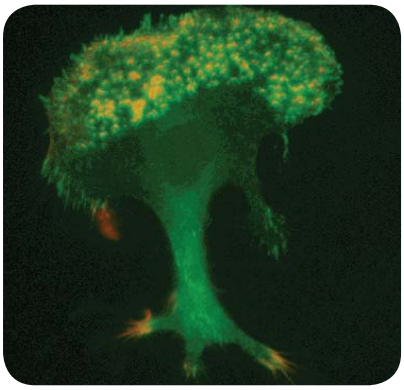
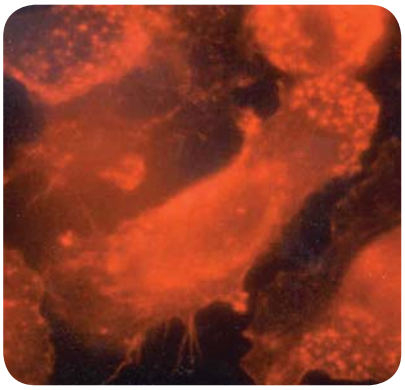
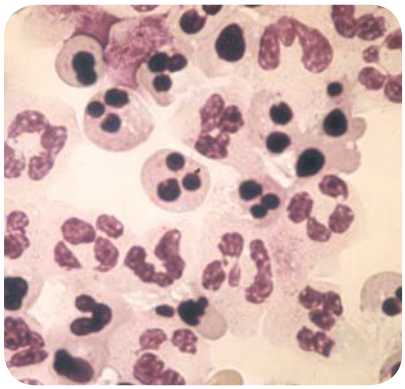
The Royal Society of Edinburgh

Caledonian Research Foundation

Inflammation and Inflammatory Disease







Report of a Conference organised by
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and
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Thursday 29 & Friday 30 November 2007

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Rapporteur: Ms Jennifer Trueland

Inflammation and Inflammatory Disease, 29-30 November 2007

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and thank the Organising Committee

Professor F Y Liew FRSE
Division of Immunology, University of Glasgow

Professor C Haslett OBE FRSE
Head, Division of Clinical Sciences and Community Health and Associate Dean, University of Edinburgh

Introduction / summary

Inflammation and the inflammatory process are central to many diseases, including cancer, heart disease and arthritis. This is not a new observation; in the first century AD, the Roman writer Aulus Cornelius Celsus noted the four cardinal signs of inflammation, namely rubor, calor, dolor and tumor (redness, heat, pain and swelling). Galen added to that loss of function.

That did not mean, however, that the ancients came up with cures for inflammatory diseases. Indeed, until well into the last century there was little in the way of treatment for some of the most common and, even now, cures for many are elusive.

There was a sense of history, however, at this Caledonian Research Foundation (CRF) conference and a feeling that times are beginning to change. Perhaps co-chair Professor Chris Haslett summed it up best when he told the audience that his previous pessimism about the whole area of inflammatory disease was being replaced with a real sense of hope.

Many of the distinguished speakers had been directly involved in recent dramatic developments which have led to a step change in the treatment of inflammatory disease. These included Professor Marc Feldmann, who described how he came to discover anti-TNF therapy, one of the big clinical success stories in treating chronic disease.

Others described promising new targets for potential new treatments and demonstrated how understanding of the molecular basis for diseases, including rheumatoid arthritis and vasculitis, was improving.

There was also a political dimension, however. Glasgow's Professor Ian McInnes was particularly keen to move inflammatory diseases 'up the agenda' and to see a renewed focus on finding effective new cures. He called for immunology groups to work together to find new treatments and to make a concerted effort to treat patients within clinical trials, sharing the knowledge and pushing the field forward, taking the cancer community as a model.

All in all, the conference lived up to the introduction from Professor David Baird, retired obstetrician and immediate past-Chairman of the CRF Governors. It was important, he said, to have such conferences, free from the influence of commercial sponsors, to discuss and drive forward areas of clinical and scientific importance.

Key messages

- **Inflammation and inflammatory disease is an important clinical area**
- **There have been several important discoveries in the past two decades which are transforming treatment for conditions such as rheumatoid arthritis**
- **Genetic and environmental factors are involved**
- **A number of promising therapies is in development, but there is still considerable unmet need**
- **There is good co-operation between scientists and clinicians in this field but a new, concerted focus is needed to see a further step change in treatments and outcomes.**

Summary / key messages from speakers:

Professor Hilary Critchley: Menstruation is an inflammatory event involving tissue injury and repair which may provide a model for other inflammatory conditions.

Professor George Kollias: Using animal models and modern genetic techniques is helping to improve understanding of disease processes and find potential therapeutic targets.

Dr Connie Weyand: The immune system ages prematurely in patients with rheumatoid arthritis, so finding a way to stop it ageing might be a treatment possibility.

Professor Caroline Savage: Serine proteases, such as PR3, may become an important therapeutic target for vasculitis and better understanding is needed of how the current new treatment, Rituximab, works.

Professor Timothy Williams: Cell migration or trafficking appear key to inflammation and the chemokine Eotaxin seems to play an important part in this, suggesting new therapeutic targets.

Professor Andreas Radbruch: Memory cells are key to long-term chronic infection, so removing and replacing immune systems appears to be showing good results in a small number of patients with lupus and multiple sclerosis.

Professor Marc Feldmann: Anti-TNF therapy was a major breakthrough and other cytokines are targets for new treatments.

Professor Ian McInnes: In spite of the success of anti-TNF and other new therapies, there is still unmet need. Cytokines are a good target for novel therapies.

Professor Lars Klareskog: The interplay of genetic and environmental factors must be considered when seeking treatments for rheumatoid arthritis.

Professor Adriano G Rossi: Manipulating programmed cell death pathways could be a therapeutic target for successfully resolving inflammation.

Dr David Jayne: Cytokines and cytokine receptors which act on B cells could be possible targets for developing treatments for multi-system autoimmunity.

Professor George Schett: Treatment for inflammatory joint disease should consider both how to block enhanced osteoclast production – which causes bone destruction – and also how bone formation pathways are regulated.

Inflammation and Inflammatory Disease

Thursday 29 November 2007

Mechanisms of Inflammation

Menstruation: A Pivotal Reproductive Inflammatory Event

Professor Hilary Critchley, *Centre for Reproductive Biology, University of Edinburgh*

Menstruation is an inflammatory event which involves tissue injury and repair. As such, it may serve as a paradigm for these processes elsewhere in the body. But menstrual disorders also bring their own problems and can have a considerable impact on women's physical, economic and psychological wellbeing. In order to improve the medical treatment of women with menstrual problems, it is essential to understand the mechanisms involved in uterine bleeding.

Professor Critchley said that the process of tissue injury and subsequent repair in menstruation involved a complex interplay between the endocrine and the local immune systems, with the functional layer of the human endometrium undergoing serial degeneration and renewal each menstrual cycle.

Many of the molecules involved in menstruation, however, are also those involved in the body's inflammatory response to injury. These include a dynamic population of leukocytes within the endometrium and there is also a complex interplay between sex hormones, immune system cells, locally produced cytokines and growth factors.

