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WELLCOME-WOLFSON INSTITUTE FOR EXPERIMENTAL MEDICINE

Virtual Postdoctoral Research Symposium 2022

#WWIEMpdrasympsoium2022

slack

27th January 2022

Microsoft Teams

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Symposium Timetable 27th Jan

09:00 – 09:15	**Welcome and Introduction** **Prof Jose Bengoechea**, Centre Director, Wellcome-Wolfson Institute for Experimental Medicine **Dr Claire Tonry**	
Session 1: Selected Oral Presentations **Chair: Dr. Karis Little**		
09:15 – 09:30	Dr. Amy Dumigan	"Klebsiella pneumoniae triggers a singular macrophage polarization to promote infection"
09:30 – 09:45	Dr. María Llorián-Salvador	"Regulatory T cells limit age-associated retinal degeneration"
09:45 – 10:00	Dr. Laura Gritti	"NLRP3 regulates progenitor cells after myelin damage in the central nervous system"
10:00 – 10:30	**Posters and Coffee Session 1 (Slack)** *See guidance on page 6*	
10:30 – 11:30	**KEYNOTE SPEAKER SESSION 1** **Prof. Clare Bryant** Title: TBC **Chair: Dr. Keren Turton**	
Vendor mini talks		
11:30 – 11:45	**Thermo**	ThermoFisher SCIENTIFIC
11:45 – 12:15	**Posters and Coffee Session 2 (Slack)** *See guidance on page 6*	

Session 2: Selected Oral Presentations **Chair: Dr. Elisa Peixoto**		
12:15 – 12:30	Dr. Aditi Singh	"Cell type transcriptomic signatures of human prefrontal cortex during development, ageing and neurological disorders"
12:30 – 12:45	Dr. Joana Sa Pessoa	"A trans-kingdom Klebsiella pneumoniae T6SS effector targets the mitochondria inducing NLRX1 and ROS production to promote infection"
12:45 – 13:00	Dr. Johnatas Silva	"ALGORITM (Part I): Mesenchymal Stromal Cell-Derived Extracellular Vesicles attenuate LPS-induced ARDS by Modulating Metabolic Changes in Human Lung Primary Cells"
13:00 – 14:00	**Posters and Coffee Session 3 (Slack)** *See guidance on page 6*	
14:00 – 15:00	**KEYNOTE SPEAKER SESSION 2** **Dr. Eoin Brennan** *'Decoding Non-coding RNAs in Cardiovascular disease'* **Chair: Dr. Claire Tonry**	
Session 3: Selected Oral Presentations **Chair: Dr. Aditi Singh**		
15:00 – 15:15	Dr. Hojjat Naderi-Meshkin	"Diabetic Patient iPSCs-derived Vascular Organoids Exhibit Dysfunctional Endothelial Cell Subpopulation"
15:15 – 15:30	Dr. Jonathon Dean Coey	"Photocatalytic inactivation of enveloped and non-enveloped viruses including SARS-CoV-2 by titanium dioxide (TiO2)-containing plastic film"
Vendor mini talks		
15:30 – 15:45	**Leica**	
15:45 – 16:15	**Break**	
16:15 – 16:30	**Dr. Amy Dumigan – Postdoc achievements & Updates from PCDC** **Dr. Vijay Tiwari – Closing remarks and awards**	

Poster Speed Dating and Imaging Competition

Poster Sessions

Postdocs are asked to prepare **up to 4 slides** (including a title slide) with **a 3-minute narration** of their work. You can easily record audio to accompany your slides using powerpoint or keynote (for mac users) - see links below for guidance

On the Slack workplace (which I will link below), you will see a channel with your name. You will have to upload your powerpoint to your own channel by clicking into your channel and attaching the file in the chat box and sending. **Postdocs must also upload their abstracts to their channels**.

Poster presentations must be uploaded and available for judging **by Wednesday 26th January**. On the days of the actual symposium, postdocs must be online during the poster sessions to engage with feedback on your channel.

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.

Postdocs who have engaged with **at least 10** other poster presenters will be entered into a prize draw. Winners will be announced on Yammer on **1st Feb 2022**.

