer's disease (AD)
8	Sophia Kelaini	Generation of bovine induced pluripotent stem cells and differentiation to macrophages for the study of tuberculosis caused by Mycobacterium Bovis
9	Peter Barabas	COMP-Ang1 gene therapy in mouse models of diabetic retinopathy
10	Gisli Einarsson	Effect of treatment with the TOBI Podhaler® on the airway microbiota in Pseudomonas aeruginosa-infected bronchiectasis patients: iBEST study

Full abstracts will be available on Slack

Poster Presentations

Number	Presenting Author	Title
11	Luke Johnston	Role of IL-22 signalling in colonic epithelial cells under hypoxic conditions
12	Josy Augustine	2-HDP as a novel therapeutic for diabetic retinopathy
13	Ravi Kiran Deevi	Elevated cardiac Leucine rich alpha-2-glycoprotein 1 (LRG1) in patients with heart failure
14	Connor Bamford	Genotypic and phenotypic characterisation of recent SARS-CoV-2 isolates helps define the genomic features required for efficient replication in the human respiratory epithelium
15	Aditi Singh	Deciphering the gene regulatory networks underlying cortical folding
16	Guanbo Wang	A glycoengineered antigen exploiting a conserved protein O-glycosylation pathway in the *Burkholderia* genus for diagnosis of glanders infections
17	David Courtney	Drug repurposing for SARS-CoV-2
18	Karla O'Neill	REDUCED PRO-ANGIOGENIC FUNCTION OF CORD BLOOD ENDOTHELIAL COLONY-FORMING CELLS IN HYPERGLYCAEMIA IS MEDIATED BY NOX4
19	Amy Dumigan	Macrophage sabotage: *Klebsiella pneumoniae*, a master manipulator
20	Pietro Maria Bertelli	Functional and metabolic response to hypoxia in endothelial progenitor cells

Full abstracts will be available on Slack

Poster Presentations

Number	Presenting Author	Title
21	Kevin Edgar	High fat diet induced cardiomyopathy in CX3CR1 knockout mice
22	Ciara Ross	Identification of novel small molecule inhibitors of Gremlin1 for the treatment of colorectal cancer
23	Anna Claire Devlin	Morphological, molecular and functional characteristics of iPSC-derived sensory neurons for the study of cough hypersensitivity
24	Laura Gritti	Inflammasomes: novel players in OPC proliferation and myelin regeneration
25	Alice Cheung	LEUCINE-RICH ALPHA-2-GLYCOPROTEIN-1 EXPRESSION IS UPREGULATED IN DIABETIC WOMEN WHO DEVELOP PRE-ECLAMPSIA AND SUPPRESSED BY HYPOXIA IN TROPHOBLAST CELLS
26	Joana Sa Pessoa	*Klebsiella pneumoniae* infection enhances reactive oxygen species production via NLRX1 to impair NEDDylation to promote infection
27	Arun Mariappan	NeuroD1-Tcf12 functional co-operativity drives the neurogenesis
28	Yuxin Wu	Human blood-derived endothelial colony forming cells as a new cell therapy for chronic nonhealing wounds
29	Olivier Touzelet	Sendai virus (SeV) as a vaccine vector for bovine respiratory syncytial virus (bRSV) and Nipah virus (NiV)
30	Catherine Kendall	Co-infection with SARS-CoV-2 and common nosocomially-acquired bacteria
31	Grace C Roberts	A physiologically relevant primary cell culture model for the study of RSV reveals an essential role for Toll-Like Receptor 4 in the virus lifecycle

Full abstracts will be available on Slack

Poster Presentations

Number	Presenting Author	Title
32	Arshad Rizvi	Dissecting the role of the new *Legionella* protease effector LegA7 in the manipulation of host cells
33	Varun Pathak	PENTRAXIN 3 PLAYS A KEY ROLE IN MYELOID ANGIOGENIC CELLS PHAGOCYTIC FUNCTION
34	Ashley Elliot	Utilising Ultrasound to help predict response to biologic therapy in Psoriatic Arthritis
35	Johnatas Silva	Mesenchymal Stromal Cells Extracellular Vesicles Improve Alveolar-Capillary Barrier Integrity in Acute Respiratory Syndrome through Alleviation of Mitochondrial Dysfunction
36	Elisa Peixoto	The role of the EPCR pathway in protecting endogenous repair cells in the choriocapillaris
37	Jasenka Guduric-Fuchs	RNA accumulation in aged endothelial cells links an interferon gene signature to cellular senescence
38	Sarah Chambers	Understanding the role of EPCR in Endothelial Colony Forming Cells
39	Marie Dittmer	Molecular signalling pathways underlying regulatory T cell (Treg)-enhanced oligodendrocyte differentiation
40	Kevin Harkin	Investigating the protective AT2-mediated signalling mechanism in malaria induced endothelial disruption
41	Diogo M. da Fonseca	*C. albicans* VS *C. parapsilosis*: Exploring the macrophage immune response induced by two distant cousins

Full abstracts will be available on Slack