

## Professor Adam Cunningham

Professor of Functional Immunity

**[School of Immunity and Infection \(/schools/immunity-infection/index.aspx\)](/schools/immunity-infection/index.aspx)**

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### About

Adam Cunningham is a researcher based in the College of Medical and Dental Sciences' School of Immunity and Infection. In addition he is part of the vaccinology and immunomodulation theme in the newly formed cross-College Institute of Microbiology and Infection and a member of the well established MRC Centre for Immune Regulation.

Adam's research primarily focuses on the how immune responses develop to vaccines and infection and how they impact on host immune homeostasis.

### Qualifications

- BSc (Hons) – 2.1 in Pathobiology 1991
- PhD - The detection, epidemiology and immunobiology of Chlamydia pneumoniae 1995

### Biography

Cunningham graduated from The University of Reading in 1991 with a 2.1 in Pathobiology. He was awarded his PhD in Southampton in 1995 on "The detection, epidemiology and immunobiology of Chlamydia pneumoniae", supervised by Prof Mike Ward. After a short stay in The Gambia working at the MRC Centre in Fajara, funded by the WHO, he came to Birmingham in the summer of 1995. His first post-doctoral position, as part of the (then) Glaxo Wellcome Action TB initiative, examined how Mycobacterium tuberculosis adapted to changes in oxygen tension, a common feature of granulomas and associated with conversion of tubercle bacilli to a persisting state. In 1999 he undertook a post-doctoral position with Prof. Ian MacLennan examining how antibody responses develop and are regulated. During this time Cunningham incorporated the use of Salmonella and its component antigens into this work. This led to his first independent position in Birmingham in January 2005 as a tenure-track RCUK Roberts Academic Fellow studying how immune responses develop to pathogens and vaccines. He was made Senior Research Fellow in October 2009, Senior Lecturer in June 2010, Reader in October 2010 and Professor of Functional Immunity in August 2011.

### Teaching

Cunningham teaches on the MBChB, BMedSc and postgraduate courses and also teaches qualified medics in various aspects of immunology and research. He has also successfully supervised PhD students in immunology and infection and is interested in hearing from individuals who wish to pursue a PhD.

- **[Medicine and Surgery MBChB \(/undergraduate/courses/med/medicine.aspx\)](/undergraduate/courses/med/medicine.aspx)**
- **[Medical Science BMedSc \(/undergraduate/courses/med/biomedical-science.aspx\)](/undergraduate/courses/med/biomedical-science.aspx)**

### Postgraduate supervision

Adam supervises doctoral research students in the areas of adaptive immunity, vaccine function, immunomodulation and immunity to infection.

If you are interested in studying any of these subject areas please contact Adam 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)** (mailto:dr@contacts.bham.ac.uk) or call +44 (0)121 414 5005.

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### Research

#### Themes:

B cells, T cells, generation and maintenance of adaptive responses, infection, vaccination, immune homeostasis, immune modulation

#### Lay summary

Our work aims to understand how infection changes who we are as individuals. Our work has a particular focus on how infection can educate the immune system and the specific elements and cells of the immune system that respond to pathogens. An exciting consequence of this work is that by comparing how we react to the whole pathogen or to individual components of the pathogen we can generate new vaccines to infections and new treatments to infectious and non-infectious diseases.

#### Overview

Our research uses in vivo models of infection to study how infection and vaccination modifies immune homeostasis, with a particular emphasis on adaptive immunity. Relating these changes to bacterial clearance helps identify how infections are controlled and how vaccines function. Furthermore, understanding the relationship between host and pathogen helps identify novel approaches to exploit bacteria and their components as prophylactics and therapeutics in infectious and non-infectious diseases (Fig. 1).