Vendor Networking

Sponsors of this year's symposium will each have a 'virtual' stand on the **Slack platform**. All attendees are strongly encouraged to visit sponsors at their 'channels' and engage with them using the chat function.

Engagement with sponsors will be monitored by the symposium organisers and anyone who has **engaged with all sponsors** will be entered into a **prize draw!**

Imaging Competition

During the week of the symposium, 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.

Images will also be displayed in Slack and voters can vote by sending a private message to Claire Tonry on Teams

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 **Journal of Medical Microbiology**, who have sponsored this year's prizes.

Poster Judges

Effie Kostareli, David Simpson, Derek Brazil, Yvonne Dombrowski, Eric Campbell, Rebecca Coll, Mei Chen, Chris Watson, Guilherme Costa, Eleni Beli

Oral Session Judges

Tim Curtis, David Grieve, Andriana Margariti, Alan Stitt, David Courtney, Adrien Kissenpfennig

Abstract Selection Panel

Dr Amy Dumigan, Dr Claire Tonry, Dr Aditi Singh, Dr Rebecca Coll, Dr Vijay Tiwari, Dr Desi Malinova

Keynote and Oral Presentation Session Chairs

Dr Claire Tonry, Dr Keren Turton, Dr Aditi Singh, Dr Amy Dumigan
Dr Elisa Peixoto, Dr Karis Little

Symposium Organising Committee

Dr Claire Tonry, Dr Keren Turton, Dr Aditi Singh, Dr Amy Dumigan
Dr Rebecca Coll, Dr Vijay Tiwari, Dr Desi Malinova, Dr Elisa Peixoto, Dr Karis Little

Keynote Speakers

Professor Clare Bryant

University of Cambridge

Prof Bryant is one of few veterinarians who have a thriving research career whilst also working as a consultant in clinical pharmacology, as well as holding a significant teaching position

Prof Bryant and her research team use multidisciplinary approaches to understand **how bacteria are detected by the host** (through Pattern Recognition Receptors (PRRs)), Prof. Bryant's team are also studying how PRR recognition of allergen proteins or toxic proteins produced by patients **link to chronic inflammatory diseases** such as allergies and Alzheimer's disease.

Prof Bryant's group also study the molecular mechanisms underlying how ligands, such as endotoxin, interact with **TLR4/MD2 receptor complex** to recruit their adaptor signalling molecules, such as Mal and Tram, to initiate intracellular signalling. They are **using FRET analysis** and **single molecule florescence techniques** to study how TLRs form active signalling protein complexes and recruit adaptor proteins in real-time in live cells. Prof Bryant is also interested in how allergens, such as the cat dander protein Fel d1 enhances TLR4 signalling and whether inhibitors can be designed to prevent **allergen recognition**. Similarly other "toxic" proteins (amyloid beta and alpha synuclein) produced during diseases such as Alzheimer's and Parkinson's (respectively) can be recognised by TLR4 to induce inflammation and Prof Bryant's is also carrying out research to understand the molecular basis by which host recognition of these proteins occurs may lead to new treatments for these neuroinflammatory diseases.

Keynote Speakers

Dr. Eoin Brennan

Stanford University, CA

Dr. Eoin Brennan is an Assistant Professor at the Conway Institute, University College Dublin. Eoin graduated from University College Cork in 2004 with an honours degree in Genetics and in 2008 completed his **PhD at Queen's University Belfast**, studying the genetic and epigenetic mechanisms of diabetic kidney disease. In 2009, Eoin moved to the **UCD Diabetes Complications Research Centre** to study the genetics and signalling pathways implicated in diabetes and of vascular complications of diabetes. In 2014, Eoin continued his research at the **Baker IDI Heart and Diabetes Institute, Melbourne, Australia**. During this time he investigated endogenous lipids and non-coding RNA therapeutics in complications of Diabetes, with a specific focus on targeting vascular lesions in aortas and kidneys.

