Executive Summary: The overarching objective of our Group is to improve clinical outcomes for those with, and at risk of developing, rheumatoid arthritis (RA), Sjogren’s syndrome (SS) and systemic lupus erythematosus (SLE) by developing diagnostic tests, drugs, cell based therapies and lifestyle assessments to predict, prevent and reverse disease pathology. A unique feature of our translational research is that we have pioneered a first in class, “process-driven pathway-focused” approach to the biology of inflammatory arthritis. A particular strength of research in Birmingham is our therapeutic targeting of the tissue microenvironment and our skills in comparing and contrasting the biological processes underpinning the development, maintenance and resolution of inflammation. This process driven approach has allowed us to develop a unique approach to inflammation research in which shared biological mechanisms are compared across a number of traditionally independent organ based medical disciplines to develop biologically meaningful and therapeutically tractable process-driven links to other disease areas in Inflammation biology. Furthermore, it breaks down traditional bench to bedside, bedside to bedside and primary care-secondary care silos that have prevented a truly integrated, multidisciplinary, patient centred approach to treating chronic inflammation.
**The Rheumatology Research Group (RRG)** is based in the Institute of Inflammation and Ageing (IIA) in the College of Medical and Dental Sciences. The Institute has major research themes in Inflammation, Ageing, Trauma, Regeneration and Repair. These research themes are linked to the clinical disciplines of Gerontology, Critical Care, Nephrology, Rheumatology, Ophthalmology, Gastroenterology and Respiratory Medicine. Academic Rheumatology in Birmingham is therefore ideally placed to act as a focus for translational clinical research in inflammation, where a clear understanding of how immune cells behave in inflamed microenvironments is likely to be of critical importance for future experimental medicine studies.

The RRG has focused on collaborative research along lines of common interest. Such collaborations include those with basic scientists, those running clinical studies and researchers using qualitative methods in a community setting. This has provided a clear focus on important biological and clinical questions that cross traditional disciplines and often require long-term commitment and investment. The key objective of our research is to improve clinical outcomes for those with, and at risk of developing, rheumatoid arthritis, Sjogren’s syndrome and SLE. Our research is underpinned by well characterised cohorts of patients. Our multidisciplinary team of academic and clinical rheumatologists, general practitioners, biological and social scientists, allied health professionals and patient representatives works in an integrated way to develop and deliver our research objectives. Our major focus is on inflammatory arthritis and in particular the pathobiology and comorbidity associated with rheumatoid arthritis and Sjogren’s syndrome as well as the epidemiology, clinical management and outcome of SLE. A particular strength of research in Birmingham is exploring the role of the tissue microenvironment in shaping immune and inflammatory responses.

We run a substantial research group comprising over 50 research scientists, clinicians and allied health professionals. Our basic science research focuses on why inflammation persists. Our model has become an internationally accepted paradigm; a concept we have termed “process-driven pathway-focused pathology”. Our studies have proved critically important in driving the clinical models that we investigate. While this has historically been focussed on established rheumatoid arthritis, vasculitis and SLE, we have developed research programmes in very early rheumatoid arthritis, the pathology of Sjogren’s syndrome and the relationship between periodontal disease and inflammatory arthritis. This work also provides an axis for collaborative interaction between basic scientists in the adjacent Institute of Biomedical Research in Birmingham as well as clinical colleagues including those in primary care.

We are one of the three new themes in the Birmingham NIHR BRC in Inflammation awarded in 2017. In addition we are one of nine Centres in the NIHR TRC in Joint and Inflammatory Disease and one of six ARUK Experimental Arthritis Treatment Centres. We are also members of the MRC-ABPI initiative (RA-MAP), the MRC Stratified Medicine consortia (MATURA (RA) and MASTERPLANS (SLE) as well as the recently funded ARUK microbiome project in collaboration with colleagues in Oxford. We are one of two UK centres selected to take part in the NIH funded Accelerating Medicines Partnership (AMP) [https://www.nih.gov/research-training/accelerating-medicines-partnership-amp](https://www.nih.gov/research-training/accelerating-medicines-partnership-amp) based on our ability to perform and evaluate synovial and salivary gland biopsy. All of this places us in an ideal position to translate our laboratory findings into clinically relevant treatments. We are also one of three Universities that form the ARUK Centre of Excellence in the Pathogenesis of RA ([http://www.race-ubn.org/](http://www.race-ubn.org/)). We are partners in the IMI RT-Cure, exploring how therapeutic targeting of tollerance affects the progression of RA as well as the EU funded eSSential consortium in Sjorgens Syndrome.