Menstrual bleeding occurs when the sex hormone, progesterone, is withdrawn, although how that happens is not fully understood. Several compounds could be involved, including uterine cytokines, VEGF and glucocorticoids.

Professor Critchley concluded that menstruation was an inflammatory event and there are a number of local mediators at play. A better understanding of the regulation of normal menstruation could open the way to better treatments for menstrual problems. It could also help to improve understanding of inflammation mechanisms and tumour formation elsewhere in the body.

Questions

Professor Haslett was taken with the concept of menstruation as a dramatic inflammatory event which got better and asked if menorrhagia could be partly due to inflammation. Professor Critchley compared it with labour and pre-term labour, where the inflammatory mechanisms were reprised with an influx of neutrophils. She added that while people often thought this was systemic, it could be controlled at a local level with steroids.

One questioner pointed out that joint inflammation could flare in rheumatology patients during menstruation. Professor Critchley said that inflammatory bowel disease could also be exacerbated at this time.

Professor Baird pointed out that the phenotype in heavy menstrual bleeding was not defined, so until the pathology was better defined, it would be hard to find a single cause. Professor Critchley agreed that better definitions were needed.

The Pathophysiology of Tumor Necrosis Factor: Insights from Animal Models

Professor George Kollias, *President and Director, Biomedical Sciences Research Centre Alexander Fleming, Greece*

Tumor Necrosis Factor (TNF) is one of the big stories in inflammation and autoimmunity. The protein plays an essential part in the development of rheumatoid arthritis, spondyloarthritis and Crohn's disease; and anti-TNF therapies have proved to be a breakthrough treatment for these conditions.

Professor Kollias, who is renowned for the development and characterisation of transgenic and knockout mice, spoke about the work done in his lab and beyond, to try to find out more about the specific function of TNF and its receptors.

Using animal models, researchers are trying to map molecular and cellular pathways which involve TNF. As yet, the specific mechanisms are unknown, but there are promising lines of inquiry. Professor Kollias and colleagues are using functional genomics and high-throughput technologies to investigate genes or pathways potentially involved in TNF-mediated disease. Using a large-scale integrated expression approach in transgenic mice, they have identified many genes and pathways which are deregulated in diseased cells. Mesenchymal cells and follicular dendritic cells for example, are involved in the TNF-signalling process.

Again, however, Professor Kollias stressed that there was not likely to be a single cause or mechanism for these diseases or processes. He concluded that a

number of different mechanisms could be at play and that this might help to explain the efficacy and safety of anti-TNF therapies.

Using information from both animal and patient samples, the researchers are building up a database of potential therapeutic targets and treatments.

Questions

Dr Weyand asked if the Col VI activity in mesenchymal cells noted in the joint and gut was also in other organ systems. Professor Kollias responded that this was not at a level that the researchers could detect. He was asked by one rheumatologist why only 30% of his patients responded to an anti-TNF therapy. Professor Kollias said that it was unexpected to find human disease so homogeneous and regarded himself lucky to have seen this level of response.

Rheumatoid Arthritis as a Syndrome of Accelerated Immune Senescence

Dr Connie Weyand, *Division of Rheumatology, Emory University School of Medicine, Atlanta*

Dr Weyand began by defining various national reactions to ageing: while the French think it's a nuisance and the British think it's a fact, for Americans it's regarded as 'an option'.

Although this may have been a joke, it underlined the point of her paper, which was that rheumatoid arthritis (RA) is caused by an ageing and failing immune system. The 'option' part comes into it because, if it's possible to stop the immune system ageing, then RA might also be halted.

Dr Weyand pointed out the paradox that it is when our immune systems begin to age that we develop conditions like RA, which rely on immune and inflammatory responses. Her hypothesis is that RA is, paradoxically, caused by a failing, rather than efficient immune system.

The immune system depends on massive expansion and contraction of cell numbers, imposing intense proliferative stress and restricting the lifespan of lymphocytes.

She demonstrated that the body's production of thymic T cells falls away dramatically once it reaches the fifth decade, leading to a remodelling of the immune system. This was illustrated by a progressive loss of telomeres – the cell's internal 'clock' – and Dr Weyand said that immunoregulatory receptors were different in 'young' and 'old' T cells. Patients with RA accumulate senescent T cells – indeed, their immune system is 20-30 years pre-aged, she said. Not only does this affect the 'memory cells' which are inflammation-mediating, but also the immune system's 'reserve' of naive T cells.

She concluded that there was a mechanistic link between accelerated immunosenescence and chronic inflammatory disease in RA.

Questions

Asked if there was a therapeutic answer, Dr Weyand said it would be important to understand what makes the cells get older, then try to help them stay younger, or make them younger.

She was asked about the role of chronic viral infections and said that these did not seem to affect the repopulation of immune cells – while it might affect the memory pool, the naive pool was not affected. While it's a 'hen and egg' situation, in her view the immune system gets old first, then lets the viruses in, rather than the other way round.

Inflammation and Destruction in Rheumatoid Arthritis: Pathways and Therapeutic Implications

Professor Josef Smolen, *Division of Rheumatology, Medical School of Vienna*

Rheumatoid arthritis (RA) is characterised by the propensity for destruction of cartilage and bone, which is brought about by the inflammatory response of the disease. Other disorders which have a similar inflammatory response have less potential for destruction, however.

Professor Smolen said that the pathways leading to joint damage in RA seem to be connected to the high level of proinflammatory cytokines which allow osteoclasts – the cells which destroy bone – to become hyperactive.

He said TNF is an important cytokine in the pathogenesis of destructive arthritis, but it is not the only one. What is known is that TNF enhances osteoclastogenesis, primarily via TNF-R1, while TNF-R2 may prove to have a protective role.

In practice, when treating patients with RA it is important to aim to prevent damage both occurring in the first place and building up, but the only certain way to do that is by inducing remission. In all other situations, joint destruction will continue.

He stressed that since anti-TNF therapy with MTX reduces joint damage at any disease activity state, it is important to report disease state in clinical trials.

Professor Smolen concluded that the effects of therapy could be predicted within 3 – 6 months, which allowed a flexible approach. Although reducing inflammation helps to slow destruction, progression to disability will only be stopped by turning off the inflammatory response completely.

Questions

He was asked about side-effects from combination therapy and pointed out the benefits of a dynamic approach; if one therapy isn't working and, indeed, is proving toxic, then you shift to another. He would like to see novel therapies, however.

He was also interested in combining anti-TNF with bisphosphonates to see whether it was possible to regenerate bone and repair joints.

Pathogenic Mechanisms in Vasculitis

Professor Caroline Savage, *Institute of Biological Research, University of Birmingham*

Vasculitides can attack large, medium or small vessels, causing damage to blood vessel walls. Small vessel vasculitides (SSV) can cause significant damage to major organs including the kidney, respiratory and cardiovascular systems.

SSV have strong autoimmune features and, although it is not known precisely what agents cause them, SSV do have an association with anti-neutrophil cytoplasm antibodies (ANCA), which may play an important part in the development of the disease.

Remission can be induced with corticosteroids and cyclophosphamide and recent successful treatment with anti-CD20 therapy (Rituximab), which depletes B cells, suggests they are also involved. Professor Savage described results in patients in Birmingham using Rituximab, where remission and B-cell depletion were induced in all, but the mechanism of action is still now known. Her hypothesis is that autoantibodies might predominantly be produced by short-lived Ab-producing cells, dependent on repopulation by precursor B cells.

