

## Professor Andy Clark BA (Cantab), PhD

Professor of Inflammation Biology

School of Immunity and Infection

### Contact details

**Telephone** [+44 \(0\)121 371 3244 \(tel:+44 121 371 3244\)](tel:+44%20121%20371%203244)

**Email** [a.r.clark@bham.ac.uk \(mailto:a.r.clark@bham.ac.uk\)](mailto:a.r.clark@bham.ac.uk)

Centre for Translational Inflammation Research  
The University of Birmingham Research Labs  
The New Queen Elizabeth Hospital Birmingham  
Mindelsohn Way  
University of Birmingham  
Edgbaston  
Birmingham  
B15 2TT  
UK

### About

Andy Clark is Professor of Inflammation Biology at Birmingham University.

Andy has published over sixty research papers, reviews and book chapters in the fields of transcriptional and post-transcriptional control of gene expression during inflammation. A number of his primary research articles have been cited several hundred times. Andy has received major funding from Wellcome, the Medical Research Council and Arthritis Research UK.

His research focuses on the intracellular mechanisms by which pro-inflammatory signalling pathways are switched off and the production of inflammatory mediators is made to cease. The underlying philosophy is that a better understanding of negative regulation of inflammation may provide clues to why (and where) chronic inflammation develops. It may also suggest more effective ways of treating rheumatoid arthritis and other diseases. For example, he and his colleagues have discovered that glucocorticoids exert anti-inflammatory effects by inducing expression of a phosphatase enzyme that can switch off pro-inflammatory signalling pathways. It may be possible to capitalise on this discovery by finding ways to increase or prolong the expression of this phosphatase, so augmenting the anti-inflammatory effects of glucocorticoids.

### Qualifications

- PhD, Department of Medicine, University of Birmingham
- BA Natural Sciences, University of Cambridge, UK

### Biography

Andy qualified with a BA Natural Sciences, University of Cambridge, UK in 1987. In 1992 he successfully completed a PhD in Medical Molecular Biology at the University of Birmingham before gaining a Redcliffe-Maud post-doctoral fellowship from Diabetes UK. He then worked as a postdoctoral research fellow for Cancer Research UK before taking up a permanent academic appointment at the Kennedy Institute of Rheumatology, Imperial College London where he worked as Lecturer, Senior Lecturer and then Reader in Cell Signalling. In March 2012 he was appointed Professor of Inflammation Biology at the University of Birmingham.

Andy is an experienced molecular biologist with a strong track record in the study of gene regulation during inflammatory responses. His particular interest is negative feedback mechanisms that either limit inflammatory responses or promote their resolution. He has published many influential and widely-cited papers on the post-transcriptional control of inflammatory gene expression, and on anti-inflammatory mechanisms of glucocorticoids.

Andy is an associate editor of the Journal of Endocrinology, and acts as referee for many other journals (including Nature, Molecular Cell, Oncogene, EMBO Journal, Journal of Immunology, Journal of Biological Chemistry etc.). He also referee grants for BBSRC, ESRC, MRC, Wellcome Trust and other funding bodies. He has been an invited speaker at several national and international meetings. He also has well established international collaborations with colleagues in Australia, Germany, Canada.

### Postgraduate supervision

Andy is interested in supervising doctoral research students in the following areas:

- Anti-inflammatory mechanisms of glucocorticoids.
- Transcriptional or post-transcriptional control of cytokine gene expression.

If you are interesting in studying any of these subject areas please contact Andy at the contact details above, or for any general doctoral research enquiries, please email: [dr@contacts.bham.ac.uk \(mailto:dr@contacts.bham.ac.uk\)](mailto:dr@contacts.bham.ac.uk) or call +44 (0)121 414 5005.

For a full list of available Doctoral Research opportunities, please visit our [Doctoral Research programme listings \(http://www.bham.findaphd.com/?es=y&apl=y&aplt=&show\)](http://www.bham.findaphd.com/?es=y&apl=y&aplt=&show)

### Research

#### RESEARCH THEMES

Inflammation, Resolution, Rheumatology, Gene expression.