In 2017 the Kennedy Trust for Rheumatology Research (KTRR) funded a new Arthritis Therapy Acceleration Programme (A-TAP); a partnership between the Institute of Inflammation and Ageing (Birmingham) and the Kennedy Institute for Rheumatology Research (Oxford). The primary purpose of the A-TAP is to provide a translational vehicle to “pull though” basic science discoveries into the clinic in early proof of principle experimental medicine studies along the M40 corridor, by providing the missing link that will ensure that world class basic science observations are “accelerated” into early
phase experimental therapy for patients by providing the “infrastructure of people “ to allow this to happen. The A-TAP will use pathology based outcome measures in basket trials in Immune Mediated Inflammatory Diseases to determine therapeutic utility and aid tissue based biomarker discovery. The results will help treat the cause of disease not just their symptoms and ensure expensive treatments are targeted to those most likely to benefit.

Using Rheumatoid Arthritis, Sjogren’s Syndrome and SLE as exemplars our aim is to:

1. To **predict** the development disease using new diagnostic tests and psychological assessments
2. To **prevent** the progression of disease using novel cell based and microbiome based therapies
3. To identify and target mechanisms driving the **progression** of disease and harness the therapeutic potential of mechanisms involved in regulating the **resolution** of disease to **prevent joint damage**
Kennedy Professor of Translational Rheumatology  
Director, Birmingham NIHR Clinical Research Facility

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Career History
2017  Kennedy Professor of Translational Rheumatology  
2002  Arthritis Research UK Professor of Rheumatology, Birmingham  
2001  MRC Senior Clinical Fellow, Birmingham  
1996  Wellcome Trust Clinician Scientist, Honorary Clinical Lecturer, Birmingham  
1993  Wellcome Trust Research Training Fellow, Oxford. (Dr. David Simmons)  
1993  Senior House Officer, Central Oxford Hospitals, Oxford  
1992  Senior House Officer in Cardiology, Royal Brompton National Heart and Lung Hospital. (Prof P Poole-Wilson, Dr K Fox, Dr J Somerville)  
1991  Senior House Officer in Rheumatology and Neurology, Hammersmith Hospital, London (Prof M. Walport, Prof. R. Frackowiak)  
1990  House Officer posts Royal Free Hospital, London

Research Summary

A characteristic feature of chronic inflammatory reactions is their persistence and predilection for certain sites. Our group investigates the role that tissue resident stromal cells (fibroblasts) play in determining both the switch to persistence as well as the site at which inflammation occurs. In chronic inflammation the resolution phase is prolonged and disordered leading to the persistent accumulation of an inflammatory infiltrate. Our work has allowed us to propose that a "stromal area post code", predominantly defined by fibroblasts, exists within tissues. Our hypothesis predicts that components of this stromal area post code become disordered during inflammation, leading to the accumulation of lymphocytes in structures that resemble lymphoid tissues.

We have proposed that inflammation is not generic but contextual and therefore differences in the response of different inflammatory diseases to therapy are likely to be due to intrinsic differences in the behavior of stromal cells within different environments. Ignoring the contribution of stromal cells to the pathogenesis of chronic inflammatory disease may account for the failure of current therapies to affect a permanent cure. We suggest that stromal cells in general and fibroblasts in particular offer a new family of organ specific targets to treat chronic immune mediated inflammatory diseases such as rheumatoid arthritis. To test these ideas we have established an Arthritis-Therapy Acceleration Programme between the Universities of Birmingham and Oxford and seven NHS trusts funded by the Kennedy Trust for Rheumatology Research.
Emeritus Professor of Rheumatology

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Career History

2018  Emeritus Professor, University of Birmingham
2007  Professor in Rheumatology, University of Birmingham; Consultant Rheumatologist at Sandwell & West Birmingham Hospitals NHS Trust & University Hospital Birmingham Foundation NHS Trust
          Chair of the NIHR/UKCRN National Specialty Group for Immunology and Inflammation
2003  Reader in Rheumatology, University of Birmingham and Consultant Rheumatologist at Sandwell and West Birmingham Hospitals NHS Trust and University Hospital Birmingham Foundation NHS Trust; NHS Trust
1996  Senior Lecturer and Consultant Rheumatologist, University of Birmingham with clinical work based at City Hospital NHS Trust and University Hospital Birmingham NHS Trust.
1989  Clinical Lecturer and Honorary Senior Registrar in Rheumatology, University of Birmingham
1987  Harkness Research Fellow, Rheumatology & Immunology, University of California, San Francisco
1985  Registrar, General and Thoracic Medicine and Gastroenterology, Frenchay Hospital, Bristol
1983  Senior House Officer: Neurology, Cardiology, General Medicine and Diabetes, Frenchay
1982  Senior House Officer rotation: General Medicine with Chest Medicine, Gastroenterology and Rheumatology, Bevendean Hospital and Royal Sussex County Hospital, Brighton
1981  House Officer posts in Brighton General Hospital & Royal Sussex County Hospital, Brighton