In 2019, Eoin was appointed an **Ad Astra Assistant Professor, School of Medicine, UCD**. His translationally-focused research group are investigating the **non-coding genome, and the development of novel treatment strategies for arresting the progression of atherosclerosis**. His multidisciplinary research programme **utilises preclinical in vivo models of atherosclerosis, RNA sequencing technologies and biobank development** and expansion in collaboration with local clinical sites. He is also a lead investigator in the **GENIE** (Genetics of Nephropathy, an International Effort) Consortium, an international consortium investigating **genetic mechanisms underpinning diabetic kidney disease**. His team are developing pipelines to prioritise and functionally interrogate lead genetic and epigenetic signals emerging from large case-control studies. These pipelines integrate kidney-cell based assays, kidney organoids and zebrafish models of kidney damage to interrogate candidate loci at the gene and mutation level.

Klebsiella pneumoniae triggers a singular macrophage polarization to promote infection

Amy Dumigan, Oisin Cappa, Brenda Morris, Joana Sá Pessoa, Ricardo Calderon Gonzalez, Grant Mills, Rebecca Lancaster, David Simpson, Adrien Kissenpfennig, Jose A. Bengoechea

Klebsiella pneumoniae (K.p) is a Gram-negative bacterial pathogen responsible for nosocomial and community-acquired infections including devastating necrotising pneumonia. Indeed K.p is an important secondary infection in relation to COVID-19 patients. Treatment options are rapidly diminishing as “last resort” antimicrobials are becoming ineffective. Therefore, better understanding of K.p pathogenesis has never been more urgent. As the first line of cellular response to infection, macrophages play an integral part in control of K.p. Herein, we demonstrate in detail that K.p can actively redirect macrophage polarisation for its own benefit, provide evidence for myeloid cells involved in this process and have delineated the cell signalling mechanism involved in full. .

Given the highly plastic nature of macrophage polarisation, it is no longer adequate consigned to one of two broad subgroups. Using scRNAseq, flow cytometry and Seahorse assays, we have successfully described what we term M2kp macrophages in detail. Via in vitro analysis we were able to discern that K.p induces and M2kp response via TLR4-Myd88-TRAM/TRIF-STAT6- PPAR pathway. We were able to reverse this phenotype via inhibition of STAT6 signalling in vivo leading to bacterial clearance in mice. Key features of M2kp were repeated in human macrophages and previously in porcine tissues and cell lines, inferring this robust response is ubiquitous across three species. In conclusion we have identified a novel mechanism by which K.p averts destruction and a method to utilise host immunity to counteract it- a highly important issue as we approach a post-antibiotic era.

Regulatory T cells limit age-associated retinal degeneration

María Llorián-Salvador, Alerie Guzmán de la Fuente (equal contribution); Alan Stitt; Denise Fitzgerald.

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

Age is a major risk factor for many retinal diseases estimated to severely impact on the visual function of >1.8 billion people by 2050. The pathobiology underpinning age-associated retinal degeneration remains ill-defined although low-grade chronic inflammation plays an important role. Regulatory T cells (Treg) have recently emerged as key players in tissue homeostasis, due to their capacity to regulate local immune responses, limit inflammation and their role in neuroprotection. However, Treg role in retinal homeostasis and age-associated retinal neurodegeneration is yet to be investigated.
Treg depletion was induced by intraperitoneal diphtheria toxin (DT) administration to Foxp3-DTR mice over 17 days (3 consecutive DT injections and then one DT injection every 4 days). The retinal neurovascular unit was assessed in young (3-4m) and aged (16-23m) Foxp3-DTR mice receiving DT, its vehicle (PBS) and C57BL/6 WT mice receiving DT.
We observed by immunohistochemistry in aged but not young Treg-depleted mice a significant decrease in photoreceptors and an alteration in the typical laminarity of cone and rod bipolar cells in the INL, indicating retinal neurodegeneration. Aged Treg depleted mice also showed enhanced Müller cell gliosis, especially in the ONL. Interestingly, an increase in Iba-1+ cells was found in the ONL of aged Treg-depleted mice and in the subretinal space of both aged and young Foxp3-DTR, together with RPE dysmorphology.
Depletion of Foxp3+ Tregs exacerbates retinal neurodegeneration, Muller gliosis and microglia infiltration in aged animals compared to young controls. Hence, Treg have a key role in regulating immune homeostasis in age-related retinal pathology.

