University of Birmingham

Dr Lucy Walker BSc, PhD,

Honorary Reader in Immune Regulation and Autoimmunity

School of Immunity and Infection

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About

Lucy Walker holds an MRC Senior Non-Clinical Fellowship and is a Reader in Immune Regulation and Autoimmunity in the School of Immunity & Infection.

Lucy has published extensively in internationally recognised scientific journals in the area of T cell activation and regulation, particularly in the context of Type 1 Diabetes. She has received substantial grant support from a wide range of national and international funding bodies.

She gives frequent talks at scientific meetings and other research institutes, as well as to lay audiences with a personal interest in Type 1 Diabetes.

Qualifications

- PhD Immunology 1998
- BSc (Hons) Biology 1994

Teaching

Teaching Programmes

- **MBChB (/undergraduate/courses/med/medicine.aspx)**
- **MRes (/postgraduate/courses/combined/med/health-research.aspx)** Molecular and Cellular Medicine – theme lead for “Regulation of adaptive immune responses” module
- **Intercalating BMedSci (/undergraduate/courses/med/medical-sci.aspx)**
- **BMedSci (/undergraduate/courses/med/medical-sci.aspx)** - Experimental Immunology Module

Postgraduate supervision

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

- CD4 T cell responses, particularly in the context of Type 1 Diabetes.
- Influence of cytokines, such as IL-21, on regulatory T cell function
- Control of autoimmune diabetes by CD28 and CTLA-4.
- Contribution of different B cell subsets to autoimmune diabetes

If you are interesting in studying any of these subject areas please contact Lucy on the email address above.

Research

RESEARCH THEMES

Immune Regulation. T cell responses in Autoimmune Diabetes. Regulatory T cells. Cytokines. T and B lymphocytes.

RESEARCH ACTIVITY

Type 1 Diabetes

The incidence of Type 1 Diabetes is rising at a remarkable rate, particularly in children under the age of 5. The overarching aim of the Walker lab is to understand why certain individuals develop an immune response against pancreatic islet antigens that leads to this condition. We believe that a more precise understanding of the mechanisms underlying this immune response will provide new clues about how to halt the disease process. Diabetes research in the Walker lab focuses in particular on the areas highlighted below.

Regulatory T cells

Regulatory T cells (Treg) are a specialized population of T cells that have the capacity to downregulate immune responses. The activity of Treg is thought to be particularly important in preventing the induction of autoimmune diseases such as Type 1 Diabetes, Rheumatoid Arthritis and Multiple Sclerosis. The Walker lab has a long-standing interest in understanding how Treg populations are maintained and how they elicit their suppressive function. The group has been particularly interested in the role of co-stimulatory molecules such as CD28, and it’s homologue CTLA-4, in controlling Treg homeostasis and activity. We have recently published that Treg that are deficient for CTLA-4 are unable to control diabetes in a murine adoptive transfer model.

T cell differentiation

The phenotype of the T cells that drive Type 1 Diabetes is currently unclear. The Walker lab is pursuing this question using the DO11 x rip-mOVA mouse model of diabetes in combination with analysis of T cells isolated from the blood of Type 1 Diabetes patients (in collaboration with Dr Parth Narendran). We have found that particular cytokines, including IL-21, can have a profound effect on Treg function and we are interested in determining how the cytokine profile of diabetogenic T cells might influence local Treg suppression.

Contribution of B cells to Type 1 Diabetes

B cells can be seen to infiltrate the pancreatic islets of both mice and humans with Type 1 Diabetes. The Walker lab has found that B cell depletion can inhibit diabetes in the DO11 x rip-mOVA mouse model. We are interested in dissecting how particular B cell populations, including B1 cells, contribute to the disease process in Type 1 Diabetes.

Other activities

- Member of NC3Rs Grant Assessment Panel
- Member of Asthma UK Research Review Panel
- Member of MDS College Research Strategy Committee
- Member of Research Stategy Committee for the School of Immunity & Infection
- Member of Postdoctoral Training and Career Development Committee (Deputy lead for Immunity & Infection)

Publications

The emerging role of CTLA-4 as a cell extrinsic regulator of T cell responses (2011) Lucy S.K. Walker and David M. Sansom. Nature Reviews Immunology (in press)

Qureshi, O.S., Y. Zheng, K. Nakamura, K. Attridge, C.N. Manzotti, E.M. Schmidt, J. Baker, L.E. Jeffery, S. Kaur, Z. Briggs, T.Z. Hou, C. Futter, G. Anderson, L.S.K. Walker, and D.M. Sansom. (2011) Trans-endocytosis of CD80 and CD86: A molecular basis for the cell extrinsic function of CTLA-4. Science 332(6029):600-603

Ryan GA, Wang CJ, Chamberlain JL, Attridge K, Schmidt EM, Kenefeck R, Clough LE, Dunussi-Joannopoulos K, Toellner KM, Walker L.S.K. (2010) B1 cells promote pancreas infiltration by autoreactive T cells. Journal of Immunology 185(5):2800-7

Walker L.S.K. (2009) Regulatory T cells overturned: the effectors fight back. Immunology 126(4):466-74.

Schmidt, E.M., C.J. Wang, G.A. Ryan, L.E. Clough, O.S. Qureshi, M. Goodall, A.K. Abbas, A.H. Sharpe, D.M. Sansom, and L.S.K. Walker. 2009. CTLA-4 controls regulatory T cell peripheral homeostasis and is required for suppression of pancreatic islet autoimmunity.
Journal of Immunology 182:274-282.

Qizhi Tang, Lucy S.K. Walker and Jeffrey A. Bluestone. (2008) Regulating Treg Cells at Sites of Inflammation. Immunity 29 (4):512

Clough, L.E., C.J. Wang, E.M. Schmidt, G. Booth, T.Z. Hou, R. G.A., and Lucy S.K. Walker. (2008) Release from Treg-mediated suppression during the onset of tissue-specific autoimmunity is associated with elevated IL-21. Journal of Immunology 180:5393-5401

Lucy S.K. Walker, Anna Chodos, Mark Eggena, Hans Dooms and Abul K. Abbas. (2003) Antigen-specific proliferation of CD4+CD25+ regulatory T cells in vivo. Journal of Experimental Medicine 198(2):249-258.