University of Birmingham

Professor Christopher Buckley DPhil FRCP

Arthritis Research Campaign Chair Of Rheumatology

School of Immunity and Infection (/schools/immunity-infection/index.aspx)

Contact details

Telephone +44 (0)121 414 4383 (tel:+44 121 414 4383)

Email c.d.buckley@bham.ac.uk (mailto:c.d.buckley@bham.ac.uk)

Rheumatology Research Group
MRC Centre for Immune Regulation
School of Immunity and Infection
College of Medical and Dental Sciences
University of Birmingham
Edgbaston
Birmingham
B15 2TT
UK

About

Chris has made major contributions to scientific administration through his roles with the arthritis research UK (Chair of the Fellowship Committee and Clinical Studies Group on early inflammatory arthritis) as well as EULAR (Scientific Programme organizing committee). He is closely involved in the development of clinical academics and he is a strong and eloquent advocate for clinician scientists in several fora.

Qualifications

- FRCP Medicine London 2006
- DPhil. Medicine Oxford 1996
- B.A. Biochemistry (1st Class) Oxford198

Biography

Chris Buckley obtained his first degree (BA) in Biochemistry from the University of Oxford (1985) with subsequent undergraduate training in Medicine MBBS at the Royal Free Hospital, London (1990). Postgraduate Medical training MRCP (1993) in General Medicine at Hammersmith Hospital, London (Mark Walport, Dorian Haskard), and John Radcliffe Hospital, Oxford. DPhil (1996) arising from a Wellcome Training Fellowship with Professor J Bell and Dr D Simmons at the Institute Molecular Medicine, Oxford. In 1996, funded by a Wellcome Clinician Scientist Fellowship, he joined the Department of Rheumatology in Birmingham. In 2001 he was awarded an MRC Senior Clinical Fellowship and in October 2002 became ARUK Professor of Rheumatology, Division of Immunity and Infection in Birmingham

Teaching

Teaching Programmes

MBCHB (/undergraduate/courses/med/medicine.aspx) - 2nd 3rd and 4th Year

Postgraduate supervision

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

- The role of stromal cells in inflammation.

He is particularly keen on mentoring and supporting medical doctors early in their academic careers. He sits on the College's Integrated Clinical Academic Training committee
If you are interesting in studying any of these subject areas please contact Chris on the contact details above.

Research

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 the 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

For more information about Chris Buckley's research follow the links below:

BBC Radio 4 (2004) Inflammation
http://www.bbc.co.uk/radio4/science/frontiers_20041020.shtml (http://www.bbc.co.uk/radio4/science/frontiers_20041020.shtml)

BBC Radio 4 (2008) Inflamed Response
http://www.bbc.co.uk/radio4/inflamedresponse/pip/11voq/ (http://www.bbc.co.uk/radio4/inflamedresponse/pip/11voq/)

Publications

Association of T-zone reticular networks and conduits with ectopic lymphoid tissues in mice and humans.Link A, Hardie DL, Favre S, Britschgi MR, Adams DH, Sixt M, Cyster JG, Buckley CD, Luther SA.Am J Pathol. 2011 Apr;178(4):1662-75.

Why does chronic inflammation persist: An unexpected role for fibroblasts.Buckley CD.Immunol Lett. 2011 Feb 17. [Epub ahead of print] Performance of the 2010 ACR/EULAR criteria for rheumatoid arthritis: comparison with 1987 ACR criteria in a very early synovitis cohort.

Cader MZ, Filer A, Hazlehurst J, de Pablo P, Buckley CD, Karim R.Ann Rheum Dis. 2011 Feb 1. [Epub ahead of print]PMID: 21285117

Altered expression of microRNA-203 in rheumatoid arthritis synovial fibroblasts and its role in fibroblast activation.Stanczyk J, Ospelt C, Karouzakis E, Filer A, Raza K, Kolling C, Gay R, Buckley CD, Tak PP, Gay S, Kyburz D.Arthritis Rheum. 2011 Feb;63(2):373-81. doi: 10.1002/art.30115.

Utility of ultrasound joint counts in the prediction of rheumatoid arthritis in patients with very early synovitis.Filer A, de Pablo P, Allen G, Nightingale P, Jordan A, Jobanputra P, Bowman S, Buckley CD, Raza K.Ann Rheum Dis. 2011 Mar;70(3):500-7. Epub 2010 Nov 29.

Genome-wide association study of genetic predictors of anti-tumor necrosis factor treatment efficacy in rheumatoid arthritis identifies associations with polymorphisms at seven loci.Plant D, Bowes J, Potter C, Hyrich KL, Morgan AW, Wilson AG, Isaacs JD; Wellcome Trust Case Control Consortium; British Society for Rheumatology Biologics Register, Barton A.Arthritis Rheum. 2011 Mar;63(3):645-53. doi: 10.1002/art.30130.

Rheumatoid synovial fluid interleukin-17-producing CD4 T cells have abundant tumor necrosis factor-alpha co-expression, but little interleukin-22 and interleukin-23R expression.Church LD, Filer AD, Hidalgo E, Howlett KA, Thomas AM, Rapecki S, Scheel-Toellner D, Buckley CD, Raza K.Arthritis Res Ther. 2010;12(5):R184. Epub 2010 Oct

Monocytes/macrophages express chemokine receptor CCR9 in rheumatoid arthritis and CCL25 stimulates their differentiation.Schmutz C, Cartwright A, Williams H, Haworth O, Williams JH, Filer A, Salmon M, Buckley CD, Middleton J.Arthritis Res Ther. 2010;12(4):R161. Epub 2010 Aug 25.

Expertise

Factors involved in the triggering and perpetuation of chronic inflammatory arthritis, such as rheumatoid, reactive and juvenile arthritis; targets for effective future therapy; related systemic connective tissue diseases, particularly lupus

Alternative contact number available for this expert: **contact the press office (http://www.birmingham.ac.uk/news/contacts/index.aspx)**

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