Research Summary

My research programme focuses on systemic lupus erythematosus (SLE). I am a member of the British Isles Lupus Assessment Group (BILAG), the Systemic Lupus International Collaborating Clinics (SLICC), and Co-Chair of the European League Against Rheumatism (EULAR) Task Force for Systemic Lupus Erythematosus and have been a member of several American College of Rheumatology and Lupus Foundation of America committees for lupus research. In 2013 I gave the prestigious Heberden Round at the British Society for Rheumatology (BSR) Annual Meeting. I have been an active member of the BSR Guidelines Working Group on Prescribing Drugs in Pregnancy and Breast-feeding and has led the BSR Guidelines Working Group on the Management of SLE. I am also a consultant to the Centre for Disease Control on epidemiological studies of lupus. Much of my work has focused on disease assessment for clinical trials and outcome studies, particularly the development of the BILAG disease activity index and the epidemiology of lupus. I have been involved in the development of the SLICC/ACR damage index and in the assessment of quality of life in lupus patients using the SF-36 and the Lupus QoL surveys. I have a longstanding interest in improving the treatment of SLE and have been involved in organising five investigator-led clinical trials including the current RituXilup trial and Beat-Lupus trial (funded by ARUK). I led the initiative producing EULAR points to consider for conducting clinical trials in SLE and advise the pharmaceutical industry on organising and analysing clinical trials. I have been a consultant to the ESF Research Networking Programme: The Identification of Novel Genes and Biomarkers for Systemic Lupus Erythematosus (BIOLUPUS). I am interested in both clinical and laboratory markers of disease flare, the genetic susceptibility to lupus, predictors of response to treatment, and the health of children born to mothers with lupus. She is part of the MRC funded Strategic Medicine Consortium “Masterplans”.

RRG Brochure November 2017
Arthritis Research UK Professor of Rheumatology  
Director of R&D at Sandwell and West Birmingham NHS Trust

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Career History
2017 ARUK Professor of Rheumatology  
2013 Professor of Clinical Rheumatology (University of Birmingham) and Honorary Consultant  
    Rheumatologist (Sandwell and West Birmingham Hospitals NHS Trust)  
2011 Reader in Clinical Rheumatology (University of Birmingham) and Honorary Consultant  
    Rheumatologist (Sandwell and West Birmingham Hospitals NHS Trust)  
2004 Senior Lecturer in Rheumatology (University of Birmingham) and Honorary Consultant  
    Rheumatologist (Sandwell and West Birmingham Hospitals NHS Trust)  
2003 Rheumatology Specialist Registrar (City Hospital, Birmingham)  
2000 Arthritis Research UK Clinical Research Fellow (University of Birmingham)  
1997 Rheumatology Specialist Registrar (Royal National Hospital for Rheumatic Diseases, Bath)  
1996 Clinical Research Fellow (Royal National Hospital for Rheumatic Diseases, Bath)  
1994 Senior House Office (Queen’s Medical Centre, Nottingham)  
1993 House Officer (John Radcliffe Hospital, Oxford; Royal United Hospital, Bath)

Research Summary
Rheumatoid arthritis (RA) is common and causes significant disability and reduced life expectancy. Early treatment is associated with improved outcomes. To optimise early treatment, four areas of unmet need have to be addressed and my research focuses on these:

1. To understand early disease mechanisms to allow the development of targeted therapies.  
2. To identify biomarkers of outcome in patients with early inflammatory arthritis and inflammatory  
    arthralgia to allow the development of personalised therapy.  
3. To understand the reasons for delay in assessment of patients with a new onset of inflammatory  
    joint symptoms to reduce delay and allow earlier therapy.  
4. To understand perspectives of patients and members of the public about RA and the concept  
    of ‘risk’ in relation to RA to facilitate informed engagement with predictive and early treatment  
    strategies.

In addition, I am a very strong advocate for Patient and Public involvement (PPI) in research. PPI manifests as research that is carried out ‘with’ or ‘by’ patients and members of the public rather than ‘to’, ‘about’ or ‘for’ them. We have worked actively and successfully with patients and members of the public across a wide range of projects.  

