CARDIFF UNIVERSITY
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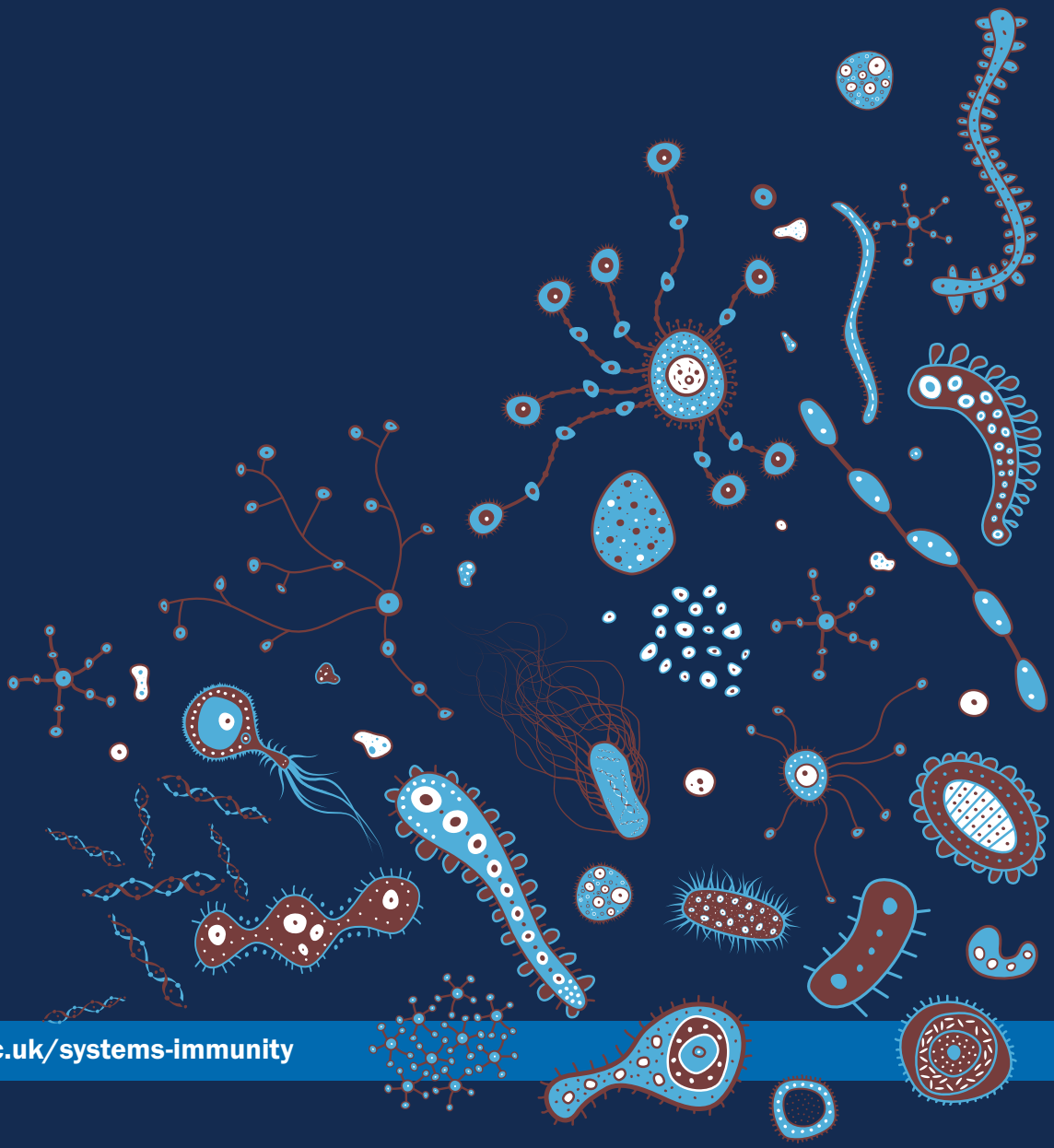
Systems Immunity Research Institute

Sefydliad Ymchwil Systemau Imiwnedd

Systems Immunity Research Institute

Our research provides a holistic view of chronic disease progression, the control of infection and mechanisms that determine an effective immune response



www.cardiff.ac.uk/systems-immunity

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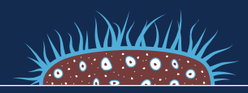
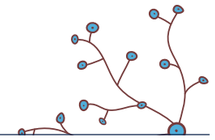




Message from the **Directors**

Professor Paul Morgan
Director of the Systems Immunity Research Institute

Professor Valerie O'Donnell
Co-Director of Systems Immunity Research Institute

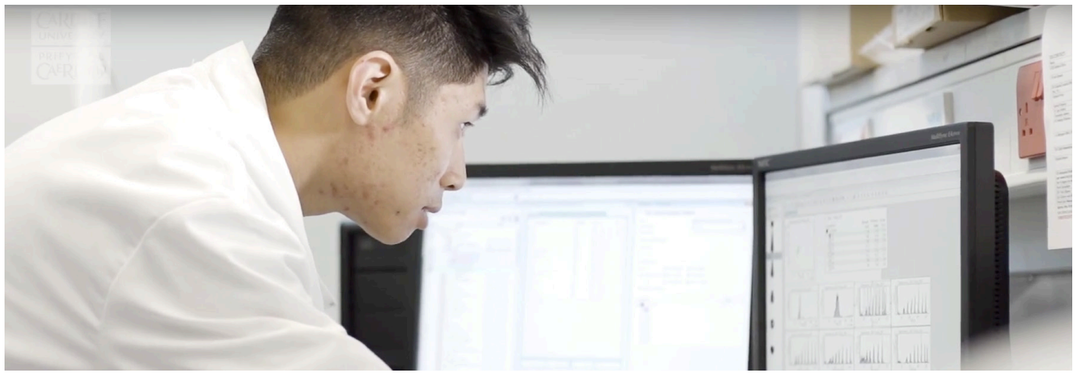
We are delighted to present the Systems Immunity University Research Institute (SIURI), established in 2015.

SIURI was awarded a £4.5M core grant from Cardiff University over 5 years to recruit new skilled faculty, embed infrastructure and support training activities including a Systems Immunity 4-year PhD programme. Our aim at that time was to bring together our large and diverse faculty, around 45 separate research groupings, located throughout two Cardiff Colleges into immune, infection and inflammation research. Biomedical research has entered a new era where our experiments increasingly generate a wealth of data requiring the power of mathematics and informatics in order to make sense of it. Harnessing the power of big data provides enormous opportunities for understanding immune disease, whether this is biochemical, cellular, model or human patient/ population based.

In our relatively short time, we have achieved some notable successes. Partnering with the Neurosciences and Mental Health Research Institute, we were awarded two new centres, the Hodge Centre for Neuropsychiatric-immunology and the MRC Dementia Research Institute Cardiff, which focuses primarily on translating immune and inflammatory genetic findings into mechanisms. We were also delighted to be recently awarded a Sêr Cymru II Chair in Systems Medicine (£5.5M) from Welsh Government, which will establish a Hub in Immunophenotyping initially researching sepsis in neonates. We host several Wellcome Trust Investigators and Fellows, and a major research portfolio of basic sciences and clinical programmes valued at over £75.6m funded by MRC, EU and all major research charities.

SIURI's research covers a broad portfolio that includes all our biggest killer diseases, and increasingly we see that these diseases do not strike in isolation, but susceptible individuals often contract more than one in tandem, and often present with acute or chronic neuropsychiatric complications. Understanding why inflammatory diseases present as a spectrum and on a continuum is of increasing interest to SIURI and will be a major priority area for new investment.

Systems Immunity Research Institute (SIURI) **Overview**



Our institute focuses on frontier-challenging "big-data-led" research into basic and applied immunology, infection and inflammation.

Embedding Big Data approaches, we aim to **better understand** the **complex 'systems'** that govern the immune system in health and disease.

We promote an integrated ethos that combines **world-leading experimental** and **clinical research** with **computational modeling, statistics and informatics.**

Our Institute builds on Cardiff's extensive strength in infection, immunity and big data, exploiting our findings in the development of new diagnostic and treatment modalities for our major killer diseases. We apply systems biology based-approaches to immunological research, embedding and incorporating the necessary skills and technologies across disciplines.

We host many collaborations with academic, clinical and industrial partners from across Cardiff University, the UK and international. A better understanding of the underlying highly complex mechanisms will allow us to develop new therapies and vaccines for disease intervention and prevention, and novel diagnostics for better patient management.

Our research covers many of the greatest health challenges of the 21st century including antimicrobial resistance, acute and chronic inflammatory disease (cardiovascular, diabetes, rheumatoid, kidney). We deliver long-term impacts on science, education and society, through tackling the national and global challenges of our time. Our ultimate aim is to make a difference in the clinic, improving patient outcome and public health as a whole.

SIURI - **Metrics at a glance**

Cardiff University was ranked 5th in the most recent Research Excellence Framework (REF) based on the quality of our research, a meteoric rise that confirms our place as a world-leading university. An impressive 87% of our research was assessed as world-leading or internationally excellent. The Systems Immunity Research Institute is part of the School of Medicine, ranked '51-100' in the QS rankings of World Medical Schools.

[153] Researchers

[66+] New grants since SIURI's inception in August 2015

[300+] Publications per year by SIURI faculty staff in top tier journals such as nature, science, pnas and embo j

[£75.6M] Current value of grants held by SIURI faculty members

[£4.6M+] Research council funding in 2017

[110] PhD students supervised by the research institute

Cardiff University ranked **[5th]** amongst UK universities in the recent Research Excellenece Framework

[£5.5M] Award from Wales Government for Ser Cymru Ii Chair in Systems Medicine

2 New Centres:

[£1M] Hodge Centre for Neuropsychiatric-Immunology

[£13M] UK Dementia Research Institute

Both centres are joint collaborations with the Neuroscience and Mental Health Research Institute (NMHRI), Cardiff

Innovation and **Impact**

Innovation, and the delivery of improvements in healthcare, is a key element of what we do within SIURI.

These improvements have involved all aspects of human disease, from improving our understanding of the mechanisms which underlie disease processes, to the design of new technologies to speed diagnosis and the design/delivery of new therapies. These innovations are targeted at improving the management and outcome of patients in a range of diseases from chronic- e.g. arthritis, diabetes and kidney disease, to life-limiting conditions e.g. infections and cancer. The translational and transformative element of the work has been witnessed in 2017, by the 10 human studies of technologies and therapies which were invented or developed within the URI.