She concluded that inflammation in vasculitis may be driven in many instances by ANCA-neutrophil interactions. But modulation by cytokines is key to this. In addition, certain microvascular beds are more susceptible to injury, so B cells may be needed to support inflammatory niches where plasma cells can contribute to autoantibody formation and T cells may be overactive. Injury may be largely driven by neutrophil serine proteases, making them potentially important therapeutic targets.

Questions

Asked if local antibodies were more important in vasculitis than systemic ones, Professor Savage said that while the plasma cells may be in the bone marrow, when a lot of plasma cells take hold they can do so locally. One neutrophil can go elsewhere and cause injury, for example to the lung, she added. Dr Weyand asked about other organs and was told that no organ was exempt from vasculitis, including the skin and the brain. It is a heterogeneous disease, there may be a genetic predisposition and environmental factors such as smoking may also be involved.

Chemo Attraction of Inflammatory Cells to Sites of Allergic Inflammation

Professor Timothy Williams,*National Health & Lung Institute, Imperial College, London*

Allergic diseases such as asthma involve an accumulation of inflammatory cells in tissues. While the interplay between the cells (leukocytes) and tissue gives rise to symptoms, it is not known exactly how it works.

Professor Williams and his team have been studying the chemoattractants released during allergic reactions and looking at their involvement in trafficking leukocytes from the blood to the tissue. One discovery is that mast cell progenitors express the BLT1 receptor, which may provide a possible mechanism for increasing mast cells in tissues at sites of allergic inflammation, as activated mature mast cells produce the ligand for this receptor, LTB4.

The team discovered Eotaxin, a chemokine produced in allergic reactions which is important in the recruitment of eosinophils, which have been associated with lung damage. Eotaxin is produced by several different cells types and signals via the CCR3 receptor, which is present on eosinophils, mast cells and Th2 cells.

Professor Williams said that future treatments could target the trafficking of specific leukocyte types, such as eosinophils or mast cell progenitors, by blocking particular chemoattractant receptors. Therapies could inhibit mast cell hyperplasia, which occurs in allergic rhinitis, asthma and parasitic infection.

Questions

Professor Critchley asked if there could be a synergistic effect with other compounds. Professor Williams replied that the effectiveness might depend on blood supply, but that he would like to try other

compounds. Asked if IGE was chemotactic, he said he had not explored it.

Immunological Memory and Chronic Inflammation

Professor Andreas Radbruch, *Scientific Director, Deutsches Rheuma-Forschunszentrum, Berlin*

Immunosuppression (damping down the immune response) is the current therapeutic strategy for treating chronic inflammation, but this does not provide a cure for many patients. Professor Radbruch said the reason for this could be that it does not target pathogenic immunological memory. In other words, the immune system is protected by cells which remember how to resist therapies which tackle the primary immune responses. These protective cells are not proliferating, which removes the possibility of tackling them through mechanisms involved, in or inducing, proliferation.

Using animal models, Professor Radbruch has shown that the protective cells depended on their ability to express a particular gene - twist 1 in memory Th1 cells for example, to form survival niches for memory cells. Targeting these memory cells could, therefore, be key to effective therapies for chronic inflammation. In other words, making patients' bodies 'forget' that they had rheumatic inflammation could be the key to curing it.

He showed results in patients who had undergone immunablation and whose immune system had been rebuilt using stem cells. Pathogenic memory was deleted in these patients with SLE or MS, their autoantibodies disappeared and a new, young immune system developed.

Questions

Dr Weyand asked if he was right in believing that the immune system is necessary to the brain and that would there not be risks with this type of treatment. Professor Radbruch responded that there were risks and consequences with all experimental therapies.

Friday, 30 November 2007

Therapeutic Approach to Inflammatory Disease Anti-TNF Therapy Heralds a Major Therapeutic Development: Anti-Cytokines

Professor Marc Feldmann, *Kennedy Institute of Rheumatology, Faculty of Medicine, Imperial College, London*

Professor Feldmann discovered (in the early 1980s) anti-TNF therapy, which has had a tremendous impact on the treatment of rheumatoid arthritis and other autoimmune diseases and has since been used to treat more than a million patients. He described how the discovery came about and possible ways forward for new treatments.

He pointed out that cytokines are important in every biological process, including inflammation and immunity. Many cytokines are produced in rheumatoid synovium, so he and colleagues were looking to see if there were any therapeutic targets. Analysis of cytokine regulation revealed the importance of tumor necrosis factor (TNF).

By 1991, the rationale behind the treatment had been established and clinical trials began the following year. They were successful and the first drugs were registered at the end of the millennium.

Professor Feldmann pointed out that it has since been clear that TNF was the body's 'fire alarm' and that many cytokines were effective therapeutic targets. The issue is linking the disease with the appropriate cytokine target. While successful in treating symptoms, however, anti-cytokine therapies do not cure; most work better in combination with other drugs and they are expensive.

He concluded that cytokine medicine had come of age in chronic conditions, with many promising new therapies now in trials, but as yet there was nothing for acute disease.

Cytokines as Therapeutic Targets in Inflammatory Disease Responses

Professor Ian McInnes, *Division of Immunology, Infection & Inflammation, Biomedical Research Centre, University of Glasgow*

Despite the success of therapies such as anti-TNF, Professor McInnes pointed out that there was still unmet clinical need in rheumatoid and psoriatic arthritis. In his view, the important thing is to aim for remission. He pointed out that it is still necessary to know more about disease processes, and added that cytokines other than TNF might also be good therapeutic targets. Even so, he would like to see treatment initiated at as early a stage as possible because there is evidence that this leads to better outcomes, whatever the therapeutic agent.

He would like treatment to reduce inflammation given to patients as soon as they present at a clinic, to try to prevent chronic damage. Cytokines may be good candidates for such treatments because of their role in the early stages of inflammation - in particular, he

wants to know more about IL-12, IL-23, IL-15 and IL-33. Different cytokines may be targets for different conditions, for example, anti-IL-33 antibody is in preclinical trials with asthma as a first target and anti-IL-18 is being tested in psoriasis.

Professor McInnes said he would like to see more collaborative working to find the best targets for each condition and would also like more political engagement with the issues.

Questions

Professor Haslett commented that there was an explosion of potential therapeutic agents to use with patients and clinicians didn't want to miss anything that might kill off disease. Professor McInnes said there were lots of molecules, but as they were not cheap, he was concerned that the pharmaceutical industry would lose interest in rheumatoid arthritis.

Professor Feldmann said there was a lot of 'life' after TNF, but Professor McInnes said it was up to the immunology community to make sure that new treatments came on stream by working together and offering patients the chance to enter clinical trials – much as the cancer community had done. "We have a duty of care to the next generation as well", he concluded.