PPI has included working with researchers to identify research priorities, offering advice as members of a project steering group, commenting on and developing research materials and undertaking interviews with research participants. Such involvement has ensured that the resulting research is grounded in the reality of the patient experience, enhancing relevance and deliverability and facilitating dissemination and adoption.
Professor of Inflammation Biology

Professor Andy R Clark
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Career History
2012  Professor of Inflammation Biology, Birmingham University
2011  Reader in Cell Signalling, Oxford University
2009  Reader in Cell Signalling, Imperial College London
2002  Senior Lecturer, Imperial College London
1996  Lecturer, Imperial College London
1993  Postdoctoral fellow, Cancer Research UK
1992  Postdoctoral fellow, Birmingham University (holder of Redcliffe-Maud fellowship from Diabetes UK)

Research Summary
Inflammation is essential for our healthy response to injury and our protection against pathogens. Yet it can also be extremely damaging if it is not tightly controlled. Several negative feedback mechanisms have evolved to prevent unprovoked inflammation and assist in the resolution of inflammation. For example, inflammation induces the expression of endogenous anti-inflammatory molecules including glucocorticoids, which promote resolution. Rheumatoid arthritis and other chronic inflammatory diseases can develop only if such natural barriers to inflammation fail or are subverted. Negative regulation of inflammation is key to understanding why rheumatoid arthritis develops and why it manifests itself in joints. Understanding this process will also suggest new methods to treat or prevent chronic inflammatory diseases. My group focuses on negative feedback mechanisms that constrain the inflammatory responses of macrophages and synovial fibroblasts, two cell types that contribute importantly to the pathogenesis of rheumatoid arthritis. We have characterised a molecular switch that controls the transition from the activation phase to the resolution phase of macrophage inflammatory responses by regulating the stability of pro-inflammatory mRNAs. By manipulating this switch genetically or pharmacologically, arthritis could be prevented or treated in mice. We have also found that endogenous anti-inflammatory molecules like glucocorticoids work by reinforcing negative feedback mechanisms. The challenges now are to explain how and why these inflammation-limiting mechanisms fail in rheumatoid arthritis, and then to exploit this knowledge to devise new anti-inflammatory treatments.
Professor of Inflammation Research

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Career History

2018 Promoted to Professor in Translational Inflammation Research
2016 Promoted to Reader in Translational Inflammation Research
2010 Arthritis Research UK Career Progression Fellow
2007 Promoted to Senior Research Fellow
2004 ARC Non-clinical Career Development Fellow, University of Birmingham
2003 Member of the Higher Education Academy
1994 Postdoctoral Research Fellow with Dr. Michael Salmon und Dr. Janet Lord in the MRC Birmingham Centre for Immune Regulation.
1994 Awarded Dr. rer nat (equivalent to PhD in Natural Sciences) by the Christian Albrechts Universität, Kiel “summa cum laude” for the thesis: Investigation of the expression of CD26 in granulomatous tissue and normal peripheral blood.
1990 PhD student at the Forschungszentrum Borstel. Dept of Immunology and Cellular Biology, with Prof J Gerdes.
1989 Degree in Pharmacy, Christian Albrechts Universität, Kiel, Germany

Research Summary

The main research interest of my team focuses on disease mechanisms in chronic inflammation. We use a range of methods of both molecular and cellular immunology to unravel the contribution of immune cell populations to the network of cells and factors that cause joint destruction in patients with rheumatoid arthritis. Our long term aim is to identify new therapeutic targets and strategies for improved treatment of rheumatoid arthritis.

In a study of immune cell populations infiltrating the RA joint, we have identified a novel pro-inflammatory B cell population at the site of inflammation in the rheumatoid joint. Our findings suggest a pro-inflammatory and destructive role for this cell type via production of a range of cytokines including RANKL. This finding sheds a new light at the way we perceive the role of B cells in RA and is a plausible explanation for the success of B cell depleting therapy in RA. We ran a series of extensive phenotyping experiments to characterize the RANKL expressing B cell population and found that they can be specifically identified by their expression of the membrane protein FcRL4. Investigation of the origin, differentiation and role in inflammation these cells play is now a major focus of our work.

Translation of these findings as a therapeutic target is now supported by major funding from the MRC DPFS funding scheme. Furthermore, I have a long term interest in the contribution neutrophils make to joint inflammation. In the context of this work we have shown that neutrophils contribute to the production of citrullinated autoantigens in the inflamed joints, thus continuously fuelling autoimmunity..
Honorary Professor of Rheumatology

Professor Simon Bowman

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Career History

2017  Consultant Rheumatologist Milton Keynes Hospital
2015  Honorary Professor (University of Birmingham)
2012  Honorary Reader (University of Birmingham
2003  Consultant Rheumatologist, University Hospitals Birmingham & Honorary
       Senior Lecturer, University of Birmingham
1996  Senior Lecturer & MRC Clinician Scientist (1996-2000), University of
       Birmingham & Honorary Consultant Rheumatologist, Birmingham Heartlands
       Hospital
1994  Senior Registrar in Rheumatology, Nuffield Orthopaedic Centre, Oxford
1991  MRC Training Fellowship (PhD Studentship). UMDS Guy’s Hospital, London
1989  Registrar posts, UCLH/Middlesex rotation, London
1987  SHO posts, Royal Brompton Hospital & Guy’s/Lewisham Hospitals
1986  House officer posts, UCLH/Middlesex & Edgware General Hospitals