**Fig. 1. Studying host responses to pathogens and their components can help in the design of novel vaccines and treatments to infectious and non-infectious disease**

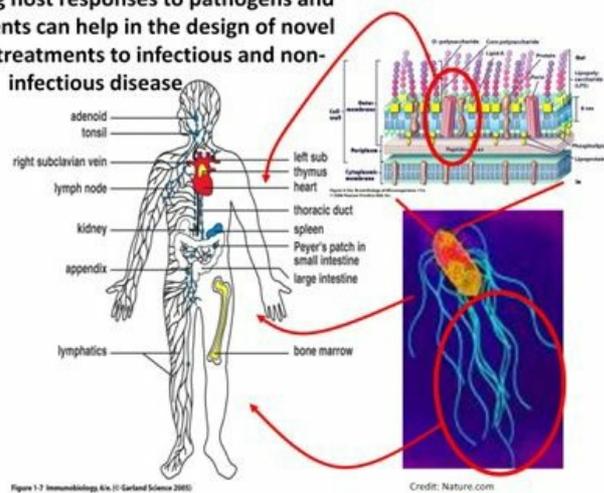


Figure 1-7 Immunobiology, 6/e, © Garland Science 2005

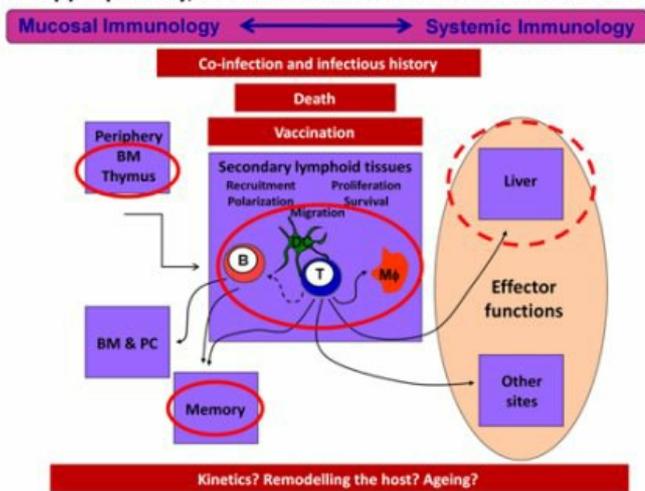
Examples of current themes ongoing in the laboratory include:

1. The development of vaccines against non-typhoidal Salmonella and the potential of B1b cells as targets for vaccination
2. How T and B cell responses are regulated after infection and vaccination
3. How lymphopoiesis is regulated in the thymus and bone marrow during infection
4. How bacteria and their components immunomodulate the host during infectious and non-infectious disease
5. How multiple and co-infections impact upon host homeostasis and immune function
6. How dormant tuberculosis infections in humans are maintained

Such investigations require studies of immune homeostasis in multiple anatomical locations concurrently, the different cellular subsets involved and the

kinetics of these events (Fig. 2)

**Fig. 2. Control of systemic infection requires co-ordination between multiple sites and cells to generate sufficient lymphocytes, activate them appropriately, to travel to sites of infection and direct killing**



Though this work is performed primarily in the context of infection and vaccination, with a particular focus on adaptive immunity and its direction by the innate immune system, the principles allow us to investigate how non-infectious disease impacts on the host.

Our work has shown that the immune system responds differently to the same antigen when presented in different immunological contexts and in different anatomical compartments. This is important since it means we can modify the response to an antigen or promote a particular host function simply by altering how we deliver antigen to the immune system. This capacity to modulate the host response to a pathogen or a component of a pathogen or a vaccine underlies our translational work (see publications). Lastly, using this knowledge we can exploit our findings to maintain and improve health as we age through directing and stimulating beneficial, long-term immune responses. Collectively, our work therefore helps understand how we can improve immunity to infectious and non-infectious disease.