In all of this activity the URI works closely with key industrial partners and SME's across Europe, Scandinavia and the US.

Key exemplars of this have been:

The award of $62M to fund Phase 2 studies of CXA 10, developed with Prof Val O'Donnell and her team, to modify inflammation, in renal disease and pulmonary arterial hypertension.

The award of €6.5M in EU Horizons 2020, and $11M from the US CFF to fund the ongoing human studies of alginate oligomer therapies in the management of cystic fibrosis.

In this activity the URI has worked closely with **NHS Wales**, to establish a "centripetal hub" of academic and clinical research excellence. The work has attracted not only prestigious inward research (and Company) investment to Wales, but has also seen investment in pivotal clinical trials at Cardiff and Vale Health Board.

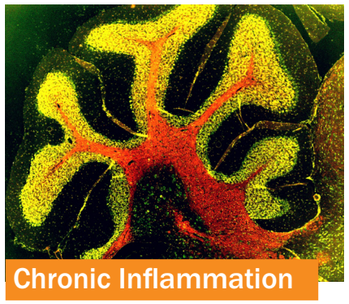
Professor Colin Dayan and his team leading the UK clinical studies of peptide immunotherapy in patients with Type 1 diabetes and their demonstration of promising results.
www.ft.com/content/3a685418-7cf9-11e7-9108-edda0bcbc928

Professor Andy Godkin and his team completed the groundbreaking TaCTiCC immunotherapy trial, in association with Cancer Research Wales in bowel cancer.
www.itv.com/news/wales/2017-09-22/breakthrough-in-bowel-cancer-treatment

Research Themes

SUIRI's basic, translational and clinical research utilises state-of-the-art technological big data generating platforms, bioinformatics, modelling and biostatistics expertise, combined with in vitro, model systems and patient/ population-based research.

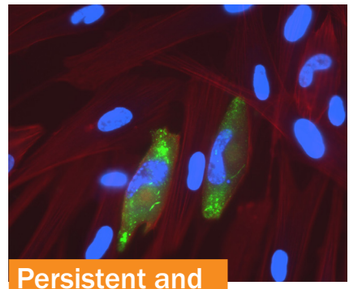
Our institute houses three separate but linked themes, which aim to dissect the multilayered interactions between the immune system, tissues and microorganisms.



Chronic Inflammation

Failure to regulate acute inflammation and the subsequent transition into chronicity is recognised as a key step in the development of cancer, auto-immunity and non-healing wounds, and the many diseases associated with ageing; among them arthritis, vascular inflammation, diabetes, dementia and vision loss.

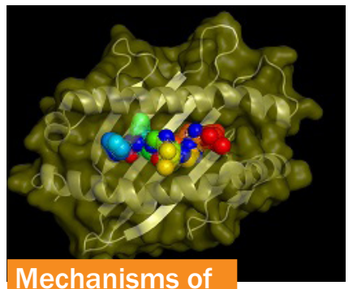
We apply systems approaches to understand how immune responses give rise to pathology, and explore dysregulated immune responses in disease to identify common pathways that are amenable to therapeutic intervention and aid patient stratification.



Persistent and resistant infections

The emergence of antimicrobial resistance represents one of the major global health threats. Persistent infections cause debilitating disease in the elderly and in immunocompromised individuals. At the same time, little is known about how our body interacts with the myriad of microorganisms it is constantly exposed to.

We use infection models, cutting edge bioinformatics frameworks and cohort studies in defined patient groups to elucidate complex host-pathogen relationships and define how the microbiome influences human health. The knowledge gained from these interdisciplinary and systems-based approaches informs the development of novel treatments, vaccines and diagnostics.

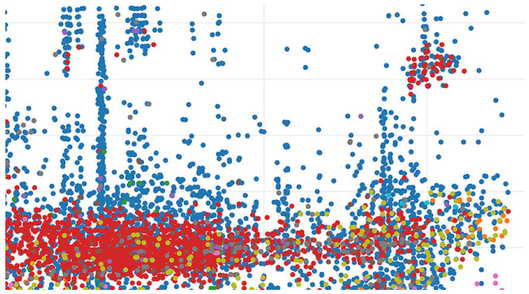


Mechanisms of immunity

Immune surveillance plays a key role in protecting the body against tumour development and maintaining cellular homeostasis. The underlying mechanisms can be exploited for novel therapies harnessing the immune system to combat cancer and autoimmune disorders.

We study fundamental processes including leukocyte trafficking, cytokine signalling, immune modulation and antigen recognition. We use bench-to-bedside approaches taking preclinical disease models through to the clinic, and clinical findings back to the bench. Systems approaches are essential for understanding why some individuals respond to treatment whilst others do not, thereby opening doors for tailored combination therapies comprising immune-modulating agents and stratified medicine approaches.

SIURI **Big Data Case Studies**



Lipid discovery and characteristation using informatics and statistics

The Cardiff Lipidomic Group, led by Prof Valerie O'Donnell, develops new methods to enable discovery of lipid classes important for health and disease, using mass spectrometry.

We roughly estimate that a large proportion of existing lipids in mammalian cells have not been structurally characterised to date. Many key discoveries remain to be made regarding this important class of endogenous biomolecules.

Using mass spectrometry we discovered large numbers of new lipids generated by human innate immune cells that can signal in inflammation. Many can stimulate blood clotting and are formed at higher levels than normal in patients with thrombotic disease.

With funding from Wellcome Trust and the European Research Council, we have developed new algorithms and software that allow high resolution mass spectrometry datasets to be rapidly analysed.

Starting with typically 66,000 features in a single cell lipid extract dataset, we need to reduce this to the approximately 5,000 signals that we know are real lipids. This tool is now available on GitHub (https://github.com/cjbrasher/LipidFinder)

We are particularly focusing on lipid discovery in innate immunity, infection, cell development, cardiovascular disease and dementia.

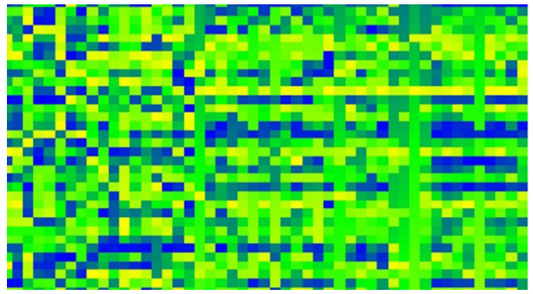
Peptide Identification Tool

Dr Barbara Szomolay has teamed up with ARCCA and the Warwick Systems Biology Centre (WSB) to expand the open-access tool PICPL (Peptide Identification from Combinatorial Peptide Libraries).

PICPL, which was developed at WSB, uses readouts of a powerful peptide scanning method called combinatorial peptide libraries (CPLs).

The new tool ranks peptides from self, viral, bacterial and fungal proteins based on CPL data, allowing the user to make use of the computational power of the University's Supercomputer, Raven, and thus reduce the time taken before results are available for analysis.

A web-based front end, running on a virtual machine and integrated into Raven, allows users to upload their CPL data and select the database they wish to scan against, automating what would normally be a complex technical exercise.



Diagnostic immune fingerprints

Research in the Eberl lab aims to identify diagnostically relevant biomarker signatures (immune fingerprints) for point-of-care diagnosis of patients presenting with symptoms of acute infections.

We assess such pathogen-specific local patterns quantitatively and qualitatively in different patient cohorts, long before traditional test results including microbiological cultures become available. The definition of the best possible biomarker combination and its validation poses considerable computational challenges, with approximately 1.27×1030 possible combinations of a panel of 100 different biomarkers in 100 individual patients.

We develop ad apply novel statistical tools to interrogate highly complex datasets and utilise supervised and unsupervised machine learning approaches and exhaustive searches on the RAVEN supercomputer in order to define and cross-validate relevant biomarker signatures.

This cross-disciplinary research has received support from the NIHR Invention for Innovation (i4i) programme, the MRC Confidence in Concept scheme and Health Technology Challenge Wales, and is conducted in collaboration with basic, computational and clinical scientists as well as with industrial partners.

If the concept of pathogen-specific diagnostic fingerprints were to be introduced into clinical practice, targeted therapy could be given immediately, improving outcomes, avoiding the emergence of antibiotic resistant strains, and saving costs for the NHS.

Identifying therapeutic targets during virus infection

Research within the Viral Immunology group aims to characterise the ways in which virus infections manipulate infected cells, and how the host immune system responds to those infected cells. These studies reveal both the molecular underpinnings of immune-cell function, and have identified novel therapeutic targets.

The herpesvirus human cytomegalovirus (HCMV) causes severe disease in patients who become immunocompromised, and can cause lifelong disability in babies if caught during pregnancy. Like all herpesviruses, once caught, the virus is never cleared but remains latent within the individual, reactivating and causing disease at later times. A vaccine for HCMV has been recognised as a high priority, but none is licensed. In collaboration with colleagues at Cambridge (Dr. Michael Weekes and Prof. Paul Lehner), and Harvard University (Prof. Steve Gygi), Cardiff researchers are using high resolution comparative proteomics to dissect how HCMV manipulates the host cell, and how different arms of the immune system respond to infected cells.

These studies have characterised over 8,000 proteins during the course of an infection, and have identified hundreds of these that are targeted for modification by specific viral functions. They have identified unique signatures of infection that can be used to identify infected cells, they have found novel anti-viral factors within cells, and have revealed novel ways in which HCMV manipulates the host cell surface to avoid being killed by the immune system. Furthermore, by studying the ways in which HCMV avoids the immune system, these studies have identified novel and unexpected ways by which immune cells differentiate healthy or pathogen infected cells.

SIURI **Research Highlights**

[1]

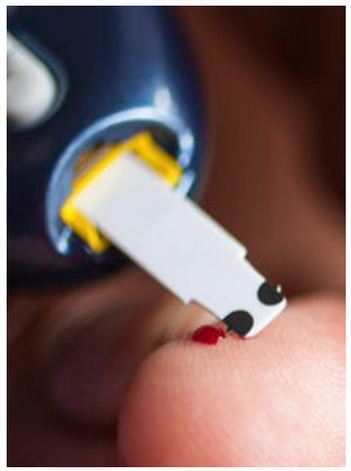
Halting the spread of Dysentry

SIURI scientists have shed new light on a disease estimated to affect 165M people worldwide. Despite recent improvements in sanitation and the provision of clean water around the world, dysentery remains a major worldwide public health burden that most frequently affects children in low-income countries. **Dr Thomas Connor** from SIURI and **Professor Nick Thomson** from The Wellcome Trust Sanger Institute have used the latest big data genomic techniques to reveal more about the bacteria Shigella flexneri, known as a leading cause of the disease. Published in the academic journal eLife, the team sequenced the DNA of Shigella flexneri from samples taken from Africa, Asia, South and Central America along with samples from historical collections dating back to 1913.