NLRP3 regulates progenitor cells after myelin damage in the central nervous system

Laura Gritti, Emma McKay, Joseph Curran, Katherine Feeney, Daniel Crooks, Anne-Laure Boinet, Samara Fleville, Denise C. Fitzgerald, Peter Bankhead, Yvonne Dombrowsk

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast
Institute of Genetics and Cancer, University of Edinburgh, UK

Multiple Sclerosis (MS) is an immune-mediated demyelinating disease affecting about 2.5 million people worldwide. MS is characterised by a progressive loss of myelin and by the death of oligodendrocytes in a process called demyelination, which can ultimately lead to degeneration of neurons. In early stages of MS, neurodegeneration can be prevented by regeneration of myelin (remyelination). Although the molecular mechanism underlying this process is poorly understood, it has been demonstrated that immune mechanisms are required for efficient remyelination. Inflammasome associated proteins, such as the sensor protein NLRP3 and interleukin-1β (IL-1β), are abundant in plasma and central nervous system (CNS) tissue of MS patients as well as in murine models of the disease. Using a lysolecithin-induced focal demyelination model, we are able to study the different stages required for myelin regeneration, in order to elucidate whether NLRP3 is involved in remyelination of CNS lesions.

Our preliminary data indicate that inflammasome components were present within demyelinated CNS lesions from early stages of the myelin regeneration process. Using mice deficient in the inflammasome sensor NLRP3, we analysed the glial cells involved in the repair processes. Fewer proliferating oligodendrocyte progenitor cells (OPC), more differentiated oligodendrocytes and more microglia/macrophages were present within CNS lesions at early stages of the repair process, suggesting that NLRP3 may have an active role in regulating OPC proliferation and/or differentiation after demyelination. Further analyses into the underlying mechanisms will help to define the role of NLRP3 and the inflammasome complex in remyelination and might uncover molecular targets to regulate myelin repair.

Cell type transcriptomic signatures of human prefrontal cortex during development, ageing and neurological disorders

Aditi Singh, Vijay Tiwari

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

Human brain is divided into various anatomical regions that control and coordinate unique functions. Prefrontal cortex (PFC) is the large brain region comprises of a range of neuronal and non-neuronal cell types, sharing extensive interconnections with subcortical areas and involved in cognition control and memory functions. Here, using single cell transcriptome (scRNA-seq) data of human PFC from fetal (early, mid and late) to postnatal and ageing brain, we aim to discover novel transcriptomic regulators of neurogenesis and unravel how they contribute to the transcriptional reprogramming underlying neuronal development and play crucial role in lifetime cortical functions. Our studies have dissected the age specific, cell type transcriptional signatures of excitatory and inhibitory neurons in PFC as well as biological and molecular processes guided by them, which are relevant to development and ageing. In addition, transcription factor (TF) motif and TF network analysis on the gene expression datasets have identified key regulatory TFs whose activity changes in a spatiotemporally defined fashion along distinct lineage trajectories. Age specific cell type transcriptional signatures were then mapped to disease databases to find their relevance in neurological disorders and define age specific disease susceptibility. Our data shows that many TFs controlling the late onset disease are also differentially expressed in fetal brain. Ultimately, our results provide a mechanistic insight into novel regulators of cortical development and how their malfunction leads to various neurological disorders.

A trans-kingdom Klebsiella pneumoniae T6SS effector targets the mitochondria inducing NLRX1 and ROS production to promote infection

Joana Sá-Pessoa, Sara Lopez*, Kornelia Przybyszewska, Helina Marshall, Adelia Ova, Peter Barabas, Maria Molina*, Tim Curtis, Vitor J. Cid*, José A. Bengoechea

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast
*Departamento de Microbiología y Parasitología, Facultad de Farmacia, Universidad Complutense de Madrid and Instituto Ramón y Cajal de Investigaciones Sanitarias, Madrid, Spain