Genes, Environment and Immunity in the Development of Rheumatoid Arthritis

Professor Lars Klareskog, *Rheumatology Unit, Karolinska Institute, Sweden*

Much work has been done to try to identify genes implicated in rheumatoid arthritis (RA) but environmental factors are also involved. Professor Klareskog described the EIRA (Environment & Immunity in Rhematoid Arthritis) Study, which looks at the involvement of genes and the environment in the triggering of RA, which has recruited around 3,000 cases and 3,000 controls to date. Several genes, including HLA-D and PTPN22, are known to be risk factors for RA and there are now several new candidate genes. Collaborations are being set up and expanding between different groups internationally, who are pooling the findings of studies such as EIRA.

Environmental factors are also important, however. Professor Klareskog demonstrated that smoking is a risk factor for ACPA/RF-positive RA, but not for ACPA-negative RA. Risk increases with the amount smoked.

Professor Klareskog posited that certain genes, particularly those coding for some MHC class II transplantation antigens, may act together with environmental factors to cause immune reactions towards proteins which have been modified by a process of citrullination. Antibodies to citrullated peptides are present in the majority of RA patients, but rare in the population generally. There are still many questions to be answered, including whether citrullination takes place before, during, or after the onset of RA.

He concluded that the scene was now set for immunologists to research the aetiologies and molecular pathways of RA subsets in the light of new information from genetic epidemiology. Genetics, however, must be combined with information on the environment, he stressed.

Questions

Professor Haslett asked if there were risk antibodies present or identifiable in children. Professor Klareskog said he was not aware of them being looked for in children. In response to Professor Feldmann's enquiring about how the environment affected co-morbidities, Professor Klarekog said that he hoped to know the answer within half a year.

Novel Strategies to Resolve Inflammation

Professor Adriano G Rossi, *MRC Centre for Inflammation Research, University of Edinburgh*

Tackling the processes which lead to programmed cell death (apoptosis) may well provide new therapeutic targets for reducing inflammation, said Professor Rossi.

Apoptosis is an efficient way of clearing potentially histotoxic cells from inflamed sites and is therefore an important factor in resolving inflammation. Dr Rossi and colleagues have been looking at neutrophil apoptosis and also the phagocytosis of apoptotic cells by macrophages and how they can be regulated by pharmacological intervention.

They have shown that signalling pathways and kinases have been shown to regulate cell death and survival *in vitro* and have also been shown to be involved in reducing inflammation in animal models.

There are several candidates for therapeutic targets, including proteins involved in apoptosis (such as Bcl2 family) and cyclin-dependent kinase inhibitor drugs, which induce neutrophil apoptosis.

The team has also produced evidence that CDK inhibitors (being developed as a cancer treatment) promote neutrophil cell death in inflammation where neutrophils are dominant, including in pleurisy and

arthritis.

Professor Rossi explained that their plans for future work include defining the mechanisms of CDK inhibitor-driven resolution of inflammation and testing their efficacy in animal models and in humans with lung inflammation.

Questions

Professor Williams asked if CDK affected cell turnover. Professor Rossi said it could affect proliferation as a side-effect and that it could also affect bone marrow.

Targeting the B Cell in Multi-System Immunity
Dr David Jayne, Dialysis Centre, Addenbrooke's Hospital, Cambridge

Dr Jayne raised the realistic prospect of new treatments for multi-system immune disease such as vasculitis and lupus. He gave an overview of treatments available to date, from steroids in 1948, through immunosuppression in the 1960s to biological means in the 21st century. Newer treatments had resulted from a better understanding of the pathogenesis of the conditions and from developments in recombinant gene technology.

The particular thrust of his presentation was tackling B cells, which are key to autoantibody production and implicated in the development of autoimmune disease.

Therapies, such as Rituximab, which deplete B cells have the potential to treat vasculitis and lupus but many questions remain. It may be that Rituximab is unsuccessful when B cells are able to find a place to 'hide' and therefore avoid depletion. It is also the case that Rituximab does not work for everybody and there have been concerns about side-effects, including infection.

Dr Jayne said that other therapeutic targets aimed at the B cell were coming on board, including Blyss, a B-cell stimulating cytokine and the receptor TAC1. Although B-cell therapy will not adequately address induction and scarring, he said, it could be used in combination with other treatments.

Questions

Professor Rossi asked about treatment for lupus. Dr Jayne replied that lupus was associated with poor clearance of apoptotic cells. While steroids were an effective treatment, he had not seen a difference between lupus and vasculitis.

Joint Remodelling:
Pathways of Destruction and Rebuilding

Professor George Schett, *Department of Medicine, University of Erlangen-Nuremberg, Germany*

Inflammatory joint disease leads to destruction of bone and cartilage and changes the architecture of the joint. Osteoclasts are implicated in degradation of subchondral bone and mineralised cartilage – if there are no osteoclasts, there is no bone erosion.

Various molecules, including the RANK ligand, drive osteoclast formation, but generation of these cells can be affected by inflammatory cytokines and chemokines such as MCP-1, which is expressed in the synovial tissue. Further, although some T cells (possibly Th17 cells) enhance osteoclast production, regulatory T cells suppress it.

The remodelling pattern of the joint architecture seems to be different in various inflammatory diseases such as rheumatoid arthritis, ankylosing spondylitis and psoriatic arthritis.

Professor Schett concluded that in order to preserve joint architecture, therapeutic strategies may have to regulate bone formation pathways – such as Wnt-signalling and BMP-signalling – as well as blocking enhanced osteoclast formation in the joint.

Questions

Professor Rossi asked if osteoclast formation was blocked by steroids. Professor Williams said that steroids work through osteoblasts, but do not prevent osteoclast formation. When a question was raised about drug treatments, Professor Williams added that TNF therapy was effective in limiting structural damage, but that there was potential for drugs through targeting bone formation.

Appendix One
Programme

Thursday 29 November 2007

DAY 1: Mechanisms of Inflammation

09.30: CRF Chairman's Welcome

09.40: Conference Chairman's Welcome
Professor F Y Liew FRSE
Division of Immunology, University of Glasgow

09.50: Menstruation: A Pivotal Reproductive Inflammatory Event
Professor H O D Critchley
Centre for Reproductive Biology, University of Edinburgh

10.20: Q & A

10.35: The Pathophysiology of Tumor Necrosis Factor: Insights from Animal Models
Professor George Kollias
President and Director, Biomedical Sciences Research Center 'Alexander Fleming', Greece

11.05: Q & A

11.20: Tea & Coffee

11.40: Rheumatoid Arthritis as a Syndrome of Accelerated Immune Senescence
Dr Connie Weyand
Division of Rheumatology, Emory University School of Medicine, Atlanta

12.10: Q & A

12.25: Inflammation and Destruction in Rheumatoid Arthritis: Pathways and Therapeutic Implications
Professor Josef Smolen
Division of Rheumatology, Medical University of Vienna

12.55: Q & A

13.10: Lunch

14.10: Pathogenic Mechanisms in Vasculitis
Professor Caroline Savage
Institute of Biological Research, University of Birmingham

14.40: Q & A

14.55: Chemo Attraction of Inflammatory Cells to Sites of Allergic Inflammation
Professor Timothy Williams
National Heart and Lung Institute, Imperial College London

15.25: Q & A

15.40: Tea & Coffee

16.00: Immunological Memory and Chronic Inflammation
Professor Andreas Radbruch
Scientific Director, Deutsches Rheuma-Forschungszentrum, Berlin