Research Summary

I have a longstanding interest in clinical research in primary Sjögren’s syndrome (pSS) in collaboration
with colleagues from oral medicine and ophthalmology. I have led a number of projects to develop and
validate Sjögren’s-specific symptom questionnaires and outcome tools. This has led to further
collaborations with Professor Xavier Mariette (France) and Dr Claudio Vitali (Italy) and other European
colleagues sponsored by the European League against Rheumatism (EULAR) to develop international
consensus outcome measures for use in clinical trials and longitudinal observational studies in
Sjögren’s syndrome (ESSDAI & ESSPRI). More recently, I have been co-applicant with Professor Fai
Ng (Newcastle) on the MRC funded UK Registry of patients with Primary Sjögren’s syndrome
(UKPSSR). I have been chief investigator in collaboration with Professor Paul Emery (Leeds), Professor
Costantino Pitzalis (London), the Leeds Clinical Trials Unit, Roche Pharmaceuticals and other
colleagues of the TRACTISS study of rituximab in patients with pSS funded by Arthritis Research UK.
In collaboration with Professor Chris Buckley, Dr Francesca Barone, Dr Ben Fisher and other
colleagues at the University of Birmingham we have set up a programme of basic science research in
pSS and the clinical OASIS cohort and are engaged in a number of clinical trials of novel agents.
Reader in Experimental Rheumatology

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Career History

2016 Reader in Rheumatology, Institute of Inflammation and Ageing, University of Birmingham
1996 Senior Lecturer in Rheumatology, School of Immunity & Infection, University of Birmingham.
1985 Lecturer in Rheumatology, School of Immunity & Infection, University of Birmingham.
1981 MRC Research Fellow, The Liver Unit, King’s College Hospital Medical School, University of London.
1978 NIH Research Fellow, Department of Biophysics and Physiology, and Liver Research Centre, Albert Einstein College of Medicine, New York.
1974 Research Assistant, Department of Clinical Haematology, University College Hospital Medical School, University of London.

Research Summary

We are investigating the synergism between inflammatory cytokines and reactive oxygen species in promoting this depression. Redox damage may underlie the severe depression in the phosphatase activity of the leukocyte common antigen CD45, which we have identified not only in arthritis patients but in the healthy elderly. We are also investigating the function of phosphatase Lyp (PTPN22) variants of which are strongly associated with a number of autoimmune diseases including rheumatoid arthritis. This contributes to the decline in immune function in ageing, and may be a predisposing factor in the initiation of chronic disease. We have developed new technologies to be able to assess signalling in arrayed single cells in highly heterogeneous cell populations. We have extended this work to studies on endothelial cells in an attempt to explain the increase in cardiovascular disease associated with rheumatoid arthritis. We have shown that the earliest stages of the atherosclerotic process, the induction of endothelial cell dysfunction, can be mediated by TNF-induced sphingomyelinase. This dysfunction in lymphocyte and endothelial function may result from a complex interaction between factors including genes, lifestyle, nutrition and infection. Metabolomics is a powerful new approach to the analysis of the overall metabolic activity of an organism, which may allow generation of a composite picture resulting from these many factors. We have established NMR-based Metabolomics to study human inflammatory disease and ageing and this has proven to be a powerful tool for stratifying patients with complex diseases such as Uveitis and Rheumatoid Arthritis. Further development will allow identification of novel markers of disease to provide new insights into the pathological processes involved. Auto-antibodies and antibodies to new biological therapeutics are a common feature of autoimmune diseases. We are using Surface Plasmon Resonance (BIAcore) to characterise these antibodies and to assess important molecular interactions regulating inflammatory disease in the liver and the vascular system.
Reader in Translational Rheumatology
Arthritis Research UK Senior Fellow

Dr Francesca Barone
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Career History
2017  Reader in Translational Rheumatology
2016  Senior Lecturer in Rheumatology
2010  Wellcome Trust Clinician Scientist Fellowship
2009  Postdoctoral Research Fellow with Dr Jo Spencer, Immunobiology Department, Kings College London
2008  PhD University of London
2007  Specialization in Rheumatology – University of Rome – Sapienza
2001  Degree in Medicine – University of Rome – Sapienza

Research Summary

My group research is focused on understanding the mechanisms regulating the complex interaction between stromal cells and leukocytes in different phases of the inflammatory process and its resolution.