Klebsiella pneumoniae is an important cause of multidrug resistant infections worldwide with limited therapeutic options for its treatment. Subversion of inflammation is essential for K. pneumoniae survival during infection and work from our lab suggests that K. pneumoniae has evolved the capacity to manipulate host pathways to control the immune balance. Here, we uncover a hitherto unknown immune evasion strategy based on the deployment of a type VI secretion system (T6SS) effector to manipulate the mitochondria to induce ROS to limit inflammation. Mechanistically, a Klebsiella type VI secretion system (T6SS) effector induces an increase in the levels of calcium in the mitochondria from ER resources. This increase in calcium induces the activation of the Drp1 protein perturbing the mitochondria dynamics. Furthermore, the calcium increase is associated with the activation of mitochondria located innate receptor NLRX1, enhancing the production of ROS. Increased ROS levels led to the inactivation of the NEDD8-conjugating enzyme Ubc12. Ubc12 is responsible for the activation of the ubiquitin ligase complex SCF-βTRCP impairing the NEDDylation of its cullin-1 (Cul1) subunit in K. pneumoniae infected cells. The impairment of Cul1 NEDDylation leads to the lack of ubiquitylation of IκBα and β-catenin impairing innate inflammatory signalling in K. pneumoniae infection. In summary, our results indicate that by production of ROS via activating NLRX1, K. pneumoniae impairs the innate immune system via modulation of the host post translational modification NEDDylation. Our work illustrates new roles of the T6SS beyond antimicrobial warfare.

ALGORITM (Part I): Mesenchymal Stromal Cell-Derived Extracellular Vesicles attenuate LPS-induced ARDS by Modulating Metabolic Changes in Human Lung Primary Cells

Johnatas Silva, Declan Doherty, Catherine McClintock, Danny McAuley, Cecilia M. O'Kane, Anna Krasnodembskaya

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

Introduction: Previously, we demonstrated that mitochondrial dysfunction is an important mechanism of ARDS pathogenesis, and MSC EVs can restore functional activity of the injured cells through restoration of mitochondrial homeostasis. However the relative contribution of the two major energy-producing pathways: oxidative phosphorylation and glycolysis, to the pathophysiology of ARDS is not well understood as yet. Here we hypothesized that ARDS microenvironment will induce alterations in the levels of glycolysis and oxidative phosphorylation and MSC EVs will be able to modulate these alterations via mitochondrial transfer. Methods: EVs were isolated from bone-marrow MSCs with normal or dysfunctional mitochondria by ultracentrifugation. Primary human distal lung epithelial cells (HSAECs), pulmonary microvascular endothelial cells (HPMECs) and monocyte derived macrophages (MDMs) were stimulated with LPS or plasma from ARDS patients. Effects of MSC EVs on mitochondrial respiration and glycolytic flux were assessed using Seahorse metabolic analyser. Levels of inflammatory mediators were measured by ELISA, barrier properties were assessed by xCELLigence, MDM phagocytosis was assessed by flow cytometry. Results and Conclusion: Inflammatory stimulation resulted in pronounced cellular mitochondrial dysfunction, reduction of mitochondrial respiration, increase in glycolysis, TNF-a and IL-8 levels and barrier dysfunction in HSAECs, HPMECs and MDMs. MSC-EVs isolated from normal MSCs inhibited glycolytic flux, restored mitochondrial respiration and cell function while EV preparation which did not contain mitochondria was not effective. Analysis of the single cell seq data from in vivo lung injury model showed that MSC EVs administration can regulate the expression of essential genes involved in mitochondrial metabolism.

Diabetic Patient iPSCs-derived Vascular Organoids Exhibit Dysfunctional Endothelial Cell Subpopulation

Hojjat Naderi-Meshkin1, Magdalini Eleftheriadou1, Garrett Carney1, Victoria Cornelius1, Sophia Kelaini1, Andrew Yacoub1, Alan W Stitt1, David J Grieve1, Andriana Margariti1*.