[2]

Do germs cause type 1 diabetes?

Germs could play a role in the development of type 1 diabetes by triggering the body's immune system to destroy the cells that produce insulin, new University research suggests. SIURI research, published in The Journal of Clinical Investigation, provides a first ever glimpse of how germs might trigger killer T-cells to cause type 1 diabetes, but also points towards a more general mechanism for the cause of other autoimmune diseases. **Professor Andy Sewell**, lead author, said: "Killer T-cells are extremely effective at killing off germs, but when they mistakenly attack our own tissues, the effects can be devastating." During type 1 diabetes, killer T-cells are thought to attack pancreatic beta cells. These cells make the insulin that is essential for control of blood sugar levels. "When beta cells are destroyed, patients have to inject insulin every day to remain healthy."



[3]

Fresh insight into rheumatoid arthritis offers hope for transforming patient care

Scientists have discovered what they believe has the potential to prevent the onset of an aggressive and hard-to-treat form of rheumatoid arthritis/RA - a condition that affects 700,000 adults in the UK. Published in the Journal of Experimental Medicine, a team of immunologists from Cardiff University tread new ground in describing how an immune system protein - interleukin-27 - regulates the inflammatory process in lymphoid-rich rheumatoid arthritis, which causes the characteristic symptoms of swollen and painful joints. Dr Gareth Jones, from Cardiff University School of Medicine's Institute of Infection & Immunity, said: "In all forms of rheumatoid arthritis, it is widely understood that early intervention offers the best chance for clinical remission. The sooner treatment begins, the more effective the therapeutic response is likely to be. The key is identifying which drug is best suited for an individual patient. Making the correct treatment decisions, sufficiently early in the disease process will improve disease outcome, enhance a patients wellbeing and overall quality of life. Our research is identifying crucial pathways and mechanisms that allow us to distinguish between different sub-types of rheumatoid arthritis, using experimental models that mirror human forms of the disease." Each year the NHS spends £560M on biological drug treatments to mitigate its effect. The research is funded by Arthritis Research UK.

[4]

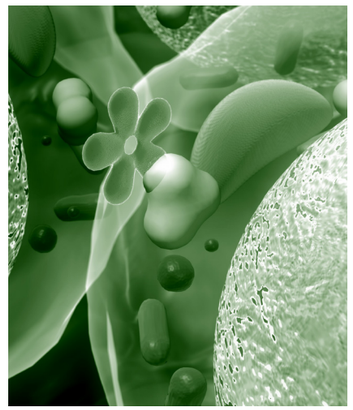
Burden of Antibiotic Resistance in Neonates from Developing Societies (BARNARDS)

Professor Timothy Walsh is the PI of a Gates funded research grant entitled the Burden of Antibiotic Resistance in Neonates from Developing Societies (BARNARDS) the research team are part of the Institute of Infection and Immunity, within the School of Medicine. BARNARDS aims to provide the means, support, network and tools to understand the impact of antibiotic resistance on neonatal morbidity and mortality in addition to identifying possible interventions to minimise its impact. Local clinical epidemiology is supported with state-of-the-art molecular genetics by performing the characterisation of the microbiota of the samples in study, particularly of the multi-drug resistant Gram-negative Bacteria (MDR-GNB). In addition, the UK team are identifying the prevalence of MDR-GNB which is carried as normal microbiota in pregnant women which can be identified as aetiological agents of MDR-GNB neonatal sepsis. BARNARDS also looks socio-demographic traits such as education, overcrowding, access to clean water, sanitation conditions, living conditions, socioeconomic status and antibiotic usage which may have a role on the development of neonatal sepsis. In addition, hospital environmental samples are collected, including from medical devices and surfaces, in order to understand if a link between the microbiota of the hospital environment and the late onset of sepsis can be established. The UK team have just been successful in obtaining a second round of funding until 2022 for this ground-breaking research programme after a recent visit to Seattle (October 2017). The Gates Foundation are using BARNARDS as the focal point for their new AMR programme.

[5]

Immune systems of type 1 diabetics can be 'retrained'to stop destroying insulin.

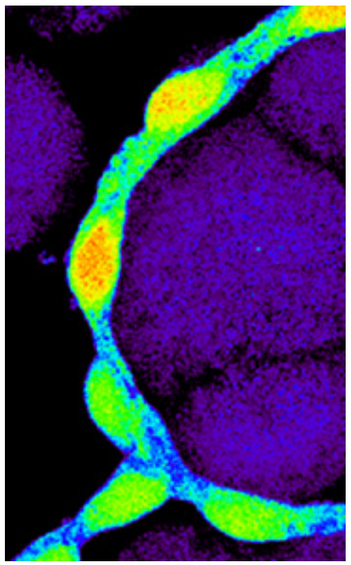
SIURI received widespread UK media coverage following successful trials of a pioneering new therapy which showed that damaged immune systems of diabetics can be 'retrained' to stop them destroying insulin. Researchers observed noticeable changes in the behaviour of the immune systems of type 1 diabetes patients that had been injected with peptides, small fragments of the protein molecules found in the beta cells of the pancreas. Prof Colin Dayan, the clinical Chief Investigator for the study, said: "It was encouraging to see that people who receive the treatment needed less insulin to control their blood glucose levels, suggesting that their pancreas was working better." The study 'Metabolic and immune effects of immunotherapy with proinsulin peptide in new-onset type 1 diabetes' is published in Science Translational Medicine.



[6]

New insights into AcutePancreatitis

Studying signalling events in living segments of the pancreas, allowing simultaneous recordings from different cell types, researchers from SIURI have discovered that bile acids have a hitherto unexpected destructive action on a novel cell type in the pancreas, namely the so-called stellate (star) cells. MRC Professor Ole Petersen FRS, the senior author of the study, said: "These new findings radically change our view of the mechanism by which bile acids cause Acute Pancreatitis and now focuses our thinking on the hitherto neglected role of the stellate cells in initiating the disease process." The study – Bile acids induce necrosis in pancreatic stellate cells dependent on calcium entry and sodium-driven bile uptake – is published in The Journal of Physiology.



Engagement and **Involvement**

Our research on the immune system and the exploitation of our findings in the clinic have long-term impacts on science and society in Wales, the UK and globally.

The Institute's interdisciplinary teams address many of the greatest health challenges of the 21st century. We strive to deliver long-term impacts on science, education and society as a whole, and make a real difference in the clinic. Our engagement and involvement activities therefore form a central part of our research. We aim to reach out to all stake-holders and engage with patients, health care providers, schools, funders, policy makers and industry.

We develop documents, activities, workshops, games, seminars and exhibits that focus on all aspects of our research and deliver activities in both English and Welsh.

Our Lay Faculty play a crucial role in reviewing research priorities within the institute and fostering a close dialogue between our basic and clinical researchers with the general public. The Lay Faculty gives input into research proposals, impact statements, press releases and study protocols, and contributes to the dissemination of research findings to relevant target groups. The creation of the Lay Faculty has benefited greatly from our close links with Health and Care Research Wales via the Involving People Network, Wales Cancer Research Centre, Wales Kidney Research Unit and HealthWise Wales.

We engage with the wider academic community by hosting scientific events and seminars in close partnership with professional bodies such as the British Society for Immunology and Academia Europaea.

Updates of our research and engagement activities feature regularly on the Cardiff University website and social media channels, as well as in print and online magazines such as ReMEDy, Cardiff Connect and Advances Wales, and are disseminated via our presence in social media including Facebook and Twitter.

Simone Cuff at Hay Festival 2017

At Techniquest in Cardiff Bay, August 2016

We engage with patients to define real clinical need and inform them about findings that may be of direct relevance for understanding and managing their condition, and for improving public health via diagnostics, therapies, vaccines and life-style choices. Patients are also directly involved in our research via participating in HealthWise Wales, recruitment to clinical trials and observational studies, and donation of samples to tissue banks.

We raise awareness for acute and chronic diseases in close collaboration with local, regional and national charities and together with dedicated patient focus groups.

We develop workshops, seminars and exhibits that focus on all aspects of our research and work closely together with Techniquest Science Museum on public engagement activities. We participate in the public Science in Public Health Lectures series and are involved in events such as the Cardiff Science Festival, Soapbox Science, Hay Festival, Pint of Science and The National Eisteddfod.

At the Soapbox festival in Cardiff 2016

We provide evidence and make recommendations to governments and international agencies such as the World Health Organisation concerning matters affecting science, scholarship and public health in Wales, the UK, Europe and globally.

We reach out to teachers and school children via the Science in Health: Live! events as part of Cardiff University's contribution to the National Science and Engineering Week, and via the STEM ambassador scheme. We participate in staging a Wales-wide inter-school competition for year 10 pupils modelled on the TV quiz show University Challenge and held both in English (The Life Sciences Challenge) and in Welsh (Her Gwyddorau Bywyd).

We cooperate with local and national newspapers, radio and TV stations, including BBC Wales and Made in Cardiff TV, and have close links to Radio Glamorgan, the award-winning hospital radio station serving the University Hospital of Wales.

We co-host an annual 'Infection and Immunity Away Day' in the Millennium centre, Cardiff bay with around 200 delegates, sponsors and external speakers

SIURI **Collaboration**

We collaborate globally, working with top researchers across the world including in UK, Europe, USA, South America and Asia. By adopting experimental model systems, bioinformatic approaches and observational clinical studies, our collaborative investigations have identified fundamental mechanisms of disease progression that contribute to improvements in diagnosis, patient stratification and treatment.

In 2017, we agreed a strategic partnership with the new **Biomedicine Discovery Institute** at Monash University (Melbourne, Australia). This partnership, led by our joint faculty member, Prof Jamie Rossjohn is based on long established and emerging research streams at Monash, the Hudson Institute of Medical Research and the University of Melbourne. Our partnership will explore the application of Big Data science to key societal challenges that address mechanisms of inflammatory and infectious diseases. Topics of ongoing collaborative studies include the molecular determinants and biological relevance of immune interactions in health and diseases such as cancer, autoimmunity, infection and atherosclerosis.