#### RESEARCH ACTIVITY

##### Control of mRNA stability

Inflammatory mediators are commonly expressed in a very transient manner; expression is rapidly increased in response to a pro-inflammatory challenge such as an

infectious pathogen, then quickly returns to baseline. This pattern of gene expression depends on the mRNAs that encode inflammatory mediators having short half-lives. It is crucial for ensuring timely resolution of inflammatory responses, and for preventing unprovoked or excessive inflammation. With former colleagues at the Kennedy Institute of Rheumatology, Andy helped to identify a mechanism by which the p38 mitogen-activated protein kinase signalling pathway controls the stability of many pro-inflammatory mRNAs. p38 modulates the expression, localisation and activity of a key mRNA destabilising protein known as tristetraprolin or TTP.

### Anti-inflammatory actions of glucocorticoids

Glucocorticoids are very widely used in the treatment of chronic inflammatory diseases such as rheumatoid arthritis and asthma, but their use is constrained by a large number of side effects. Major worldwide research efforts are focused on the molecular mechanisms that underlie the beneficial and harmful effects of glucocorticoids. An ultimate aim is to achieve anti-inflammatory effects without the usual burden of side effects associated with prolonged exposure to glucocorticoids. Andy identified Dual Specificity Phosphatase 1 (DUSP1) as a gene that is activated by glucocorticoids, and appears to be responsible for many of the anti-inflammatory effects of glucocorticoids.

## Publications

Clark AR, Belvisi MG. (2012) Maps and legends: the quest for dissociated ligands of the glucocorticoid receptor. *Pharmacol Ther.* 134: 54-67.

Joanny E, Ding Q, Gong L, Kong P, Saklatvala J, Clark AR. (2012) Anti-inflammatory effects of selective glucocorticoid receptor modulators are partially dependent on up-regulation of dual specificity phosphatase 1. *B J Pharmacol.* 165: 1124-1136.

Vattakuzhi Y, Abraham S, Freidin A, Clark AR, Horwood N. (2012) Dual specificity phosphatase 1 null mice exhibit spontaneous osteolytic disease and enhanced inflammatory osteolysis in experimental arthritis. *Arthritis and Rheumatism.* In press.

Ahasan M, Hardy R, Jones C, Kaur K, Hassan-Smith Z, Bénézec C, Caamaño JH, Hewison M, Lavery G, Rabbitt EH, Clark AR, Buckley CD, Raza K, Stewart PM, Cooper MS. (2012) Inflammatory regulation of cortisol metabolism in mesenchymal stromal cells. *Arthritis and Rheumatism.* In press.

Smallie T, Ricchetti G, Horwood NJ, Feldmann M, Clark AR, Williams LM. (2010). IL-10 inhibits the RelA-mediated release of paused RNA polymerase II at the TNF gene in human macrophages. *J Exp Med.* 207: 2081-2088.

Marchese FP, Aubareda A, Tudor C, Saklatvala J, Clark AR, Dean JLE. (2010). MK2 blocks tristetraprolin-directed mRNA decay by inhibiting CAF1 deadenylase recruitment. *J Biol Chem.* 285: 27590-27600.

Tchen CR, Martins JR, Paktiawal N, Perelli R, Saklatvala J, Clark AR. (2010). Glucocorticoid regulation of mouse and human dual specificity phosphatase 1 (DUSP1) genes: unusual cis-acting elements and unexpected evolutionary divergence. *J Biol Chem* 285: 2642-2652.

Abraham SM, Lawrence T, Kleiman A, Warden P, Medghalchi M, Tuckermann J, Saklatvala J, Clark AR. (2006). Antiinflammatory effects of dexamethasone are partly dependent on induction of dual specificity phosphatase 1. *J Exp Med* 203: 1883-1889.

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