1. Leukocyte stromal cell interaction in ectopic lymphoneogenesis:
   During organogenesis, the interaction between leukocytes and stroma is critical for stromal cell activation and formation of lymphoid organs. In target organs of autoimmune diseases the inflammatory cells infiltrating the issue organise themselves in structures that closely resemble secondary lymphoid organs in a process called ectopic lymphoneogenesis. While physiologic lymphoneogenesis has been largely defined in its mechanisms, little is known about the cells/signals that regulate the formation of ectopic lymphoid structures during disease. My research aims to understand using in vivo models of inflammation the mechanisms regulating the formation of ectopic lymphoid structures with particular emphasis on the signals that regulates the activation of stromal cells to the acquisition of a lymphoid-like phenotype.

2. Role of ectopic lymphoneogenesis in inflammation:
   The role of ectopic lymphoid structures in the dynamic of the inflammatory process is not clear. Some evidence even suggests that ectopic germinal centres might support lymphoma development during chronic inflammatory processes. I aim to understand the functional role of ectopic lymphoneogenesis in the balance between persistence and resolution of the inflammatory process and the role that stromal cells, including blood endothelial and lymphatic cells play in this process.
Reader in Rheumatology

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Career History

2018  Reader in Translational Rheumatology
2010  HEFCE Senior Lecturer in Rheumatology, University of Birmingham; Honorary Consultant Rheumatologist University Hospitals Birmingham NHS Foundation Trust
2009  Arthritis Research UK Clinician Scientist
2006  Clinical Lecturer in Rheumatology
2002  MRC Clinical Research Training Fellow, University of Birmingham
1999  Rheumatology Specialist Registrar (Walsgrave Hospital Coventry, Worcester Royal Infirmary, Birmingham Heartlands & Solihull Hospitals, City Hospital)
1997  Medical SHO rotation, University Hospital Birmingham
1996  Resident posts in ED and ITU, Sir Charles Gairdner Hospital, Perth, Western Australia
1995  House Officer, City Hospital and University Hospital, Birmingham

Research Summary

Rheumatoid arthritis (RA) is a common chronic disease which leads to significant disability and reduced life expectancy. Biological treatments have had significant impact, but new techniques are required to understand disease mechanisms and develop novel clinical trial outcomes. My work combines ultrasound imaging with novel analyses of synovial tissue to address these needs:

1. **The identification of tissue biomarkers of outcome in the early stages of joint inflammation.** Alongside collaborators in Italy and London, I pioneered the minimally invasive ultrasound-guided synovial biopsy technique used to provide a tissue resource to identify tissue biomarkers of outcome. I explore development of tissue biomarkers of outcome and related therapeutic targets through internal (Clark group, Schell-Toellner group) and external (Ospelt/Gay Group, Brenner group (Harvard), NIH AMP group) collaborations. This work also aims to improve understanding of pathogenic mechanisms operating in the stromal cells of patients with early disease, in order to allow the development of novel targeted therapies (with McGettrick group and Novartis).

2. **Ultrasound imaging as a biomarker and clinical trial outcome.** Ultrasound imaging is a key technology enabling the collection of tissue and cellular resources, but has an increasing role in objective measurement of synovial inflammation. I am a member of the global OMERACT clinical trials outcome group; I lead the OMERACT “minimal disease” multicentre international study, and run ultrasound imaging in the APIPPRA international multicentre trial intervening with biologic therapy in patients with inflammatory arthralgia at high risk of RA.

3. **The delivery of early phase trials in RA focused on stromal cell biopathology.** This work includes development of mesenchymal stromal cellular therapies in arthritis (Orbsen therapeutics) and repurposing of therapies targeting stromal cells in cancer for use in rheumatoid arthritis (MRC DPFS funded TRAFIC trial with AR-UK RACE Centres).

My research is linked nationally to the Arthritis Research UK Experimental Arthritis Treatment Centre network and the RACE Arthritis Research UK Centre of Excellence in RA pathology (Birmingham,
Newcastle and Glasgow). Internationally I have a funded role and collaborations within the National Institutes of Health: Accelerating Medicines Partnership.

**Senior Clinical Lecturer in Rheumatology**

Dr Benjamin Fisher

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**Career History**

2012 Senior Lecturer in Experimental Translational Inflammation, University of Birmingham
2011 Consultant Rheumatologist, Imperial College Healthcare NHS Trust, Charing Cross Hospital, and Honorary Senior Lecturer, Imperial College London
2009 NIHR Walport Clinical Lecturer: Kennedy Institute of Rheumatology, Imperial College London, and Imperial College Healthcare NHS Trust
2008 Rheumatology SpR: Charing Cross Hospital, London
2005 Clinical Research Fellow: Kennedy Institute of Rheumatology, London
2004 Rheumatology SpR: Charing Cross Hospital, London
2003 Rheumatology SpR: Watford General Hospital
2000 Senior House Officer: Mayday Hospital, West Middlesex University Hospital, Hammersmith Hospital, Chelsea and Westminster Hospital and St George's Hospital, London
1999 House Officer: St Thomas’s Hospital and University Hospital Lewisham, London

**Research Summary**

A key focus of my research has been anti-citrullinated protein antibodies which are a characteristic feature of rheumatoid arthritis (RA). I have been particularly interested in determining which specific proteins are targeted in RA, how immunological tolerance is broken, and whether these novel antibodies can predict clinical outcomes in RA.