Embedded Centres:

Our faculty lead or are members of several externally-funded centres and institutes, including:

1. MRC Dementia Research Institute Cardiff (Julie Williams, Paul Morgan, Philip Taylor)
   www.cardiff.ac.uk/dementia-research-institute
2. Wales Kidney Research Unit (Donald Fraser)
   kidneyresearchunit.wales/en/
3. Diabetes Research Unit Cymru (Colin Dayan)
   www.diabeteswales.org.uk
4. Hodge Centre for Neuropsychiatric Immunology (Paul Morgan)
   www.cardiff.ac.uk/news/view/522470-the-hodge-centre-for-neuropsychiatric-immunology
5. LIPID MAPS, the global online database and resource for lipidomics
   www.lipidmaps.org
6. Cardiff Regional Experimental Arthritis Treatment and Evaluation Centre (Ernest Choy)
   www.cardiff.ac.uk/research/explore/research-units/cardiff-regional-experimental-arthritis-treatment-and-evaluation-centre

Associated External Centres

We work closely with several local research institutes, centres and networks.

1. MRC UK Dementias Platform (UKDP) NISCHR Wales Cancer Research Centre
   www.healthandcareresearch.gov.wales/wales-cancer-research-centre
2. The MRC Centre for Neuropsychiatric Genetics and Genomic (MRC CNGG)
   www.cardiff.ac.uk/mrc-centre-neuropsychiatric-genetics-genomics
3. All-Wales Life Sciences and Health Network in Drug Discovery (LSHNDD)
   www.lsrnw.ac.uk
4. Wales Gene Park
   www.walesgenepark.cardiff.ac.uk
5. Healthwise Wales
   www.healthwisewales.gov.wales

www.cardiff.ac.uk/systems-immunity

Careers and Training

4 year PhD programme in Systems Immunity

Our four year PhD programme in Systems Immunity provides an outstanding research environment and training for experimental, clinical and theoretical immunologists.

Our emphasis is on training a new generation of highly skilled interdisciplinary researchers equipped for the demands of increasingly common ‘big data’ projects, specifically those utilising ‘omics approaches, large clinical datasets and mathematical modelling.

We recruit from a broad profile of undergraduate disciplines, from the biological to computer sciences, and provide core training in bioinformatics and biostatistics and laboratory sciences. Training is drawn from our MSc courses, as appropriate for the individual student, but is supplemented by bespoke offerings based on one-to-one assessments made by in house bioinformaticians at the start of studies. All students are assigned mentors for the course of their studies as part of a proactive support structure.

The PhD programme in Systems Immunity commenced in 2015. Current research projects, include, for example, study in the fields of cancer immunology, biomarker discovery and disease stratification and viral immune evasion.

Funding is awarded to the most competitive applicants and pays stipend (standard research council rates for the four years), fees (‘home rate’ for UK and EU students) and consumables.

Enquiries:

For further information, please email the Institute’s Postgraduate Research Team:

landl-PGR@cardiff.ac.uk

Other PhD opportunities in the Systems Immunity Research Institute and the broader Immunology community?

PhD positions are usually advertised both by individual supervisors within the Systems Immunity Research Institute:
www.cardiff.ac.uk/systems-immunity

and Division of Infection and Immunity:
www.cardiff.ac.uk/medicine/research/divisions/infection-and-immunity

In addition, Immunological projects are advertised as part of the Cardiff’s School of Medicine annual PhD studentship call:
www.cardiff.ac.uk/medicine/courses/postgraduate-research

and as part of the MRC GW4 BioMed Doctoral Training Partnership:
www.gw4biomed.ac.uk

Note that all of these projects/studentship opportunities will be advertised on: **www.FindaPhD.com**

Self-funded students:

Students with their own funding should contact their preferred supervisors directly or via the Institute’s postgraduate research admin team:
landl-PGR@cardiff.ac.uk for more general enquiries.

SIURI and associated **Technology Platforms**

Our research takes advantage of state-of-the-art 'big data' generating platforms, which comprise whole genome sequencing, genome-wide linkage and association studies, cutting edge imaging and 'omics platforms, polychromatic flow cytometry and protein crystallography.

Expertise in advanced bioinformatics, biostatistics, programming and mathematical modelling is facilitated through the SIURI Bioinformatics Hub led by Dr Robert Andrews. We have close links to the Data Innovation Institute at Cardiff University. Central Biotechnology Services (CBS) is a certified technology facility that offers expertise and experimental platforms in genomics and bioinformatics, proteins and diagnostics, and cell analysis and imaging.

The Cardiff Lipidomic Group houses state-of-the-art mass spectrometry for lipid/metabolite measurement including high sensitivity Q-Traps and a high resolution Orbitrap Elite.

A dedicated biological service unit provides a barrier area (including an embedded Cat-3 laboratory) for the study of physiological model systems relevant to disease and immunity. This also houses imaging facilities allowing researchers to utilise X-ray, radioisotope, fluorescence and luminescence in experimental models. Large scale resources include experimental magnetic resonance imaging and magnetic resonance spectroscopy. Provision of research imaging is supported by the Wales Research and Diagnostic PET Imaging Centre (PETIC), and the Cardiff University Brain Research Imaging Centre (CUBRIC).

We access next generation sequencing and bioinformatics support for exome and genome sequencing, and whole transcriptome analysis via our close links with Wales Gene Park and MRC Center for Neuropsychiatric Genetics and Genomics.

Clinical research and tissue banks

We partner with the Clinical Research Facility (CRF) to access infrastructure for the successful translation of basic biomedical science developments into clinical practice, in close collaboration with the South East Wales Trials Unit (SEWTU) and the Wales Cancer Trials Unit.

Access to human tissues is facilitated via the Biobank Wales alliance that hosts biobanks such as the Wales Cancer Bank, the Wales Kidney Tissue Bank, the Welsh Neuroscience Research Tissue Bank and the Cardiff Regional Experimental Arthritis Treatment and Evaluation Centre (CREATE) Biobank.

Many of our clinical studies are registered on the UK Clinical Research Network Study Portfolio, for instance:

- Immune cell studies in Type 1 Diabetes (ISTID)
- Primary care use of a C-Reactive Protein Point of Care Test in COPD (PACE)
- Patient immune responses to infection in Peritoneal Dialysis (PERITPD)
- Point of care testing for urinary tract infection in primary care (POETIC)

SIURI Faculty Lead **Profiles**

Dr Ann Ager

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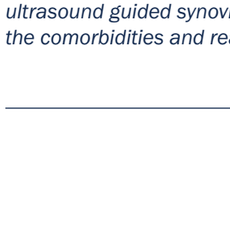
The focus of the Ager lab is to determin how T lymphocytes move around the body in order to protect against infection, fight cancer and repair damaged tissues. This has resulted in a body of work studying the molecular basis of lymphocyte-blood endothelial cell recognition which directs lymphocyte trafficking to organised lymphoid tissues and sites of infection and immunity. A major focus has been the regulation of L-selectin expression on T lymphocytes and its impact on physiological and pathological T cell trafficking via specialised high endothelial venule (HEV) blood vessels. Recent studies have revealed an essential role for L-selectin in the recruitment of killer T cells into flu-infected lungs for virus clearance which was highlighted by the BBC. Current studies are exploring pharmacological and genetic approaches to boost L-selectin expression on T lymphocytes to help killer T cells find and destroy other viruses. We are also exploring whether manipulating L-selectin on cancer-killing T lymphocytes, such as CAR-T cells, increases their ability to seek out and destroy solid cancers.

Publications

1. Ager A (2017) High Endothelial Venules and Other Blood Vessels: Critical Regulators of Lymphoid Organ Development and Function. Front Immunol 8:45.

2. Mohammed, R N, Watson H. A, Vigar,M Ohme J, Thomson A,. Humphreys I R and Ager, A. (2016) L-selectin is essential to deliver activated CD8+ T cells to virus-infected organs for protective immunity. Cell Reports 14, 760–771;.



Dr Stuart Allen

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My research focuses on the identification of cell-My research interests are in the field of mobile and social computing, covering both practical problems with direct applications to industry and policy making, and interdisciplinary collaborations exploring the opportunities and challenges of emerging technologies. I have 20 years of research experience in wireless network modelling and optimisation to improve efficiency through automated planning and management of network infrastructure and resources. Within SIURI, I collaborate with the O'Donnell group, supporting algorithm and software generation for lipidomic dataset analysis.

Publications

1. Noe, B.et al. 2017. Timing rather than user traits mediates mood sampling on smartphones. BMC Research Notes 10, article number: 481.

2. Chun, Y.et al. 2017. Device-to-device communications: a performance analysis in the context of social comparison based relaying. IEEE Transactions on Wireless Communications , pp. 1: Cell M or JCI Insight Volume: PP, Issue: 99.

Professor Ernest Choy

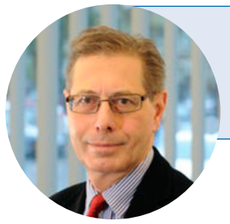
choyeh@cardiff.ac.uk
+44 (0)29 2068 7092

My research interest as Director of Arthritis Research UK CREATE Centre is in precision medicine, developing prognostic tests and new treatments for patients with inflammatory arthritis. Patients with inflammatory arthritis have increased risk of developing multiple comorbidities such as cardiovascular disease, depression, metabolic syndrome and fatigue. We have expertise in understanding the basic mechanisms that lead to these comorbidities such as the role of cytokines in accelerated atherosclerosis. Another research interest is improving translation of discovery research into clinical benefit through novel clinical trial methodology and ultrasound guided synovial biopsy. Using the SAIL databank in Wales, I have studied the comorbidities and real-life impact of rheumatic diseases.

Publications

1. Burmester, G.et al. 2017. Low immunogenicity of tocilizumab in patients with rheumatoid arthritis. Annals of the Rheumatic Diseases 76, pp. 1078-1085.

2. Porter, D.et al. 2016. Tumour necrosis factor inhibition versus rituximab for patients with rheumatoid arthritis who require biological treatment (ORBIT): an open-label, randomised controlled, non-inferiority, trial. The Lancet 388(10041), pp. 239-247.