16.30: Q & A

16.45: Chairman's closing/summary

17.00: Close

Friday 30 November 2007

DAY 2: Therapeutic Approach to Inflammatory Disease

09.30: Conference Chairman's Welcome
Professor C Haslett OBE FRSE
Head, Division of Clinical Sciences and Community Health and Associate Dean, University of Edinburgh

09.35: Anti-TNF Therapy Heralds a Major Therapeutic Development: Anti-Cytokines
Professor Marc Feldmann FRS
Kennedy Institute of Rheumatology Division, Faculty of Medicine, Imperial College London

10.05: Q & A

10.20: Cytokines as Therapeutic Targets in Inflammatory Rheumatic Diseases
Professor Ian McInnes
Division of Immunology, Infection and Inflammation, Glasgow Biomedical Research Centre

10.50: Q & A

11.05: Tea & Coffee

11.20: Genes, Environment and Immunity in the Development of Rheumatoid Arthritis
Professor Lars Klareskog
Rheumatology Unit, Karolinska Institute, Sweden

11.50: Q & A

12.05: Novel Strategies to Resolve Inflammation
Professor Adriano G Rossi
MRC Centre for Inflammation Research, University of Edinburgh

12.35: Q & A

12.50: Lunch

13.50: Targeting the B Cell in Multi-System Autoimmunity
Dr David Jayne
Dialysis Centre, Addensbrooke Hospital, Cambridge

14.20: Q & A

14.35: Joint Remodelling: Pathways of Destruction and Rebuilding
Professor George Schett
Department of Medicine, University of Erlangen-Nuremberg, Germany

15.05: Q & A

15.20: Chairman's Closing/Summary

15.40: Close / Drinks Reception

Appendix Two
Speakers' Biographies

Professor F Y Liew FRSE
Division of Immunology, University of Glasgow

Eddy Liew obtained his PhD in Immunology and Microbiology at the John Curtin School of Medical Reaseach, Australia National University, Canberra. He taught at the University of Malaysia, Kuala Lumpur and then joined the Wellcome Research Laboratories in Beckenham, London. Since 1991 he has been Gardiner Professor and Head of the Department of Immunology, Infection and Inflammation, University of Glasgow, Scotland. He has published more than 290 papers and contributed significantly to the concept of the heterogeneity of CD4+ T cells. He also identified CD4+ suppressor cells in 1981 and applied this to Leishmania and influenza infections. He later studied the role of nitric oxide in infection, inflammation and in the regulation of T cell response. His recent contributions include identifying the role of IL-15 and IL-18 in rheumatoid arthritis and the clinical application of novel cytokines. He was elected a fellow of the Royal Society of Edinburgh in 1995 and a fellow of the Academy of Medical Science in 1999. He was awarded the Hamdan Award for Medical Research Excellence in 2002. He is currently Director of the Glasgow Biomedical Research Centre and chairs the Medical Research Panel, Research Grant Committee, Hong Kong. He was President of The European Federation of Immunological Society (EFIS) from 2003 to 2006.

Professor H O D Critchley
Centre for Reproductive Biology, University of Edinburgh

Hilary Critchley is Professor of Reproductive Medicine at the University of Edinburgh and clinical Consultant in Obstetrics and Gynaecology at the Royal Infirmary, Edinburgh. Her University Personal Chair was awarded in 1999. Clinical research studies have focused upon local uterine mechanisms involved in menstruation and implantation. Projects have addressed key areas common to these reproductive processes, including injury and repair, endocrine- immune interactions and the role and regulation of inflammatory mediators. A particular area of interest has been the local endometrial response to withdrawal of progesterone, both physiological and pharmacological. Her fundamental studies on endometrial physiology in these areas have made a major contribution to the understanding of endometrial biology particularly regarding mechanisms regulating the onset of menstruation and unscheduled endometrial bleeding. Clinical areas of study include studies on assessment and evaluation of abnormal uterine bleeding and the descriptors for the contemporary complaint of heavy menstrual bleeding. Collaborative clinical studies with Paediatric Oncology and Paediatric Endocrinology have addressed the late effects of treatment of childhood cancer upon uterine and ovarian function.

Professor George Kollias
President and Director, Biomedical Sciences Research Centre 'Alexander Fleming', Greece

Dr. George Kollias, born in 1958, received his B.Sc. in Biology from the University of Athens, Greece and Ph.D. training from 1980 to 1984 in Molecular Biology at the National Hellenic Research Foundation. He has post-doctoral experience in the field of gene structure and expression (Laboratory of Gene Structure and Expression, National Institute for Medical Research, Mill Hill, London, UK) and from 1990 - 2000 he established the Laboratory of Molecular Genetics at the Hellenic Pasteur Institute in Athens, Greece. From 2000-2002, he was appointed Director of the Institute for Immunology at the Biomedical Sciences Research Center 'Al. Fleming', and from 2002 he was appointed the Center's President and Director.

Dr. Kollias has a sound expertise in transgenic, gene mutational and knockout technology, molecular and cellular biology and immunology. His interests are in the field of the role of cytokines in inflammation and autoimmunity and his lab is best known for transgenic and knockout research in the TNF field and for establishing and characterizing disease pathways in animal models for chronic inflammation and autoimmunity (e.g. Rheumatoid Arthritis, Inflammatory Bowel Disease, Multiple Sclerosis, endotoxic shock and systemic lupus).

He has published over 90 primary research articles in peer-reviewed journals and his work has received over 8000 citations. He has been the recipient of the 1993 International Prize-Award for achievements in "Molecular Biomedicine" under the scheme "International Scientific and Cultural Awards of the Bodosaki

Foundation" and from 2000, an elected member of the European Molecular Biology Organization (EMBO). His laboratory is supported by several competitive grants from European Commission and National sources, as well as by international biopharmaceutical industry.

Dr. Kollias serves as an advisor for scientific organizations and consults for industry. He also served as invited speaker and chairman and as a member of the Organising and Scientific Committees of several scientific meetings throughout the world. Dr. Kollias is also a regular member of the National Council on Research and Technology of the Ministry of Development (2001-2003 and 2005-present). Recently, Dr. Kollias was appointed coordinator of a consortium of 24 European organizations constituting a Network of Excellence on 'Functional Genomics in mutant mouse models as tools to investigate the complexity of human immunological disease' [6th Framework Program (2005-2009) – 11M].

Dr Connie Weyand
Division of Rheumatology, Emory University School of Medicine, Atlanta

CORNELIA M. WEYAND, M.D., Ph.D. obtained her M.D. degree from the University of Aachen in Germany. She graduated summa cum laude with a doctorate in Medicine from the University of Bonn in Germany in 1980. She received a doctorate in Medical Science from the University of Heidelberg in Germany in 1988. Her postgraduate training in Medicine was completed in the Department of Medicine at the Hannover Medical School in Germany and she was a Fellow in Rheumatology in the Division of Immunology at Stanford University, Stanford, California. From 1988 to 1989 she served as the Chief of Rheumatology at the University of Heidelberg before she joined the faculty of the Mayo Medical School in 1990. At Mayo she became the Barbara Woodward Lips Professor of Medicine and Immunology of the Mayo Medical and Graduate School and the Director of the Clinical Immunology and Immunotherapies Program. In 2004 she was recruited to Emory University where she currently is the David C. Lowance Professor of Medicine and Director of the Kathleen B. and Mason I. Lowance Center for Human Immunology at Emory University.