### Other activities

- Reviewer for a number of high-impact, internationally-competitive, journals and funding agencies in the UK and abroad

- Panel member of the Royal Society Joint International Projects committee
- Organized a number of national and international meetings on microbiology, infection and immunology

### Publications

Ross E.A., Coughlan R.E., Flores-Langarica A., Lax S., Nicholson J., Marshall J.L., Bobat S., Hitchcock J., White A., Jenkinson W.E., Desanti G.E., Khan M., Henderson I.R., Lavery G.G., Buckley C.D., Anderson G. and **Cunningham A.F.** (2012). Thymic function is maintained during Salmonella-induced atrophy and recovery. *Journal of Immunology, In press*

Ross E.A., Coughlan R.E., Flores-Langarica A., Bobat S., Hussain K., Charlesworth J., Abhyankar N., Marshall J.L., Hitchcock J., Gil-Cruz C., López-Macías C., Khan M., Watson S.P., MacLennan I.C.M., Buckley C.D., and **Cunningham A.F.** (2011). CD31 is required on CD4<sup>+</sup> T cells to promote T cell survival and clearance of *Salmonella* infection. *Journal of Immunology*, 187: 1553-1565

Flores-Langarica A., Marshall J.L., Bobat S., Mohr E., Hitchcock J., Ross E.A., Coughlan R.E., Khan M., Van Rooijen N., Henderson I.R., MacLennan I.C.M., and **Cunningham A.F.** (2011). Recruited monocyte-derived dendritic cells collaborate with conventional DC to prime Th1 responses to *Salmonella*. *European Journal of Immunology*, 41: 2654-2665.

Bobat S., Flores-Langarica A., Hitchcock J., Marshall J.L., Kingsley R.A., Goodall M., Gil-Cruz C., Serre K., Leyton D.L., Letran S.E., Gaspal F., Chester R., Dougan G., López-Macías C., Henderson I.R., Alexander J., MacLennan I.C.M., and **Cunningham A.F.** (2011). Soluble flagellin, FliC, induces an antigen-specific Th2 response, yet promotes T-bet-regulated Th1 clearance of *Salmonella* Typhimurium infection. *European Journal of Immunology*, 41: 1606-1618

Lee S.K., Rigby R.J., Zotos D., Tsai L.M., Kawamoto S., Marshall J.L., Ramiscal R.R., Chan T.D., Gatto D., Brink R., Yu D., Fagarasan S., Tarlinton D.T., **Cunningham A.F.** and C.G. Vinuesa. (2011). B cell priming for extrafollicular antibody responses requires Bcl-6 expression by T cells. *Journal of Experimental Medicine*, 208: 1377-1388.

MacLennan C.A., Gilchrist, J.J., Gordon M.A., **Cunningham A.F.**, Cobbold M., Goodall M., Kingsley R.A., van Oosterhout J.J.G., Msefula C.L., Mandala W.L., Leyton D.L., Marshall J.L., Gondwe E.N., Bobat S., López-Macías C., Doffinger R., Henderson I.R., Zijlstra E.E., Dougan G., Drayson M.T., MacLennan I.C.M., and Molyneux M.E. (2010). Cobbold M., Goodall M., Kingsley R.A., van Oosterhout J.J.G., Msefula C.L., Mandala W.L., Leyton D.L., Marshall J.L., Gondwe E.N., Bobat S., López-Macías C., Doffinger R., Henderson I.R., Zijlstra E.E., Dougan G., Drayson M.T., MacLennan I.C.M., and Molyneux M.E. (2010). Dysregulated humoral immunity to nontyphoidal Salmonella in HIV- infected African adults. *Science*. 328: 508-512.

Gil-Cruz C., Bobat S., Marshall J., Kingsley R.A., Ross E.A., Henderson I.R., Leyton D.L., Coughlan R.E., Khan M., Jensen K.T., Buckley C.D., Dougan G., MacLennan I.C.M., López-Macías C., and **Cunningham A.F.** (2009). The porin OmpD from non-typhoidal Salmonella is a key target for a protective B1b cell antibody response. *Proceedings of the National Academy of Sciences USA*. 106: 9803-9808

**Cunningham A. F.**, Gaspal F., Serre K., Mohr E., Henderson I. R., Scott-Tucker A., Khan M., Toellner K.M., Lane P.J., and I.C. MacLennan. (2007). Salmonella induces a switched antibody response without germinal centers that impedes the extracellular spread of infection. *Journal of Immunology*. 178: 6200-07