Professor Peter Collins

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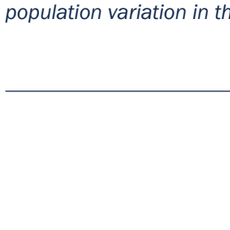
My research focuses on the diagnosis and treatment of inherited and acquired bleeding disorders. The work spans both clinical studies and basic science and I collaborate with a number of groups at Cardiff and across the world.

My clinical work is based in the Cardiff Haemophilia Centre and involves treating people with inherited and acquired disorders of haemostasis and thrombosis. I am particularly interested in the bleeding problem of post-partum haemorrhage and am developing new treatments based on pro-coagulant therapies. I also study the role of lipid surfaces in promoting blood clot formation

Publications

1. Pleines, I.et al. 2017. Mutations in tropomyosin 4 underlie a rare form of human macrothrombocytopenia. The Journal of Clinical Investigation 127(3), pp. 814-829.

2. Collins, P.et al. 2017. Viscoelastometric-guided early fibrinogen concentrate replacement during postpartum haemorrhage: a double blind randomised controlled trial: OBS2. British Journal of Anaesthesia Volume 119, Issue 3, Pages 411–42



Dr Thomas Connor

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My research is made possible by a combination of technologies that enable us to explore organisms at a resolution that has never before been possible. Firstly, whole genome sequencing, combined with high quality metatdata provides the datasets that we can use to derive the answers to the questions that we seek. Secondly, using computational and mathematical approaches, we are able to make sense of the "Big Data" challenge that is posed by the large, rich datasets.Research within my group is therefore characterised by developing and applying population genomics, comparative genomics, and phylogenetics to elucidate the natural histories of microbial pathogens. In a number of cases we have developed tools or approaches to analyse our data. However, in all cases we start first with the biological questions, and then develop the approaches to answer our question. So it could be said that while what we do is broadly Bioinformatics, the research focus is on the Biology first, and the informatics provides the tools to unlock the data that we produce.

Publications

1. Wong, V.et al. 2016. An extended genotyping framework for Salmonella enterica serovar Typhi, the cause of human typhoid. Nature Communications 7, article number: 12827.

3. Baker, K.et al. 2015. Intercontinental dissemination of azithromycin-resistant shigellosis through sexual transmission: a cross-sectional study. The Lancet Infectious Diseases 15(8), pp. 913-921.

Professor Colin Dayan

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I have a long established interest in translational research in the immunopathology of type 1 diabetes and am currently conducting early phase clinical trials in the development of antigen specific immunotherapy. I was part of 2 major EU FP7 programme grants in type 1 diabetes (and coordinator on one), a member of the Juvenile Diabetes Research Foundation Medical and Scientific Committee and the Welsh Diabetes Research Unit.

I have research interests in thyroid disease which include thyroid autoimmunity, thyroid hormone replacement and bioavailability, genetic epidemiology as applied to population variation in thyroid hormone bioavailability and thyroid eye disease.

Publications

1. Clement, M.et al. 2016. Cytomegalovirus-specific IL-10-producing CD4+ T cells are governed by type-I IFN-induced IL-27 and promote virus persistence. Plos Pathogens 12(12), article number: e1006050.

2. Sowerby, J.et al. 2017. NBEAL2 is required for neutrophil and NK cell function and pathogen defense. The Journal of Clinical Investigation 2017;127(9):3521–3526

Dr Matthias Eberl

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Since 2000 I have concentrated on defining the role of human T cells in bridging innate and adaptive immune responses. T cells are the prototype of 'unconventional' lymphocytes in that they are not restricted by classical MHC and display characteristics of 'conventional' T cells, NK cells and myeloid cells. Normally only constituting a minor population in human blood, they occupy a unique niche in microbial recognition and contribute to tumour surveillance. More recently we have started to characterise early immune responses in acutely infected patients and define pathogen-specific signatures of cellular and soluble biomarkers ('immune fingerprints').

Publications

1. Raby, A.et al. 2017. Toll-like receptors 2 and 4 are potential therapeutic targets in peritoneal dialysis-associated fibrosis. Journal of the American Society of Nephrology 28(2), pp. 461-478.

2. Liuzzi, A.et al. 2016. Unconventional human T cells accumulate at the site of infection in response to microbial ligands and induce local tissue remodeling. Journal of Immunology 197(6), pp. 2195-2207.

Dr Daniel Farewell

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+44 (0)29 2068 7247

I am interested in the development of novel statistical methodology, and its applications to medical science. I collaborate with Cardiff University colleagues on societal and environmental determinants of mental and physical health, on child protection, and on clinical trials (in dialysis and delirium, for example). I maintain a particular focus on the analysis of longitudinal data, where study subjects may be observed on multiple occasions. Such analyses are especially interesting when the observed timings of measurements can provide further information about study subjects. For instance, if (when compared to healthier participants) those subjects who are most unwell tend to be observed less frequently, or to drop out earlier, careful analysis may be needed to avoid overrepresenting the healthy subjects when drawing conclusions from the study.

Publications

1. Ahmed, H.et al. 2017. Long-term antibiotics for prevention of recurrent urinary tract infection in older adults: systematic review and meta-analysis of randomised trials. BMJ Open 7(5), pp. e015233.

2. White, J.et al. 2016. Improving mental health through the regeneration of deprived neighbourhoods: a prospective controlled quasi-experimental study. The Lancet 388(Supp 2), pp. S110.

Dr Nicholas Francis

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+44 (0)29 2068 7133

I am an academic general practitioner and my research interests are around improving the management of problems commonly seen in general practice. My research has primarily focused around three main areas:

1. *Infections, complications of infections, and antimicrobial use and resistance.*
2. *Skin conditions, including eczema, acne, skin infections and molluscum contagiosum.*
3. *Respiratory conditions, including respiratory infections, COPD and asthma.*

Publications

1. Blair, P.et al. 2017. Feasibility cluster randomised controlled trial of a within-consultation intervention to reduce antibiotic prescribing for children presenting to primary care with acute respiratory tract infection and cough. BMJ Open 7(5), article number: e014506.

2. Ahmed, H.et al. 2017. Long-term antibiotics for prevention of recurrent urinary tract infection in older adults: systematic review and meta-analysis of randomised trials. BMJ Open 7(5), pp. e015233.

Professor Donald Fraser

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+44 (0)29 2074 8449

My research addresses mechanisms underlying injury and scarring in the kidney and peritoneum. These are examples of fibro-proliferative disorders, conditions that cause significant disease and suffering, and for which treatment options are very limited. Development and testing of novel therapies is restricted by incomplete knowledge of the biology of their progression, and lack of adequate biomarkers to serve as surrogate endpoints in clinical trials. I am Director of Wales Kidney Research Unit, a Biomedical Research Unit funded by Health and Care Research Wales to deliver an All-Wales strategy for the study of diagnosis, prevention, treatment and social context of kidney disease. See: **kidneyresearchunit.wales**
I am clinically active as a consultant nephrologist within Cardiff and Vale.

Publications

1. Dutzan et al, 2017. On-going Mechanical Damage from Mastication Drives Homeostatic Th17 Cell Responses at the Oral Barrier. Immunity 46(1), pp. 133-147.

2. Stacey, M.et al. 2017. The antiviral restriction factor IFN-induced transmembrane protein 3 prevents cytokine-driven CMV pathogenesis. Journal of Clinical Investigation 127(4), pp. 1463-1474.

Professor Awen Gallimore

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Our research investigates how the power of the immune system can be used to kill cancer, focusing on the use of model systems. Specifically, we aim to identify and remove bottlenecks to effective immune-mediated tumour destruction. These include the suppressive effects of regulatory T cells and features of the tumour microenvironment that prevent effective entry of immune cells. In model systems, we are using big data approaches to formulate a map of the cancer environment which takes into account 1) the cancer cells, 2)other cell types present which render cancer cells more or less aggressive, 3)cells of the immune system and 4)blood vessels. Our group works closely with the Godkin group to translate our findings to clinical trials.

Publications

1. Colbeck, E.et al. 2017. Treg depletion licenses T cell-driven HEV neogenesis and promotes tumor destruction. Cancer Immunology Research (In press).

2. Scurr, M.et al. 2017. MVA-5T4 immunotherapy (TroVax) and low-dose cyclophosphamide for advanced colorectal cancer (TaCTiCC): an open-label, randomized phase 1/2 clinical trial. JAMA Oncology, pp. e172579.

Professor Andrew Godkin

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+44 (0)29 2068 7003

Research from my group focuses on Phase I/II clinical studies in patients with cancer. Here, big data approaches are being taken to understand the antigenic landscape of colorectal cancers and also to understand why some patients, but not others, respond favourably to immunotherapy.

The results of our findings will be used to improve on current immunotherapies and to test these in patients as quickly as possible. Our research is jointly conducted with the Gallimore group, to take forward findings from basic science and animal models to inform our clinical approaches.

Publications

1. Jönsson, P.et al. 2016. Remarkably low affinity of CD4/peptide-major histocompatibility complex class II protein interactions. Proceedings of the National Academy of Sciences of the United States of America 113(20), pp. 5682-5687.

2. Scurr, M.et al. 2017. Effect of modified vaccinia Ankara–5T4 and low-dose cyclophosphamide on antitumor immunity in metastatic colorectal cancer A Randomized Clinical Trial. JAMA Oncology , pp. e172579.

Professor Peter Holmans

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+44 (0)29 2068 8427

I am Professor of Biostatistics & Genetic Epidemiology in the Biostatistics & Bioinformatics Unit (BBU) in the Wales College of Medicine, Cardiff University, UK, and direct the research in statistical genetics carried out by the BBU. I have a long-standing interest in the analysis of genome-wide linkage and association studies of complex genetic traits. I have also recently become involved in the analysis of gene expression data, in genome-wide linkage and association studies to find eQTLs relevant to disease. I have taken an active role in developing novel statistical methodology for linkage and association analysis of complex genetic traits, notably in the use of covariates in linkage and association studies, and the effects of genotyping error on genetic studies. Currently, I am particularly interested in the analysis of functional pathways in genome-wide association, CNV and gene expression data.

Publications

1. Allardyce, J.et al. 2017. Psychosis and the level of mood incongruence in Bipolar Disorder are related to genetic liability for Schizophrenia. JAMA - The Journal of the American Medical Association (In press)

2. Weiner, D.et al. 2017. Polygenic transmission disequilibrium confirms that common and rare variation act additively to create risk for autism spectrum disorders. Nature Genetics 49(7), pp. 978-985.