She has received numerous awards and honors including the Henry Christian Award for Excellence in Research (AFCR, 1991), the Henry Kunkel Young Investigator Award, American College of Rheumatology (1992), the Carol Nachtman Award for Rheumatology (1995), and The Mayo Foundation Outstanding Investigator Award (1999). She has been elected a member of the American Society for Clinical Investigation and the Association of American Physicians. Her research has lead to more than 270 scientific publications. She has edited several books, including a textbook on "Inflammatory Blood Vessel Diseases" and the "Primer on the Rheumatic Diseases". She is the Editor of Current Opinion in Rheumatology and has served on multiple editorial boards as well as study sections of the National Institutes of Health.

Professor Josef Smolen
Division of Rheumatology, Medical University of Vienna

Josef S. Smolen received his degree from the Medical Faculty of University of Vienna, Austria. He initially trained at the Institute of Immunology, University of Vienna and subsequently performed a residency in Internal Medicine at the 2nd Department of Medicine. From 1980 to 1981 he performed a research fellowship at the Arthritis and Rheumatism Branch, National Institutes of Health. He served as head of the Rheumatology Ward of the 2nd Department of Medicine, University of Vienna, from 1983 to 1989 and was then called as Chairman of the 2nd Department of Medicine – Center of Rheumatic Diseases at Lainz Hospital (today Hietzing Hospital), Vienna. In 1995 he followed a call to become Chairman of the Department of Rheumatology, Internal Medicine III, University of Vienna Medical School (today: Medical University of Vienna). In 2005 he was elected member of the Austrian Academy of Sciences. From 2003 to 2005 he was President of the European League Against Rheumatism and also served or serves in various functions in national and international societies. He received several scientific awards and has been or is Editorial Board member of several professional Journals.

His major scientific interests with respect to basic and translational research are in the pathogenesis of autoimmune and rheumatic diseases with particular focus on autoimmunity as well as cellular and humoral pathways of inflammation and destruction, especially in rheumatoid arthritis and systemic lupus erythematosus. His clinical scientific interests are devoted mainly to outcomes research and clinical trials.

Professor Caroline Savage
Institute of Biological Research, University of Birmingham

Caroline Savage is Professor of Nephrology at the University of Birmingham and Honorary Consultant Physician at University Hospitals Birmingham, NHS Foundation Trust. She qualified from the Royal London Hospital Medical School in 1978 and trained in renal medicine at Hammersmith Hospital, London. She has a specific clinical and laboratory interest in immunologically mediated kidney diseases. Professor Savage is also Programme Director of the Wellcome Trust Clinical Research Facility in Birmingham.

Professor Timothy Williams
National Heart and Lung Institute, Imperial College London

Professor Williams is the Asthma UK Professor of Applied Pharmacology at the National Heart & Lung Institute of Imperial College London, a position he has held since 1988. He is currently the Head of the Leukocyte Biology Section and Deputy Director of the MRC and Asthma UK Centre in Allergic Mechanisms of Asthma. He formerly held positions at the CRC, Northwick Park, the Department of Pharmacology at the Royal College of Surgeons of England, and the Kennedy Institute of Rheumatology. He has a BSc in Physiology and PhD in Pharmacology from University College London. His interest is in inflammatory mechanisms, with an emphasis on those involved in the recruitment of leukocytes to sites of inflammation.

Professor Andreas Radbruch
Scientific Director, Deutsches Rheuma-Forschungszentrum, Berlin

Professor Radbruch is currently the Scientific Director of the Deutsches Rheuma-Forschungszentrum in Berlin as well as Professor of Rheunatology at the Humboldt University in the same city. He was previously Associate Professor for Genetics and Immunology at the University of Cologne. A biologist by education, Andreas Radbruch works on the immune system, and the way it provides immunity or participates in immunopathology. In particular, his work aims at a molecular understanding of the control of immune reactions and immunological memory in vaccination, and autoimmune and allergic inflammation. In early work on the regulation of antibody class switching in B lymphocytes, he demonstrated antibody class switch recombination in human memory B lymphocytes and murine antibody secreting plasma cells, demonstrating the role of switch recombination in vivo. He could show that switch recombination is controlled by cytokines from T lymphocytes. Consequently he then focussed on how expression of those cytokines is controlled in T lymphocytes. In recent and ongoing work, his group analyses the molecular basis of expression control of interleukin-4, -10 and interferon-gamma. They have identified a genetic element controlling the imprinting of the interleukin-4 gene for memory expression (Tykocinski et al. Biol Chem. 2005;280(31):28177). Currently, the group is analysing the mechanism of imprinting of cytokine genes in molecular detail. In a broader approach towards a molecular understanding of memory T lymphocytes, the group has identified by transcriptome analysis genes expressed in repeatedly activated T cells, which allow these cells to survive and resist physiological regulation in chronic inflammation. Likewise, the group is working on the biology of plasma cells which confer the humoral immunological memory, i.e. the protection provided by serum antibodies specific for antigens encountered in the past, but also pathogenic antibodies in autoimmunity and allergy. The group developed a new concept of conditional survival of longlived plasma cells (Radbruch et al., Nat Rev Immunol. 2006;(10):741). Both lines of research aim at developing "Cell Therapies" for the targeted elimination of memory T cells and memory plasma cells driving chronic inflammation, allergy and rheumatic diseases, and being resistant to physiological regulation. This strategy is also reflected in the technological developments originating from the group. This group has developed high-gradient magnetic cell sorting (MACS), with Miltenyi Biotech being a spin-off company. Another Biotech company originating from this group is AMAXA, which is developing technologies for the efficient introduction of nucleic acids into primary eukaryotic cells.

Professor Chris Haslett OBE FRSE
Head, Division of Clinical Sciences and Community Health and Associate Dean, University of Edinburgh

Professor Christopher Haslett OBE, FRSE, FMedSci, FRCP, FRCPE is Director of the Queen's Medical Research Institute, Professor of Respiratory Medicine, Director of the Rayne Laboratories at the University of Edinburgh and Director of one of the University's world-class clinical research centres, the MRC Centre for Inflammation Research. In addition, Chris is also an active clinician in his capacity as Honorary Consultant Physician at the

Royal Infirmary of Edinburgh. His key research interests are: Mechanisms of the Resolution of Inflammation; Granulocyte Apoptosis and Imaging the Inflammatory Response (Chris is spearheading Edinburgh's major research initiative into the impact of Imaging Technologies in Clinical Research) and was honoured in 2004 with an OBE for his services to Medical Research.

