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Dr David Withers PhD

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About

David Withers is a Wellcome Trust funded Research Fellow

His work is focused on CD4 T cell responses and the secondary lymphoid tissue within which these responses are initiated.

Qualifications

- PhD Immunology 2004
- BSc (Hons) Microbiology and Virology 2000

Biography

David Withers qualified with a BSc (Hons) in Virology and Microbiology from the University of Warwick in 2000. He went on to study for a PhD in Immunology at the Institute for Animal Health in conjunction with the University of Bristol. After obtaining his PhD, David continued his studies in the laboratory of Peter Lipsky at NIAMS, NIH, Bethseda (2004-2006). He then returned to the UK to study with Peter Lane at the University of Birmingham, cementing his interest in secondary lymphoid tissue development/structure and how this controlled CD4 T cell responses. In 2011 he was awarded a Wellcome Trust Career Development Fellowship to continue his research interests and establish his own research group

Teaching

Teaching Programmes

- **MBChB IIH (2nd year) (/students/courses/undergraduate/med/medicine.aspx)**
- **BMedSc3 ExpImm (/students/courses/undergraduate/med/medical-sci.aspx)**

Postgraduate supervision

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

- Generation and survival of memory CD4 T cells
- Secondary lymphoid tissue generation, organisation and maintenance

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

For a full list of available Doctoral Research opportunities, please visit our Doctoral Research programme listings.

Research

RESEARCH THEMES

T cell responses, Immunological memory, Secondary lymphoid tissue.

RESEARCH ACTIVITY

David's research is focused on understanding the signals involved in the development and maintenance of T cell responses, in particular, the development of memory CD4 T cells which are essential for developing immunological memory. Immunological memory underpins our strategies of vaccination. Understanding how memory CD4 T cells are generated and maintained is crucial for improving upon our ability to both vaccinate and modulate unwanted self reactivity. CD4 T cell responses are initiated within secondary lymphoid tissues such as lymph nodes. Understanding how these structures are formed and also maintained is also important for a full understanding of the response.

Key areas of research:

Memory CD4 T cell survival and function

To improve our ability to modulate memory CD4 T cells, greater understanding of the signals involved is required. Since any given T cell clone is present in very low numbers within the host, it is imperative to track low numbers of antigen-specific T cells within our experimental approaches. Using a combination of techniques including

MHCII tetramers and adoptive transfer of transgenic T cells, the responses of physiological cohorts of antigen specific CD4 T cells will be assessed.

Lymph node structure and function

The development of lymph nodes is dependent upon lymphoid tissue inducer cells, a specialised innate cell type that initiates the formation of these structures during embryonic development. Given that lymph node development is restricted to a window of embryonic development, it is somewhat surprising that these cells persist with adult lymph nodes, suggesting further functions within this tissue. Utilising different genetically modified mice, these cells can be removed from developed lymph nodes and the impact this has on lymph node function assessed.

Publications

Withers, D.R., Gaspal, F.M., Bekiaris, V., McConnell, F.M., Kim, M.-Y., Anderson, G., and Lane, P.J.L. (2011). Role of OX40 and CD30 in murine CD4 effector and memory function. Immunol Rev In press

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Li, Y., Innocentin, S., Withers, D.R., Roberts, N.A., Gallagher, A.R., Grigorieva, E.F., Wilhelm, C., and Veldhoen, M. (2011). Exogenous Stimuli Maintain Intraepithelial Lymphocytes via Aryl Hydrocarbon Receptor Activation. Cell.

Gaspal, F.M., Withers, D., Saini, M., Bekiaris, V., McConnell, F.M., White, A., Yagita, H., Walker, L.S.K., Anderson, G., and Lane, P.J.L. (2011). Abrogation of CD30 and OX40 signals prevents autoimmune disease in FoxP3 deficient mice. J Exp Med 208 1579-1584, 1579-1584.

Kim, S., Han, S., Withers, D.R., Gaspal, F., Bae, J., Baik, S., Shin, H.C., Kim, K.S., Bekiaris, V., Anderson, G., et al. (2011). CD117(+) CD3(-) CD56(-) OX40L(high) cells express IL-22 and display an LTi phenotype in human secondary lymphoid tissues. Eur J Immunol 41, 1563-1572.

Withers, D.R., Jaensson, E., Gaspal, F., McConnell, F.M., Eksteen, B., Anderson, G., Agace, W.W., and Lane, P.J. (2009). The survival of memory CD4+ T cells within the gut lamina propria requires OX40 and CD30 signals. J Immunol 183, 5079-5084.

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