Dr Ian Humphreys

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+44 (0)29 2068 7012

Our laboratory uses in vivo models of viral infection in combination with clinical samples to help us understand the mechanisms that regulate antiviral immunity. A significant aspect of our research to date has investigated the immune mechanisms that allow virus persistence within mucosal tissues and identify how they suppress antiviral immune responsiveness. Our research also studies how early cytokine responses influence antiviral immune responses, and we also aim to identify what mechanisms regulate cytokine-driven viral pathogenesis.

Publications

1. Clement, M.et al. 2016. Cytomegalovirus-specific IL-10-producing CD4+ T cells are governed by type-I IFN-induced IL-27 and promote virus persistence. Plos Pathogens 12(12), article number: e1006050.

2. Sowerby, J.et al. 2017. NBEAL2 is required for neutrophil and NK cell function and pathogen defense. The Journal of Clinical Investigation 2017;127(9):3521–3526

Dr Gareth Jones

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I am an Arthritis Research UK Career Development Fellow interested in inflammation biology. My translational research aims to develop new treatments for inflammatory diseases and to identify biomarkers of disease activity with diagnostic and prognostic potential. This is underpinned by basic research into the mechanisms that drive immune-mediated tissue damage. Through combining investigations in experimental models of inflammation with clinical observations in patient samples, my laboratory uses global transcriptomic analysis of inflamed tissues and immune cells to identify (i) mechanisms that govern the development of ectopic lymphoid-like structures, (ii) molecular signatures that may aid the diagnosis or stratification of patients with this form of disease, and (iii) ways of manipulating the activity of ectopic lymphoid-like structures to treat patients.

Publications

1. Jones, G.et al. 2015. Interleukin-27 inhibits ectopic lymphoid-like structure development in early inflammatory arthritis. Journal of Experimental Medicine 212(11), pp. 1793-1802.

2. Fielding, C.et al. 2014. Interleukin-6 signaling drives fibrosis in unresolved inflammation. Immunity 40(1), pp. 40-50.

Professor Simon Jones

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+44 (0)29 2068 7325

My long term ambition is to improve the diagnosis, stratification and treatment of patients with inflammatory or autoimmune disease. Research considers the mode-of-action of anti-cytokine therapies, the identification of cytokine signatures that reflect patient outcome, and the development of novel cytokine-directed therapies. With a focus on cytokines that signal through the Jak-STAT pathway, I determine how cytokine networks deliver protective immunity and the transition to pathology and chronic disease progression. I am particularly focused on IL-6, and advise several pharmaceutical boards relating to the clinical development of IL-6 targeted therapies.

Publications

1. Dutzan et al, 2017. On-going Mechanical Damage from Mastication Drives Homeostatic Th17 Cell Responses at the Oral Barrier. Immunity 46(1), pp. 133-147.

2. Stacey, M.et al. 2017. The antiviral restriction factor IFN-induced transmembrane protein 3 prevents cytokine-driven CMV pathogenesis. Journal of Clinical Investigation 127(4), pp. 1463-1474.

Professor Sailesh Kotecha

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I lead the Paediatric Respiratory Group. Our interests lie in understanding the causes that lead to the development of chronic lung disease of prematurity (CLD) in preterm babies. In particular our research has focussed on delineating the processes that lead to the development and resolution of pulmonary inflammation including the recruitment and removal of neutrophils. We have been awarded a major EU FP7 grant to assess if azithromycin treatment of at-risk preterm infants can decrease the rates of CLD. Recent work has focussed on the role of microbes in the development of CLD. We are also linking these studies to longer term outcomes, in collaboration with Professor John Henderson at the ALSPAC, by investigating the long term cardiorespiratory outcomes including of the response of pulmonary arteries to hypoxia in children now aged 8 – 12 years of age but who were born prematurely and had lung disease as a consequence of preterm birth.

Publications

1.Stuchfield, P., Kotecha, S. and Doull, I. 2016. Antenatal Betamethasone for women at risk for late preterm delivery. New England Journal of Medicine 375(5), pp. 485-487.

2.Watkins, W. J., Kotecha, S. J. and Kotecha, S. 2016. All-cause mortality of low birthweight infants in infancy, childhood, and adolescence: population study of England and Wales. Public Library of Science Medicine 13(5), article number: e1002018.

Professor Julian R Marchesi

marchesijr@cardiff.ac.uk
+44 (0)29 2087 4188

The main focus of the research in my group is to determine the role gut microbes play in host development, health and disease. We have been using a variety of molecular approached to investigate both the culturable and unculturable fractions of the gut microbiota, including microbiomics (16S rRNA gene clone libraries, DGGE), functional metagenomics and metabonomics. Additionally we are also exploring the potential of the gut associated microbes to provide novel bioactive and biocatalytic agents which can be used to treat disorders of the gut and the host, for example, Clostridium difficile associated diseases, inflammatory bowel disease and colorectal cancer.

Publications

1. Kindinger, L.et al. 2016. Relationship between vaginal microbial dysbiosis, inflammation, and pregnancy outcomes in cervical cerclage. Science Translational Medicine 8(350), article number: 350ra102.

2. Marchesi, J.et al. 2015. The gut microbiota and host health: a new clinical frontier. Gut 65(2), pp. 330-339.

Professor Paul Martin

martinp@cardiff.ac.uk
(0) 117 331 2298

My lab works on wound healing and has established models to study repair and inflammation in mouse and zebrafish and Drosophila. The current twin foci of the lab are to investigate the genetics and cell biology of wound inflammation, in order to learn how best to modulate the inflammatory response to prevent fibrosis, and identifying parallels between wound and cancer-triggered inflammation. I was elected a Fellow of the Academy of Medical Sciences in 2011 and to membership of EMBO in 2012.

Publications

1. Eming, S. A., Wynn, T. A. and Martin, P. 2017. Inflammation and metabolism in tissue repair and regeneration. Science 356, pp. 1026-1030.

2. Wood, W. and Martin, P. 2017. Macrophage functions in tissue patterning and disease: new insights from the fly. Developmental Cell 40(3), pp. 221-233.

Professor James E Morgan

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+44 (0)29 2074 3222

My research is aimed at understanding the structural and cellular changes occurring at the level of the optic nerve in glaucoma using clinical and laboratory based techniques. Glaucoma is one of the most common causes of vision loss in the UK, affecting up to 2% of the population Classically, it is associated with increased intraocular pressure, which causes the death of retinal ganglion cells and results in a slow and, if untreated, progressive loss of vision. By the time a patient notices restriction of visual field, advanced damage may have occurred to the optic nerve that is usually irreversible. Treatment is aimed at the reduction of eye pressure (intraocular pressure) to prevent this vision loss. Usually, this is achieved using eye drops although, in some cases, surgery or even laser treatment may be required. One of the key problems is in identifying the disease in its earliest stages and in detecting the first signs of retinal (optic nerve) damage.

Publications

1. Rountree, L.et al. 2017. Quantifying the signal/noise ratio with perimetric stimuli optimised to probe changing spatial summation in glaucoma. Investigative Ophthalmology and Visual Science 58(8)

2. Morgan, J.et al. 2017. The optical detection of retinal ganglion cell damage. Eye 31(2), pp. 199-205.

Professor Paul Morgan

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+44 (0)29 2068 7096

I have studied the complement system for over thirty years, leading a research group working on basic complement biology and the relevance of this system to human disease. I have had a long-standing interest in the membrane attack complex (MAC), its effects on nucleated cells and how these respond to MAC attack. This work has led to several seminal discoveries, including the process of nucleated cell recovery from membrane attack and the mechanism by which CD59 regulates MAC formation. More recently, I have demonstrated that the MAC triggers inflammasome activation in diverse cells and contributed to new understanding of MAC structure, using this knowledge to predict function.

Publications

1. Ruseva, M.et al. 2015. An anticomplement agent that homes to the damaged brain and promotes recovery after traumatic brain injury in mice. Proceedings of the National Academy of Sciences 112(46), pp. 14319-14324.

2. Serna, M.et al. 2016. Structural basis of complement membrane attack complex formation. Nature Communications 7, article number: 10587.

Professor Bernhard Moser

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+44 (0)29 2068 7058

Research of Prof. Moser's group centers on human chemokines and their receptors expressed by immune cells. Initial work focused on the identification of chemokine receptors (starting with the IL-8 receptors, CXCR1 and CXCR2), followed by their biochemical and functional characterization. His group was first in identifying the human chemokine receptors CXCR3, CXCR4, CXCR5, CXCR6, CCR3 and CCR8. These findings enabled them to investigate (i) the role of CXCR4 in HIV infection, (ii), chemokine receptor antagonists, (iii) control of T cell migration (discovery of follicular B helper T (TFH) cells), and (iv) role of homeostatic chemokines in tissue homeostasis and immune defence. Finally, his group identified gdT-APCs, human gdT cells with professional antigen presentation functions, and this discovery is now being translated into the clinics (cellular immunotherapy of cancer patients).

Publications

1. Collins, P.et al. 2017. Epithelial chemokine CXCL14 synergizes with CXCL12 via allosteric modulation of CXCR4. The FASEB Journal 31(7), pp. 3084-3097.

2. Tyler, C.et al. 2017. Antigen-presenting human T-cells promote intestinal CD4+ T-cell expression of IL-22 and mucosal release of calprotectin. The Journal of Immunology 198(9), article number: 1700003.

Professor Valerie O'Donnell

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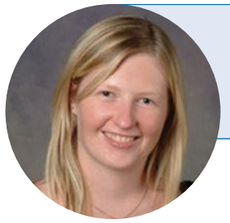
+44 (0)29 2068 7313

Our research uses mass spectrometry to discover and characterise new lipids (fats) made by circulating vascular cells that regulate immune defence and blood clotting. Recently we showed that human blood platelets generate a large number of oxidized phospholipids several of which help clotting factors in plasma work more effectively. We also uncovered a new role for phospholipases in providing energy to the cell and how lipids in different people respond individually to aspirin. Our current research is focused on understanding the role of new lipids in vascular inflammation including cardiovascular disease, dementia and wound healing. Some lipids we discovered are being developed as the basis of new treatments for bleeding excess.

Publications

1. Slatter, D.et al. 2016. Mapping the human platelet lipidome reveals cytosolic phospholipase A2 as a regulator of mitochondrial bioenergetics during activation. Cell Metabolism 23(5), pp. 930-944.