Wellcome-Wolfson Institute for Experimental Medicine, SMDBS, QUB, Belfast,

Diabetes is one of the main risk factors for cardiovascular diseases (CVDs) in which patients usually represents vasculopathy. Mechanisms and manifestations of diabetes on endothelial dysfunction and other vascular cells have not been yet fully understood. Hence, developing 3D human vascular organoids (VOs) derived from induced pluripotent stem cells (iPSCs) of diabetic patients that mimic diabetic vasculopathy is of great importance. Confocal images confirmed presence of vascular cells (both endothelial cells and mural cells) in iPSCs-derived VOs. Application of pathology-simulating stress for 42 days on diabetic VOs, showed enhanced ROS production and Ac-LDL uptake measured by flow cytometry and confocal images; confirming that lab-made VOs could recapitulate diabetic vasculopathy. We further confirmed that diabetic-related pathogenesis can be worsened over time evidenced by enhanced anti-angiogenic proteins in condition media. Additionally, single cell RNA sequencing of these VOs allowed us to find several different endothelial cell subpopulations. Functional assay of differentially expressed genes between these subpopulations in diabetic versus non-diabetic VOs showed significant enrichment of EMT, ROS, and oxidative phosphorylation hallmarks in diabetic ECs; representing early signs of dysfunctionality. The observed heterogeneity of endothelial cells in the diabetic iPSCs-derived VOs suggests that diabetic disease-related responses may be specific to a small subpopulation of vascular cells. Gain of new function by this subpopulation may represents early signs of aberrant angiogenesis in diabetes. This study may help identifying biomarkers for diabetic disease progression and finding signalling molecules amenable to drug intervention.

Keywords: Diabetes, Cardiovascular Diseases, Blood Vessel Organoids, Induced Pluripotent Stem Cells, Diabetic Vasculopathy, Angiogenesis, Regenerative Medicine

Photocatalytic inactivation of enveloped and non-enveloped viruses including SARS-CoV-2 by titanium dioxide (TiO2)-containing plastic film

Johnathon Dean Coey, Connor G G Bamford, Ri Han, Andrew Mills

Wellcome-Wolfson Institute for Experimental Medicine, Queen's University Belfast
School of Chemistry, QUB

Infectious virions can persist on animate and inanimate surfaces for months if not removed. Such contaminated surfaces can contribute to disease transmission, particularly in high-risk settings such as healthcare environments. Here, we aimed to develop a novel, cost-effective, antimicrobial material for use in personal protective equipment (PPE) or on surfaces. To this end, we developed plastic films coated with titanium dioxide (TiO2), which when exposed to UVA light, catalyses the production of reactive oxygen species that degrade organic molecules rendering virions non-infectious. Influenza A virus (IAV), severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which are enveloped, and encephalomyocarditis virus (EMCV), which is non-enveloped, were used in this study under strict biosafety conditions. Virus inoculum (~10^6 TCID50 in 50 µL) was added to TiO2-containing plastic films and incubated under UVA light for 0.25-3 hours, at a fixed distance. At each time point, the inoculum was harvested and residual infectivity was measured via titration. Plastic films lacking TiO2, as well as incubation in the dark, were used as negative controls. TiO2-film had potent virucidal activity against all viruses tested although differences in efficiency between viruses was noted. Furthermore, antiviral activity was dependent on UV incubation time and TiO2 concentration. We developed an improved TiO2-film which reduced virus titres from 10^8 TCID50/mL to below the limit of detection within 2 hours. Given TiO2-containing plastics demonstrate antiviral activity, as well as being safe and affordable, we suggest such materials are attractive candidates for use in PPE or on surfaces in high-risk environments.

Poster Presentations

Presenting author's name	Title
Alerie Guzman de la Fuente	Ageing as a key regulator of the interaction between the immune system and oligodendrocyte progenitor cells
ARITRA DEB	Integrated analysis of long noncoding RNA and mRNA expression changes during cellular senescence of endothelial colony forming cells
Arun Mariappan	Deciphering the role of bHLH transcription factors cooperativity during cortical development
Claire Tonry	Identification of Novel Protein Biomarkers of Atrial Fibrillation
Clara Matute Blanch	Inflammation in multiple sclerosis induces a specific reactive astrocyte state driving non-cell-autonomous synaptopathy
Claudia Torres-Vargas	Functional characterisation of the Legionella pneumophila T4SS effector LegA7
Connor Bamford	Influenza A virus epithelial cell infectivity is enhanced upon super-infection with the important bacterial pathogen Klebsiella pneumoniae
Declan Doherty	Can mesenchymal stromal cell extracellular vesicles regulate macrophage phagocytosis via miR-203?
Diogo M. da Fonseca	Type I IFN signalling activation enhances macrophage candidacidal activity towards C. albicans via an oxidative- and nitrosative-independent mechanism
Elisa Peixoto	The role of the EPCR pathway in the choriocapillaris in the AMD context
Evan Troendle	Acquisition and modelling of 3D microvascular networks
Guanbo Wang	Burkholderia cenocepacia small heat-shock proteins play a role in antibiotic heteroresistance and virulence
Hong Guo-Parke	Altered Differentiation and Inflammation Profiles Contribute to Enhanced Innate Responses in Severe COPD Epithelium to Rhinovirus Infection