A second and increasingly important direction of my research is early phase clinical trials in Sjögren's syndrome and the application of novel outcome measures including imaging and biopsies. Sjögren’s syndrome is an autoimmune condition resulting in dryness of the eyes and mouth, alongside a range of other complications that occur in a proportion of patients. I am the clinical director of the OASIS cohort (Optimising Assessments in Sjögren’s Syndrome) which aims to follow patients with Sjögren’s syndrome over time, with a view to improving how we measure disease activity and predict outcomes, as well as understanding the risk factors for this disease.

I also have a long-standing interest in the effect of polyunsaturated fatty acids on the immune response and how they may influence diseases such as RA.
Senior Clinical Lecturer in Rheumatology  
NIHR Career Development Fellow

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Career History
After completing Specialist Training in Rheumatology, I pursued a Masters’ Degree in Public Health at Harvard University, with a focus on Clinical Effectiveness. I obtained a Pan-American Fellowship from the Fogarty International Center-National Institutes of Health (NIH)-Conacyt, followed by a Postdoctoral Fellowship from the Arthritis Foundation (USA) to get further training in clinical research methods and epidemiology at the Department of Rheumatology, Brigham & Women’s Hospital, and Harvard Medical School. After completing the postdoctoral fellowship, I was appointed an Assistant Professor in Medicine at the Tufts University Medical School, New England Medical Center in 2005. I relocated to the United Kingdom in 2008 and was appointed a Clinical Lecturer at the University of Birmingham, obtaining my Royal College of Physicians of the United Kingdom Specialist Certificate in Rheumatology in 2012, followed by a PhD degree in Medicine from the University of Birmingham in 2013. I have recently received a prestigious NIHR Fellowship for 5 years and was appointed a Senior Lecturer at the University of Birmingham.

Research Summary
My research is focused in the epidemiology and outcomes of rheumatic diseases, in particular rheumatoid arthritis (RA) and lupus. My research interests include the modifiable risk factors, predisposing conditions, as well as the comorbidities of RA and the systemic impact of the burden of chronic inflammation. Previous work includes a meta-analysis of observational studies and randomized controlled trials showing that there is a critical period during which anti-rheumatic therapy should be initiated, a therapeutic window of opportunity early in the course of RA, associated with sustained benefit on disease progression and structural damage. I have previously investigated novel and traditional risk factors for cardiovascular disease in RA and showed that individuals with RA have higher inflammatory markers associated with cardiovascular disease and lower antioxidant levels. Our group also showed in a large epidemiologic study that systemic inflammation has an impact on bone loss. In recent work, our group has studied associations between periodontal disease, a common chronic inflammatory disease, and RA and lupus. We are conducting a clinical trial investigating the effect of periodontal therapy on RA outcomes (OPERA trial). We are currently investigating novel biomarkers and causal pathways in the development of RA.
Senior Lecturer

Dr Helen McGettrick

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Career History
2017  Senior Lecturer, University of Birmingham
2016  Garrod Prize Winner (British Society of Rheumatology)
2012  ARUK Career Development Fellow, University of Birmingham
2011  University Fellow in Inflammation Biology, Systems Science for Health, University of Birmingham
2006  Postdoctoral Researcher, Centre for Cardiovascular Sciences, University of Birmingham
2006  PhD. (Medical Sciences), Centre for Cardiovascular Sciences, University of Birmingham
2002  MSc. (Immunology), MRC Centre for Immune Regulation, University of Birmingham
2001  BSc (Biochemistry), University of Lancaster

Research Summary
My research group is interested in identifying the endogenous regulatory pathways that control the recruitment of the inflammatory infiltration during an acute inflammatory episode, and how these go wrong in immune mediated inflammatory diseases. Broadly speaking we have two main areas of interest: (i) mesenchymal stromal cell (MSC) crosstalk with vascular endothelial cells and (ii) the adiponectin-PEPITEM axis in rheumatoid arthritis and aged-related bone diseases. We combine imaging novel in vitro, multi-cellular static and flow-based culture systems incorporating primary human cells (from healthy individuals or patients), with systems biology approaches to large omics datasets and murine models of acute or persistent inflammation.
Arthritis Research UK Career Development Fellow
Senior Research Fellow