2. Uderhardt, S.et al. 2017. Enzymatic lipid oxidation by eosinophils propagates coagulation, hemostasis and thrombotic disease. Journal of Experimental Medicine 214(7), pp. 2121-2138.



Dr Selinda Orr

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I was awarded a Wellcome Trust VIP award in 2011 to relocate to Cardiff University to study the role of these C-type lectin-like receptors in host defence. I became a Wellcome Trust and The Royal Society Sir Henry Dale Fellowship in 2012 and continued my research into C-type lectin-like receptor signalling and immune responses. As part of the Systems Immunity Research Institute at Cardiff University I use a "big data" approach to determine how the immune response to various fungal pathogens differs. The goal of this research is to identify potential targets for developing novel immunotherapies that will be effective against various fungal pathogens.

Publications

1. Orr, S.et al. 2013. LAB/NTAL facilitates fungal/PAMP-induced IL-12 and IFN-γ production by repressing γcatenin activation in dendritic cells. PLoS Pathogens 9(5), article number: e1003357.

2. Patin, E.et al. 2016. IL-27 induced by Select Candida spp. via TLR7/NOD2 signaling and IFN-γproduction inhibits fungal clearance. The Journal of Immunology 197(1), pp. 208-221.

Professor Shantini Paranjothy

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My research is focused on perinatal and infant health inequalities. In 2008 I set up the Welsh Study of Mothers and Babies (WOMBS) to study the significance of ultrasound markers detected during pregnancy for the health of the baby (funded by MRC/WORD Health Research Partnership Award). The study recruited 30,000 mothers from all maternity units in Wales, with consent to follow up their children in future studies. I lead the research theme on vulnerable babies in the Wales Electronic Cohort for Children (WECC), the first complete population e-cohort in the UK of 800,000 children, aimed at investigating the widest possible range of social and environmental determinants of child health and social outcomes by exploiting the potential of routinely collected datasets.

Publications

1. Ahmed, H.et al. 2017. Long-term antibiotics for prevention of recurrent urinary tract infection in older adults: systematic review and meta-analysis of randomised trials. BMJ Open 7(5), pp. e015233.

2. Smits, S.et al. 2016. Development of a behaviour change intervention to encourage timely cancer symptom presentation among people living in deprived communities using the Behaviour Change Wheel. Annals of Behavioral Medicine 0883-6612 pp 1–15.

Professor Ole Petersen

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My research has uncovered how excessive intracellular release and subsequent entry of calcium ions cause activation of digestive enzymes inside pancreatic cells. This process leads to acute pancreatitis and an increased risk of pancreatic cancer. The pancreas secretes a cocktail of enzymes that normally become activated in the gut to help break down proteins, fat and carbohydrates. However, gallstones or an excessive intake of alcohol can lead to activation of these enzymes inside pancreatic cells, which then digest the pancreas itself. My research has identified a calcium-binding molecule inside pancreatic cells that, when boosted by introducing a calcium-like molecule to the pancreas, offers intrinsic protection against inappropriate enzyme activity.My research has also shown that specific pharmacological inhibition of entry pathways for calcium ions markedly reduces the excess enzyme activity and subsequent cell death and inflammation.

Publications

1. Petersen, O. H., Courjaret, R. and Machaca, K. 2017. Ca2+ tunnelling through the ER lumen as a mechanism for delivering Ca2+ entering via store-operated Ca2+ channels to specific target sites. The Journal of Physiology 595(10), pp. 2999-3014.

2. Gerasimenko, J.et al. 2013. Ca2+ release-activated Ca2+ channel blockade as a potential tool in antipancreatitis therapy. Proceedings of the National Academy of Sciences of the United States of America 110(32), pp. 13186-13191.

Professor David Price

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I graduated with double first class honours in medical sciences and pathology at the University of Cambridge and completed my clinical training at King's College Hospital London. I practised internal medicine, specializing in infectious and tropical diseases, before pursuing a DPhil in Immunology at the University of Oxford. After further academic clinical appointments, my research was conducted with fellowship support at the NIH Vaccine Research Center. I was appointed Chair of Infection and Immunity at Cardiff University School of Medicine in October 2007. My research program focuses on the development and application of advanced biotechnology to address fundamental issues in T-cell immunobiology with translational relevance.

Publications

1. Murrell, I.et al. 2017. The pentameric complex drives immunologically covert cell -cell transmission of wild-type human cytomegalovirus. Proceedings of the National Academy of Sciences of the United States of America 114(23), pp. 6104-6109., article number: 201704809.

2. Pymm, P.et al. 2017. MHC-I peptides get out of the groove and enable a novel mechanism of HIV-1 escape. Nature Structural & Molecular Biology 24(4), pp. 387-394.

Prof Jamie Rossjohn

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I research the molecular basis underpinning immunity. I have used structural biology to explain pre-T-cell receptor (TCR) self-association in T-cell development, and how the TCR specifically recognises polymorphic Human Leukocyte Antigen (HLA) molecules in the context of viral immunity and aberrant T-cell reactivity. I have unearthed structural mechanisms of HLA polymorphism impacting on drug and food hypersensitivities, as well as Natural Killer cell receptor recognition. I have pioneered our molecular understanding of lipid-based immunity by T cells, revealing that it can differ fundamentally from peptide-mediated adaptive immunity. Recently my team has provided a structural basis of how vitamin B metabolites can be presented and recognised by the immune system, revealing a new class of antigen.

Publications

1. Brennan, P.et al. 2017. Structural determination of lipid antigens captured at the CD1d-T-cell receptor interface. Proceedings of the National Academy of Sciences of the United States of America 114(31), pp. 8348-8353.

2. Ooi, J.et al. 2017. Dominant protection from HLA-linked autoimmunity by antigen-specific regulatory T cells. Nature 545(7653), pp. 243-247.

Professor Andrew Sewell

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T-cells orchestrate immunity and protect against pathogens and cellular malignancies by recognising short 'foreign' peptides bound to major histocompatibility complex (MHC) molecules. The antigen specificity of T-cells is conferred by the highly variable complementarity determining regions (CDRs) of the T cell receptor (TCR) that interact with the peptide-binding platform of the MHC. Thus the interaction between TCR and peptide-MHC (pMHC) is one of the most important interactions in biomedicine and is pivotal in many human diseases. There are two main subtypes of T-cell: cytotoxic T lymphocytes (CTL) which recognise peptides in the context of MHC class I (pMHCI) and helper T-cells (Th cells) which recognise peptides in the context of MHC class II (pMHCII). Our work focuses on human T-cell antigens and the receptors that recognize them.

Publications

1. Functional role of T-cell receptor nanoclusters in signal initiation and antigen discrimination, Proceedings of the National Academy of Sciences, 2016 Sep 13;113(37)

2. Cole et al (2016) Hotspot autoimmune T-cell receptor binding to pathogen and insulin peptides. Journal of Clinical Investigation J Clin Invest. 2016;126(6):2191–2204.

Dr Richard Stanton

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I am a senior lecturer within the 'Viral Immunology' group, and also the 'Cytomegalovirus and Adenovirus Virology' group. My research covers the biology, immunology and diagnosis of human cytomegalovirus (HCMV) as well as the development of recombinant adenovirus vectors. We are particularly interested in the application of proteomics to dissect the ways in which the virus modulates infected cells, how this manipulation of the cell leads to HCMV disease, and how it impacts on the ability of the virus to avoid being recognised and cleared by multiple arms of the immune system. In addition to informing on virus disease and potential routes to treatment, understanding the underlying basis for these phenomenon has dramatically improved our understanding of how the immune system functions on a molecular level.

Publications

1. Murrell, I.et al. 2017. The pentameric complex drives immunologically covert cell -cell transmission of wild-type human cytomegalovirus. Proceedings of the National Academy of Sciences of the United States of America 114(23), pp. 6104-6109., article number: 201704809.

2. Fielding, C.et al. 2017. Control of immune ligands by members of a cytomegalovirus gene expansion suppresses natural killer cell activation. eLife 6 2017; 6: e22206.

Dr Barbara Szomolay

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My research interest is in mathematical biology with applications in the areas of systems immunology and biofilm modelling. In collaboration with Warwick Systems Biology Centre (WSB), we developed the multi-species web-based tool PICPL, which predicts peptides derived from protein databases using combinatorial peptide libraries (CPLs). This has great significance in the design of T-cell vaccines and immunotherapies. Since screening of protein databases requires large scale computations, we implemented the CPL-based algorithm into a GPU-based framework. I am involved in projects which design web-based tools related to: cancer epitope-prediction with University of Helsinki, T-cell receptor (TCR) structure and CRISPR data with WSB. Ongoing is a modelling collaboration with experimentalists at Cardiff University in the area of inflammation and tumor heterogeneity. My future research will evolve around problems to better understand what drives biofilm formation, what factors control inflammation and how to design optimal therapies to control cancer and TCR signalling.

Publications

1. Szomolay B, Liu J, Brown PE, Miles JJ, Clement M, Llewellyn-Lacey S, Dolton G, Ekeruche-Makinde J, Lissina A, Schauenburg AJ, Sewell AK, Burrows SR, Roederer M, Price DA, Wooldridge L, van den Berg HA, Identification of human viral protein-derived ligands recognized by individual MHCI-restricted T-cell receptors (2016) Immunol Cell. Biol. 94:573-582.

2. Szomolay B, Cogan NG, Mechanical and chemical treatment in a biofilm model of two phenotypic resistance mechanisms (2015) Environ Microbiol. 17:1870-1883.

Professor Philip Taylor

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I study the innate immune system, in particular, macrophage and myeloid cell surface receptors involved in pathogen recognition and inflammation and the complement system. More recently, I have been engaged in fundamental questions of macrophage biology, relating to origins, development and renewal, and the transcriptional control of cellular activation. I have an additional interest in the development and validation of technologies that promote the 3Rs within a framework of scientific excellence. I have a strong foundation in the study of and development of experimental murine models of disease and immunity. Ultimately, my aim is to elucidate novel mechanisms to manipulate macrophage activity for beneficial outcome in disease.

Publications

1. Prince, L.et al. 2017. NR4A orphan nuclear receptor family members, NR4A2 and NR4A3, regulate neutrophil number and survival. Blood 3, article number: 770164.

2. Rosas, M.et al. 2014. The transcription factor Gata6 links tissue macrophage phenotype and proliferative renewal. Science 344(6184), pp. 645-648.