Professor Marc Feldmann FRS,
Kennedy Institute of Rheumatology Division, Faculty of Medicine, Imperial College London

Professor Feldmann is the Head of the Kennedy Institute of Rheumatology Division, Faculty of Medicine, Imperial College London. His chief interest is the molecular pathogenesis of inflammatory and rheumatic disease, with a special interest in the role of cytokines in rheumatoid arthritis. His major accomplishment has been to discover the important role of TNF in the pathogenesis of rheumatoid arthritis, this has led to the award, jointly with Professor Sir Ravinder Maini of the Crafoord Prize of the Royal Swedish Academy of Sciences in 2000, the Albert Lasker Clinical Medical Research Award in 2003 for the discovery of anti-TNF therapy as an effective treatment for rheumatoid arthritis and other autoimmune diseases, and more recently the Japanese Rheumatism Foundation International Rheumatism Arthritis Award 2007 for the discovery of anti-TNF treatment as an effective treatment for rheumatoid arthritis and other related diseases. On 18th April 2007 he received the 'European Inventor of the Year' Award in the "Lifetime Achievement" category for his work and inventions in developing treatments for autoimmune diseases/anti-TNF therapy.

Professor Ian McInnes
Division of Immunology, Infection and Inflammation, Glasgow Biomedical Research Centre

Studied medicine in the University of Glasgow graduating with honours in 1989 before training in internal medicine and rheumatology, completing MRCP in 1992 and becoming FRCP in 2003. Trained in PhD and post doctoral studies via fellowships from The Wellcome Trust, ARC(UK) and NIH Fogarty Fellowship Programme in NIH in both Glasgow and in the National Institutes of Health in Bethesda, MD. He is now Professor of Experimental Medicine / Honorary Consultant Rheumatologist in the Centre for Rheumatic Diseases University of Glasgow. His research interests include understanding the role of cytokines in inflammatory synovitis, both from the basic perspective of functional activity and using a translational medicine approach whereby such molecules also offer therapeutic utility. He leads a trials unit specialising in the use of biologic agents in early clinical trials in inflammatory arthritis. Recently these studies have extended to include the role of inflammation in promoting vascular disease, particularly atherogenesis. He has published widely in the immunobiology and rheumatology field. His work, together with that of his colleagues in University of Glasgow, has been recognised in receipt of the Michael Mason Prize 2001, from the British Society for Rheumatology and in the Albrecht Hasinger Lectureship 2002, Berlin, Germany. He is currently Chairman of the EULAR Scientific Committee having served since 2005 in this capacity.

Professor Lars Klareskog
Rheumatology Unit, Karolinska Institute, Sweden

Lars Klareskog is since 1993 professor of Rheumatology at Karolinska Institutet at Karolinska University Hospital, Stockholm Sweden. His major research interests are in the etiology and pathogenesis of rheumatoid arthritis and other inflammatory joint diseases, as well is in therapy and prevention towards these diseases. He is currently head of the Rheumatology Research Unit at Karolinska. Previous positions include chairmanship of clinical immunology Uppsala University, and chairmanship of the Department of Medicine at Karolinska University Hospital. He is also member of the Nobel Assembly at the Karolinska Institutet, and member of the Executive Board of Eular (European League against Rheumatism).

Professor Adriano G Rossi
MRC Centre for Inflammation Research, University of Edinburgh

After obtaining a BSc (Hons) and PhD in Pharmacology at the University of Glasgow, I carried out postdoctoral research at Wake Forest University, Winston-Salem, North Carolina, USA in Professor Joseph O'Flaherty laboratory. I returned to the UK gaining a Wellcome Trust Post-doctoral fellowship which led to a lectureship in Professor Tim William's department at the National Heart & Lung Institute, University of London. I then moved to Edinburgh and joined Professor Chris Haslett's Respiratory Medicine Unit at the University of Edinburgh as an

MRC funded senior scientist. I currently hold a Personal Chair in Respiratory and Inflammation Pharmacology in the MRC Centre for Inflammation Research at the Queen's Medical Research Institute at the University of Edinburgh. My major research aims are to gain a better understanding of the mechanisms controlling the initiation, progression and resolution of the inflammatory response with a view to help develop novel therapies for chronic inflammatory diseases.

Dr David Jayne
Dialysis Centre, Addenbrooke's Hospital, Cambridge

David Jayne is the Director of the Vasculitis and Lupus Clinic and a Consultant Nephrologist at Addenbrooke's Hospital, Cambridge. Dr Jayne gained an MBBChir in 1981 and an MD in 1993 from the University of Cambridge and became an FRCP in 1999. Between 1990 and 1995, he was employed as a Clinical Research Fellow at Gonville and Caius College, Cambridge, with a spell as Clinical Fellow in Nephrology at Harvard Medical School, Boston, USA in 1992-93. He then worked as a Senior Lecturer in Nephrology at St George's Hospital Medical School, London, from 1996 until 2001, when he took up his present post. He is the Co-ordinator of the European Vasculitis Study Group, and has been the lead investigator of various international, multicentre trials of immunosuppressive and immunotherapeutic drugs in vasculitis and lupus. His research interests include conducting clinical trials in vasculitis and lupus, and of immunotherapy for autoimmune renal disease. Other interests are identification of biomarkers in vasculitis and lupus, and investigation of and intervention in inflammatory vascular disease.

Professor George Schett
Department of Medicine, University of Erlangen-Nuremberg, Germany

Professor Schett studied medicine in the University of Innsbruck, graduating in 1994 before entering a post-doc research fellowship at the Institute for BioMedical Aging Research of the Austrian Academy of Science from 1994-1996 in Innsbruck. Training in internal medicine and rheumatology from 1996-2001 at the Department of Internal Medicine 3 of the Medical University, Vienna becoming a specialist for Internal Medicine in 2001 and for rheumatology in 2003. Research scientist at the pharmaceutical industry in 2004. Assisting professorship at the Medical University of Vienna from 2002-2003 and from 2005-2006. He is now Professor of Internal Medicine and Chairman of the Department for Internal Medicine 3 at the University Erlangen-Nuremberg since 2006. His research interests include understanding the interaction between inflammation and bone, both from the basic and translational science aspect. In particular he is interested to define the molecular pathways inflammation as using to degrade bone and to infect the architectural changes of joints. He has published widely in the fields of immunology and rheumatology and his work has been recognized in receipt of the START prize of the Austrian Ministry of Sciences in 2002.

Appendix Three
Participant List

Miss Abeer Al-Omair
PhD Student
University of Glasgow

Dr Esther Nicole Amft
Consultant Rheumatologist
NHS Lothian

Miss Elinor Anderson
PhD Student
University of Glasgow

Mr Darren Lee Asquith
Post Graduate Researcher
University of Glasgow

****Professor D T Baird CBE FRSE**
Immediate Past Chairman
Caledonian Research Foundation

Miss Lucy Ballantine
PhD Research Student
University of Glasgow

Miss Paula Beaumont
PhD Student
University of Edinburgh

Dr Laura Bence
Chief Scientist
Reactivlab Ltd

Dr Robert Benson
Post Doc
University of Strathclyde

Dr Emma Bermingham
Post-Doctoral Scientist
AG Research Ltd

Mr Stylianos Bournazos
PhD Student
University of Edinburgh

Miss Irini Bournazou
PhD Student
University of Edinburgh

Miss Jaqueline Brandon
Teacher

Miss Bei Lei Cai
PhD Student
University of Glasgow

Mrs Mary Cameron

Miss Jenna Cash
DPHIL Student
Sir William Dunn School

Dr Karen Chapman
Reader
University of Edinburgh

Dr Ian David Chapman
Development Director
Fulcrum Pharma Developments Ltd

Dr Nadine Clemo
Research and Development
GlaxoSmithKline

Professor J R Coggins FRSE
Vice-Principal for Life Sciences and Medicine
University of Glasgow