Dr Rowan Hardy
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Career History:
2015 Arthritis Research UK Career Development Fellow, University of Birmingham
2012 Arthritis Research UK Post-doctoral Researcher, University of Birmingham
2011 Arthritis Research UK Visiting Fellow, University of Sydney
2010 Arthritis Research UK/ Wellcome Trust Post-doctoral Researcher, University of Birmingham
2008 PhD in Medicine, University of Birmingham
2004 BMedSci (Cellular and Molecular Pathology), University of Birmingham

Research Summary:
I specialise in steroid metabolism, with extensive experience in inflammatory animal models. Having recently received a prestigious Career Development Fellowship I am now establishing a research group focussing on the roles of glucocorticoid signalling in inflammatory disease. Patients with RA develop complications such as muscle wasting and bone loss, which contribute to an increased risk of fractures, disability and reduced life expectancy. My primary focus is exploring the roles of pre-receptor glucocorticoid metabolism in mediating these detrimental features of chronic inflammatory disease and examining a role for therapeutic agents that modify glucocorticoid signalling in their management. The inter-disciplinary collaboration linking the Institute of Metabolism & Systems Research and the Institute of Inflammation & Ageing that I have established ensures a fully translational approach to this research, with access to state of the art cell culture, molecular biology and animal husbandry facilities.
Arthritis Research UK Career Development Fellow
Research Fellow

Dr Amy Naylor
a.naylor@bham.ac.uk
Tel: +44 (0)121 371 3266

Career History:

<table>
<thead>
<tr>
<th>Year</th>
<th>Position/Programme</th>
<th>Institution</th>
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</thead>
<tbody>
<tr>
<td>2018</td>
<td>Arthritis Research UK Career Development Fellowship</td>
<td>University of Birmingham</td>
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<tr>
<td>2012</td>
<td>Post-doctoral Researcher</td>
<td>University of Birmingham</td>
</tr>
<tr>
<td>2009</td>
<td>Arthritis Research UK Foundation Fellowship</td>
<td>University of Birmingham</td>
</tr>
<tr>
<td>2004</td>
<td>PhD</td>
<td>Nuffield Foundation, Oliver Bird Rheumatism Scheme, University of Newcastle</td>
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Research Summary:

Bone turnover and remodelling is critical for the maintenance of bone health. Many diseases associated with inflammation and ageing, including rheumatoid arthritis and osteoporosis, feature dysregulation of bone turnover with increased bone loss and a decrease in bone generation and repair. The outcome for patients is loss of joint function and bone fragility.

My research is focussed on boosting osteoblast activity in order to increase bone formation and repair. Specifically, I am investigating the crosstalk between bone resident cells including osteocytes, osteoblasts and endothelial cells to identify the molecular pathways that regulate osteoblast trafficking to sites of bone remodelling. I have a particular interest in working at the interface between scientific disciplines and using cutting edge techniques from the chemical, engineering and computer sciences to answer these complex biological questions.
Lecturer in Behavioural Rheumatology

Dr Marie Falahee
m.falahee@bham.ac.uk
Tel: +44 (0)121 371 3235

Career History:

2017 Lecturer, Institute of Inflammation and Ageing, University of Birmingham
2014 Post-doctoral Researcher, Institute of Inflammation and Ageing, University of Birmingham
2000 Career Break
1997 Lecturer in Psychology, Aston University
1995 Research Fellow, School of Psychology, University of Birmingham
1992 PhD School of Psychology, University of Birmingham
1989 BSc University of Birmingham

Research Summary:

Patient perceptions are key predictors of clinical outcomes and health behaviours, and a central focus of my research is on understanding the perspectives of individuals with, or at risk of musculoskeletal disorders and the development of psychological and/or educational interventions to improve clinical outcomes or enhance shared decision making.

There is an increasing research interest in the development of predictive biomarkers and preventive strategies for rheumatoid arthritis, and I have a particular interest in the measurement of patient preferences in this context, in order to facilitate the efficient translation of this work and to inform the design of future clinical services that are effective and acceptable to patients and other stakeholders. I am also involved in the development of international evidence-based recommendations on the methodologies used to integrate patient preferences into decision making throughout the lifecycle of medical products as a partner in a European Union Innovative Medicines Initiative funded consortium.

I am also passionate about patient and public involvement in research and as academic coordinator of the Birmingham Rheumatology Research Patient Partnership (R2P2): http://www.birmingham.ac.uk/r2p2 she facilitates patient involvement across the Rheumatology Research Group, ensuring that our research is informed by patients’ needs, experiences and expectations and is sharply focussed on public benefit.