Dr Graham Taylor BSc PhD

Lecturer in Tumour Immunology

Contact details

Telephone +44 (0)121 414 7458 (tel:+44 121 414 7458)

Fax +44 (0)121 414 4486

Email g.s.taylor@bham.ac.uk (mailto:g.s.taylor@bham.ac.uk)

School of Cancer Sciences
University of Birmingham
Edgbaston
Birmingham
B15 2TT

About

Graham Taylor joined the School of Cancer Sciences in 2000 with a background in virology and has since developed a research portfolio studying the immune response to Epstein Barr Virus, a common human pathogen that is associated with a number of different types of cancer.

Part of this work has been to help develop a vaccine to treat patients with such cancers. The vaccine enhances the patient's immunity to particular EBV proteins present in the cancer cells. Two international phase 1 clinical trials have recently been completed and the results are very encouraging.

More recent activity has seen Graham broaden his research focus to include non virus-associated cancers (particularly bladder cancer) as well as studying EBV's role in autoimmune disease.

Qualifications

- PhD Virology 1998
- BSc Cell and Molecular Biology 1994

Biography

After graduating with a BSc (Hons) in Cell and Molecular Biology Graham went on to study for a PhD in Virology at the University of Warwick. Graham then worked as a clinical scientist in a front-line diagnostic virology laboratory before joining the

Teaching

- Cancer: Causes to Cures

Postgraduate supervision

Graham has supervised several PhD students and is interested in supervising doctoral research students in the following areas:

- **Antigen Processing and Presentation**
  Exploring the fundamental cellular processes by which cells generate peptides for recognition by T cells
- **Tumour Immunology**
  How the immune response targets tumours, how this process is usurped and how it can be harnessed for clinical benefit
- **Immune Responses to Virus Infections**
  Using Epstein-Barr virus, a common human infection, to understand better how the immune system deals with infection. Also, what is the role of EBV in the development of the autoimmune disease multiple sclerosis.

Research

The main aim of Graham's work is to increase our knowledge of the immune system in health and disease and how best to harness the immune system to treat cancer. Current research programmes in basic and translational research include the following.

Antigen Processing and Presentation

Graham's group are studying the basic biology of how antigens are processed and presented by MHC II molecules for recognition by CD4+ T cells – aiming eventually to manipulate these pathways to improve tumour cell killing.

Therapeutic Vaccination to Treat Cancer

Working with colleagues locally, nationally and internationally we have developed a therapeutic vaccine to treat nasopharyngeal carcinoma, a type of head and neck cancer that is associated with Epstein Barr Virus. Our vaccine has recently completed successful safety testing in phase I clinical trials and, following administration to patients, increases immune responses to EBV proteins that are present in the tumour cells. Subsequent trials of the vaccine are imminent.

Tumour Immunology

In collaboration with members of the Birmingham urological team Graham's team are studying the immunology of bladder cancer - a cancer that is all too common and also the most expensive per patient to treat. They are seeking ways to predict which patients will respond better to immune-based therapies so that clinicians can stratify patients better – that is, give patients the treatment that is most suited to treat their cancer. We are also researching the basic mechanisms how these treatmetns work so they can be further improved.

Role of Epstein Barr Virus in autoimmune disease

Finally, EBV infection is associated not only with certain types of cancer but also multiple sclerosis. A new project, in collaboration with neurologists, has been recently started to try and understand how EBV infection is linked to this autoimmune disease.

Other activities

- Peer reviewer for a range of research bodies and journals.

Publications

Leung, CS, Haigh TA, Mackay LK, Rickinson AB, Taylor GS. Nuclear location of an endogenously expressed antigen, EBNA1, restricts access to macroautophagy and the range of CD4 epitope display. Proc Nat Acad Sci (USA) 2010 107(5):2165-70.

Mackay LK, Long HM, Brooks JM, Taylor GS, Leung CS, Chen A, Wang F, Rickinson AB. T cell detection of a B-cell tropic virus infection: newly-synthesised versus mature viral proteins as antigen sources for CD4 and CD8 epitope display. PLoS Pathog. 2009 5(12):e1000699

Long HM, Zuo J, Leese AM, Gudgeon NH, Jia H, Taylor GS, Rickinson AB. CD4+ T cell clones recognising human lymphoma-associated antigens: generation by in vitro stimulation with autologous Epstein-Barr virus-transformed B cells. Blood. 2009;114(4):807-15

Haigh TA, Lin X, Hui EP, Chan ATC, Jia H, Rickinson AB and Taylor GS. CD4+ T cell clones specific for Epstein Barr Virus (EBV) latent membrane proteins 1 and 2 recognise EBV transformed B cell lines. J Immunol. 2008;180(3):1643-54.

Taylor GS, Long HM, Haigh TA, Larsen M, Brooks J, Rickinson AB. A role for intercellular antigen transfer in the recognition of EBV-transformed B cell lines by EBV nuclear antigen-specific CD4+ T cells.
J Immunol. 2006;177(6):3746-56.

Landais E, Morice A, Long HM, Haigh TA, Charreau B, Bonneville M, Houssaint E, Taylor GS . EBV-specific CD4+ T cell clones exhibit vigorous allogeneic responses. J Immunol. 2006;177(3):1427-33.

Long HM, Haigh TA, Gudgeon NH, Leen AM, Tsang CW, Brooks J, Landais E, Houssaint E, Lee SP, Rickinson AB, Taylor GS. CD4+ T-cell responses to Epstein-Barr virus (EBV) latent-cycle antigens and the recognition of EBV-transformed lymphoblastoid cell lines. J Virol. 2005;79(8):4896-907.

Taylor GS, Haigh TA, Gudgeon NH, Phelps RJ, Lee SP, Steven NM, Rickinson AB. Dual stimulation of Epstein-Barr Virus (EBV)-specific CD4+- and CD8+-T-cell responses by a chimeric antigen construct: potential therapeutic vaccine for EBV-positive nasopharyngeal carcinoma. J Virol. 2004; 78(2):768-78.