Professor Dave W Thomas

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I am a full-time Registrar/Lecturer in Oral & Maxillofacial Surgery working on a variety of projects in wound healing and nanomedicines. Clinical utility is key to our group's activities. We obtained numerous international and national patents in the UK, EU, US and Australia (including a family of patents on the OligoG alginates). We have, with our industrial collaborators Algipharma AS and the support of funding agencies (Norwegian Research Council, MRC, Wellcome, EU) polymer therapies at all stages of development, from in-vitro screening to Phase 2b multi-centre trials. This work has moved forward latterly in collaboration with researchers in Trondheim and Swansea University with the support of US Dept of Defence, the US Cystic Fibrosis Foundation and a €3M EU Eurostars Programme (www.eurostars-eureka.eu) to our collaborators.

Publications

1. Pritchard, M.et al. 2017. Alginate Oligosaccharides modify hyphal infiltration of Candida albicans in an in vitro model of invasive Human Candidosis. Journal of Applied Microbiology 123(3), pp. 625-636.

2. Pritchard, M.et al. 2017. Alginate oligosaccharides modify hyphal infiltration of Candida albicans in an in vitro model of invasive human candidosis. Journal of Applied Microbiology ; 123(3):625-636.

Professor Timothy Walsh

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I have been studying AMR mechanisms for over 20 years. I am director of BARNARDS, the lead Gates Foundation project on AMR, examining the burden of neonatal sepsis in Pakistan, India, Bangladesh, Rwanda, South Africa, Nigeria (Abuja and Kano) and Ethiopia. This project thus far has enrolled 26,000 mothers and analysed 2000 sepsis cases. I am also PI of DETER-XDR-CHINA, a study examining the spread and burden of AMR in public health sectors and hospitals in 11 provinces in China and hold an honorary chair at the Chinese Agricultural University as well as being an AMR advisor to the Chinese Government. I am also PI of CUT-SEC, a 'one-health" project in China and Thailand. Recently, I have been awarded an MRC grant to establish an AMR surveillance system throughout Vietnam in collaboration with Oxford University and the Wellcome Trust Center in Hanoi. I also have AMR studies in Karachi and Peshawar and collaborative studies with MSF in Jijawa (Nigeria), Niger (Clean Kids Study) and have facilitated the design and construction of the MSF Microbiology/AMR lab in Jordan/Syria.

Publications

1. Wang, Y.et al. 2017. Comprehensive resistome analysis reveals the prevalence of NDM and MCR-1 in Chinese poultry production. Nature Microbiology 2, article number: 16260

2. Walsh, T. R. and Wu, Y. 2016. China bans colistin as a feed additive for animals. The Lancet Infectious Diseases 16(10), pp. 1102-1103.

Dr Eddie Wang

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My research covers the biology and cellular immunology relating to Human Cytomegalovirus (HCMV) and the biological function of Death Receptor 3, a member of the TNFR superfamily involved in control of inflammatory and autoimmune disease. My work with the Viral Immunology research group attempts to understand the cellular mechanisms that regulate inflammatory versus inhibitory immune pathways. Our belief is that by understanding these complex mechanisms we will identify how, in cases of virus-induced inflammation, we can treat disease. Moreover, these studies will identify immune pathways that may be stimulated to enhance virus-induced immune responses, for example during vaccinations with viral-based vaccine vectors.

Publications

1. Jia, L.et al. 2016. A novel role for TL1A/DR3 in protection against intestinal injury and infection. Journal of Immunology 197(1), pp. 377-386.

2. Weekes, M.et al. 2014. Quantitative temporal viromics: An approach to investigate host-pathogen interaction. Cell 157(6), pp. 1460-1472.

Professor Ian Weeks

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I research and develop in vitro diagnostic methods based on ligand binding assays such as immunoassay, nucleic acid hybridisation assay and enzymological assay using high sensitivity end-points such as chemiluminescence. Particular emphasis is on development of simple, non-invasive methods as alternatives to invasive clinical test procedures. My current research involves the application of the principles outlined above for the development of diagnostic tests for biomarkers of inflammation and cancer. The overarching strategic model for the research is to target areas where there is an unmet clinical need or where synergy of technologies can enhance clinical diagnosis and management of underlying pathologies. Such a strategy is beneficial to patients and cost-effective for the healthcare provider.

Publications

1. Zhang, J.et al. 2017. Machine-learning algorithms define pathogen-specific local immune fingerprints in peritoneal dialysis patients with bacterial infections. Kidney International 92(1), pp. 179-191.

2. Browne, K.et al. 2011. Simultaneous quantification of multiple nucleic acid targets using chemiluminescent probes. Journal of the American Chemical Society 133(37), pp. 14637-14648.

Professor Susan Wong

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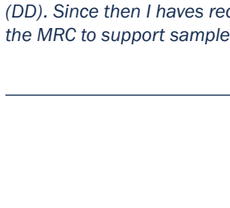
I research the aetiology and pathogenesis of type 1 diabetes. My primary focus is on aetiology and pathogenesis of type 1 diabetes and has encompassed T cell immunology, B cell immunology, regulatory T cells, innate immunity – NK cell and innate immune receptors. In recent years, our work has included studies in the role of the gut microbiome. As a member of the Type 1 Diabetes Consortium, I have also been involved in translational work in developing immunotherapy for type 1 diabetes and in early phase 1a clinical trials.

Publications

1. Alhadj Ali, M.et al. 2017. Metabolic and immune effects of immunotherapy with proinsulin peptide in human new-onset type 1 diabetes. Science Translational Medicine 9(402), article number: eaaf7779.

2. Wen, L. and Wong, F. 2017. Dietary short chain fatty acids protect against type 1 diabetes. Nature Immunology 18(5), pp. 484-486.



Professor Anwen Williams

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My research focuses on the identification of cell-associated signals that underpin the development of disease in the skeletal system (e.g., cartilage and bone) and the circulatory system (e.g, arteries and heart). Arthritis is a common condition that causes pain and inflammation in a joint. In the UK, around 10 million people have arthritis and 7 million people fight their daily battles with heart and circulatory disease. I lead innovative projects aimed at the development of medicines that manipulate the inflammatory response and the processes that orchestrate tissue destruction so that an environment for repair and regeneration is restored and maintained. In leading the pre-clinical 'discovery' platform in the section of Rheumatology I develop pharmacological targets and innovative research tools for translation to the clinic.

Publications

1. Singh, R.et al. 2017. Death receptor 3 regulates distinct pathological attributes of acute versus chronic murine allergic lung inflammation. Cellular Immunology (In press)

2. Sime, K., Choy, E. and Williams, A. 2017. Alterations to adipose tissue morphology during inflammatory arthritis is indicative of vasculopathology in DBA/1 mice. Adipocyte 6(2), pp. 87-101.

Professor Julie Williams

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My research focuses upon identifying and understanding genes which increase the risk of developing complex psychological and neurodegenerative disorders. These include Alzheimer's disease, developmental dyslexia and schizophrenia. I have contributed to or led a number of successful and long standing consortia, and have attracted research funding amounting to several millions of pounds from major grant funders. I have also published widely in top scientific Journals (e.g., Science, Nature Genetics, Lancet). Recently, I published evidence for the first new susceptibility genes for Alzheimer's disease for 17 years (Harold et. al. (2009), Nat. Genet., 41(10): 1088-93), identifying two new susceptibility genes for AD; CLU and PICALM from a study involving over 20,000 subjects from Europe and the USA . Over ten years ago I began research into the molecular genetics of developmental dyslexia (DD). Since then I haves received funding from the Wellcome Trust, The Health Foundation and the MRC to support sample collection and genotyping.

Publications

1. Sims, R.et al. 2017. Rare coding variants in PLCG2, ABI3 and TREM2 implicate microglial-mediated innate immunity in Alzheimer's disease. Nature Genetics ;49(9):1373-1384

2. Desikan, R.et al. 2015. Polygenic overlap between C-reactive protein, plasma lipids, and Alzheimer Disease. Circulation 131(23), pp. 2061-2069.

Professor Gavin Wilkinson

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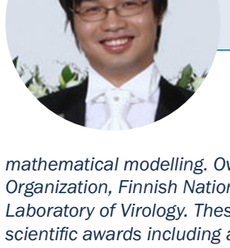
I am a molecular virologist working on CMV, an extremely common herpesvirus that causes severe congenital infections and induces the strongest immune responses of any pathogen. CMV is the most complex human virus. We are exploiting cutting-edge genomic and proteomic technology to build a comprehensive picture of how CMV overcomes our immune response to causes disease. This knowledge is being used to develop novel therapies.

Publications

1. Hsu, J.et al. 2015. Plasma membrane profiling defines an expanded class of cell surface proteins selectively targeted for degradation by HCMV US2 in cooperation with UL141. PLoS Pathogens 11(4), article number: e1004811.

2. Murrell, I.et al. 2017. The pentameric complex drives immunologically covert cell -cell transmission of wild-type human cytomegalovirus. Proceedings of the National Academy of Sciences of the United States of America 114(23), pp. 6104-6109., article number: 201704809.



Dr You Zhou

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My long-term ambition is to understand how inflammatory processes affect the onset, progression and severity of complex diseases (e.g, fatty liver diseases, diabetes, cancer) through the application of computational systems approaches and mathematical modelling. Over the course of my career, I have worked for, the World Health Organization, Finnish National Institute for Health and Welfare, and the Chinese State Key Laboratory of Virology. These positions have lead to over 40 research articles, and several scientific awards including a Young Scientist Award from the Nordic Lipid Forum. In addition, I am extremely interested in employing my computational pipeline in profiling genetic/genomic/ lipidomic landscape underlying immune homeostasis across complex diseases. This is illustrated by a number of my recent publications, in leading international scientific journals – e.g., Journal of Hepatology (IF= 12.49); Clinical Gastroenterology and Hepatology (IF=7.398); Diabetologia (IF= 6.08). Several of these studies have been highlighted by the Faculty of 1000, and received important media coverage.

Publications

1. Luukkonen, P.et al. 2016. The MBOAT7 variant rs641738 alters hepatic phosphatidylinositols and increases severity of non-alcoholic fatty liver disease in humans. Journal of Hepatology 65(6), pp. 1263-1265.

2. Luukkonen, P.et al. 2017. Impaired hepatic lipid synthesis from polyunsaturated fatty acids in TM6SF2 E167K variant carriers with NAFLD. Journal of Hepatology 67(1), pp. 128-136

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