Dr Bryan Conway
Senior Lecturer
University of Edinburgh

Mr Emmanuel Coste
PhD Student
Western General Hospital

Miss Lydia Coulthard
PhD Student
St James University Hospital, Leeds

Mrs Agnes Couthino
PhD Student
University of Edinburgh

Dr Anne Crilly
Post Doctoral Research Scientist
University of Glasgow

***Professor H O D Critchley**
Professor of Reproductive Medicine
University of Edinburgh

Dr Julia Dorin Senior Scientist
MRC Human Genetics Unit

Dr Ian Downing
Clinical Research Scientist
Scottish National Blood Transfusion Service

Professor David Eckersall
University of Glasgow

Dr Clett Erridge
Research Fellow, University of Strathclyde

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Dr Lars Erwig Senior Lecturer
University of Aberdeen

Ms Lynsey Fairbairn
Research Assistant
MRC Centre for Inflammation Research

Mrs Christine Farquhar
Senior Research Scientist, BBSRC

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Kennedy Institute of Rheumatology Division,
Faculty of Medicine, Imperial College london

Dr David Ferenbach
Clinical Training Fellow
University of Edinburgh

Dr Zelandia Fermin
University of Glasgow

Dr Paul Fitch
Post-Doc, University of Edinburgh

Dr Sue Fleetwood-Walker
Chair of Sensory Neuroscience
University of Edinburgh

Dr Stewart Fletcher

Professor Sir Patrick Forrest FRSE
Professor Emeritus (Surgery)
University of Edinburgh

Miss Sarah Fox
PhD Student, QMRI

Dr Sandra Franz
Post-Doc, University of Edinburgh

Dr Alasdair Fraser
Research Fellow, University of Glasgow

Dr Babunilayam Gangadharan
Post Doctoral Scientist, Roslin Institute

Professor Peter Ghazal
Head of Division of Pathway Medicine
University of Edinburgh

Miss Ashley Gilmour
University of Glasgow

Dr Bridget Glaysher
Scientific Officer, University of Dundee

Dr Carl Goodyear
Lecturer, University of Glasgow

Dr Alastair Gracie
Senior University Teacher, University of Glasgow

Mrs Heather Griggs-Hardie
Medical Herbalist, Aucuparia Botanicals

****Professor C Haslett OBE FRSE**
Head, Division of Clinical Sciences and
Community Health and Associate Dean
(Research), University of Edinburgh

Dr Bret Heale Post Doc
MRC Human Genetics Unit

Dr Barbara Hebeis
Principal Scientist, GlaxoSmithKline

Dr Nik Hirani
Senior Clinical Researcher
MRC Centre for Inflammation Research

Dr Marielle Hoeve
Post-Doc, University of Edinburgh

Dr Rob van't Hof
Senior Lecturer, Western General Hospital

Professor Sarah Howie
Professor of Immunopathology
University of Edinburgh

Dr Axel Hueber
Clinical Research Fellow, University of Glasgow

***Dr David Jayne FRCP**
Dialysis Centre
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Mr Dominic Jones
PhD Student, University of Leeds

Dr Mick Kadlubowski
Principal Clinical Scientist
Scottish National Blood Transfusion Service

Dr Alison Kerr
MD Biosciences

Dr David Kilpatrick
Consultant Immunologist
Scottish National Blood Transfusion Service

Miss Vicky King
PhD Student
University of Glasgow

Ms Gillian Kinnear
Research Associate, UCB Group

* Denotes Speaker ** Denotes Chairperson

Miss Tiina Kipari
Research Assistant, QMRI

***Professor Lars Klareskog**
Rheumatology Unit
Karolinska Institute, Sweden

***Professor George Kollias**
President and Difrector,
Biomedical Sciences Research Centre
'Alexander Fleming', Greece

Mr Czeslaw Kruk
Physiotherapist

Dr Mariola Kurowska-Stolarski
Post Doctoral Researcher
University of Glasgow

Mr Paul Lacaze
PhD Student, University of Edinburgh

Dr Sue Lannan
Herbal Medicine Lecturer, Napier University

Dr Pauline Last

Professor F T Last OBE FRSE

Miss Andrea Leitch
Senior Technician, MRC Human Genetics Unit

Dr Andrew Leitch
Clinical Research Training Fellow
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Mr Yubin Li
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Miss Hsin-Ni Li
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****Professor F Y Liew FRSE**
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Dr Shirley MacDonald
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Dr Sarah Mackie
Clinical Research Fellow
St James's University Hospital, Leeds

Miss Jay MacKinnon
Medical Herbalist
National Institute of Medical Herbalists

Professor R M Maizels FRSE
Professor of Zoology, University of Edinburgh

Miss Rebeccah Mathews
PhD Student, St James University Hospital, Leeds

Miss Mairi McGrath
PhD Student, University of Glasgow

***Professor Ian McInnes**
Professor of Experimental Medicine
and Rheumatology, University of Glasgow

Dr Gordon Meiklejohn
MD Biosciences

Dr Alirio Jose Melendez-Romero
Senior Lecturer, University of Glasgow

Sylvia Michlewska
PhD Student, QMRI

Dr Ashley Miller
Post Doc, University of Glasgow

Dr Stuart Milling
Lecturer, University of Glasgow

Dr Akio Mitani
Visiting Fellow, University of Glasgow

Mr Damian Mole
Clinical Lecturer in Surgery
University of Edinburgh

Mr Mark Moore
PhD Student, University of Glasgow

Dr Rong Mu
University of Glasgow

Mr Debayan Mukherjee
Research Assistant, University of Dundee

Dr Wanda Niedbala
Research Fellow, University of Glasgow

Dr Jagtar Nijjar

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PhD Student

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Dr Lorna Proudfoot
Lecturer, Napier University

Dr Bjorn RabePost Doc
MRC Human Genetics Unit

***Professor Andreas Radbruch**
Scientific Director
Deutsches Rheuma-Forschungszentrum,
Berlin (DRFZ)

Miss Jillian Rennie
Technician, CIR QMRI

Miss Natalie Louise Reynolds
PhD Student, MRC Human Genetics Unit

Dr Joyce E. Richardson
Retired, Consultant Paediatrician

Miss Rachel Rigby
Student, MRC Human Genetics Unit

Ms Nikki Riley
PhD Student
MRC Centre for Inflammation Research

Miss Catherine Risley

Miss Sara Rodriguez-MartinPhD
Student, University of Edinburgh

***Dr Adriano Rossi**
MRC Centre for Inflammation Research
University of Edinburgh

***Professor Caroline Savage**
Institute of Biological Research
University of Birmingham

***Professor George Schett**
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University of Erlangen-Nuremberg, Germany

Miss Tara Sheldrake
Research Assistant, University of Edinburgh

Professor Hamish Simpson
Professor of Orthopaedics and Trauma
University of Edinburgh

Mrs Morag E. Smith
Retired, Optometrist

***Professor Josef Smolen**
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McLeod/ Arc Professor of Rheumatology
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