Full abstracts will be available on Slack

Poster Presentations

Hortensia-Clara Radulescu	The Northern Ireland COG-UK hub provides the local Sars-CoV-2 genomic sequencing required to support surveillance of the emergence and expansion of new variants
Jessica Eyre	Diabetic-like environment causes re-arrangement of ECFC cytoskeleton and focal adhesion complexes
Josy Augustine	2-hydrazino-4,6-dimethylpyrimidine (2-HDP) as a novel therapeutic for the neurovascular pathology of diabetic retinopathy
Judith Lechner	Exploring the APC/EPCR pathway to modulate the vasoreparative role of endothelial progenitors in the context of diabetic retinopathy
Karis Little	Characterising retinal neurovascular dysfunction in a mouse model of Alzheimer's disease combined with Type 2 diabetes
Keren Turton	The Type 3 Secretion System is a crucial mediator of macrophage-Achromobacter spp. interaction
Kevin Edgar	NOVEL HEART FAILURE BIOMARKER CLEC3B IS ASSOCIATED WITH CARDIAC FIBROSIS, AND IMPACTS CARDIAC FIBROBLAST CELL FUNCTION IN VITRO
Kevin Harkin	Deletion of Angiotensin II type 2 receptor (AT2) exacerbates retinal neovascularisation and haemorrhage in oxygen-induced retinopathy (OIR).
Kiran Mcloughlin	Novel ambient-temperature transport of live endothelial colony forming cells to replace cryopreservation and enhance efficacy of cell therapies
Lauren Kerrigan	An important role for hypomethylated Integrin beta-like 1 in ischaemic cardiac fibroblasts
Lindsay Broadbent	An endogenously activated antiviral state restricts SARS-CoV-2 infection in differentiated primary airway epithelial cells
Marie Dittmer	Molecular signalling pathways underlying regulatory T cell (Treg)-enhanced oligodendrocyte differentiation
Matthew Pilgrim	Deletion of ENPP1 exon 9 is associated with Bruch's membrane calcification and altered retina function in a transgenic mouse model
Michael C. McKelvey	
Michelle Naughton	Circadian rhythm in Multiple Sclerosis – Are immune cells "in the wrong place at the wrong time"?

Full abstracts will be available on Slack

Poster Presentations

Mohammed Inayatullah	Subclonal Epithelial to Mesenchymal Transition underlies chemotherapy resistance in Triple-Negative Breast Cancer
Olivier Touzelet	Characterization of a novel recombinant paramyxovirus vectored vaccine against Bovine Respiratory Syncytial virus
Peter Barabas	Targeting polyamine oxidases to inhibit diabetes-induced changes in the retina
Pietro M. Bertelli	Metabolic and functional changes in endothelial progenitor cells in response to hypoxia
Rachel Mairs	Investigation of the antimicrobial effect of apramycin on Mycobacterium abscessus infection
Rohan Anand	A randomised trial of the effects on recruitment and retention of including a wet-ink signature and photograph in the patient invitation letter for a clinical trial: results from a Study Within a Trial (SWAT 3 and SWAT 53)
Ryan Brown	The impact of hypertonic saline and/or carbocisteine treatment on sputum viscoelastic properties, infection and inflammation in patients with bronchiectasis
Sarah Chambers	Understanding Procr function in Endothelial Colony Forming Cells as progenitor cells
Steven Hancock	Deciphering the connection between the type VI secretion system and the lipopolysaccharide
Varun Pathak	Deficiency of PTX3 protects the diabetic retina from vasodegeneration, inflammation and improves visual function

Full abstracts will be available on Slack