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About

Claire Shannon-Lowe is an MRC funded Research Fellow at the School of Cancer Sciences.

Claire has published research papers in scientific journals on the mechanism of Epstein Barr virus (EBV) entry into cell types lacking the virus receptor, including epithelial cells, T cells and NK cells. EBV infection of these cell types is strongly associated with various malignancies and lymphoproliferations. Claire has a research grant from the MRC and has been co-applicant on grants from the Association for International Cancer Research (AICR), Leukaemia and Lymphoma Research (LLR) and Cancer Research UK.

Qualifications

- PhD in Virology 2002, The Royal Free and University College Hospital
- MSc in Medical Microbiology 1997, The University of Surrey
- BSc Hons (2:1) in Applied Biology 1993

Biography

Claire Shannon-Lowe studied for a PhD in Virology at The Royal Free and University College Medical School in London where she worked on Cytomegalovirus resistance to antivirals in AIDS patients. She then went on to post doctoral research at the Cancer Research UK Institute for Cancer Studies at The University of Birmingham with Professors Henri-Jacques Delecluse, Alan Rickinson and Martin Rowe. In 2011 Claire was awarded a MRC New Investigator Award.

Her Laboratory focuses on the mechanisms used by Epstein Barr virus to gain access to cells not expressing the virus receptor, but following ectopic infection have a very strong association with various malignancies.

Teaching

- Tutor: MBChB, Cancer:Causes to Cures.

Postgraduate supervision

Claire Shannon-Lowe is supervising doctoral students in the following area:

The role of Epstein-Barr virus in non-B cell lymphomas

Research

Claire Shannon-Lowe's research has focused on the mechanisms of Epstein Barr virus (EBV) entry into EBV-receptor deficient cells including epithelial cells and lymphocytes (T- and NK cells). EBV efficiently infects resting B lymphocytes, which express the virus receptor CD21, and maintains a silent latent infection for the lifetime of the host. In a minority of patients, EBV is associated with malignancies of B cells including Burkitt's lymphoma and Hodgkin's lymphoma. However, EBV is not strictly B-lymphotropic, and EBV infection of epithelial, T- and NK cell lineages are significantly associated with lymphoproliferations and malignancies of these cell types (including nasopharyngeal and gastric carcinoma, haemophagocytic lymphohistiocytosis, NK leukaemia, extranasal NK/T cell lymphoma).

Epithelial cells, T cells and NK cells do not express the virus receptor and appeared to be refractory to infection *in vitro*. Dr Shannon-Lowe originally demonstrated efficient EBV entry into epithelial cells following co-culture of the epithelial cells with virus-loaded primary B cells. Based on this, her current research has two main focuses (1) EBV mediated manipulation of cell signalling processes to enable B cell-epithelial cell interaction and EBV-epithelial cell interaction (2) The mechanism of EBV entry into T- and NK cells and the role played by EBV in the development of T-/NK cell associated diseases and lymphomagenesis.

Publications

- Heath E, Begue-Pastor N, Chaganti S, Croom-Carter D, **Shannon-Lowe C**, Kube D, Feederle R, Delecluse HJ, Rickinson AB, Bell AI. **Epstein-barr virus infection of naïve B cells in vitro frequently selects clones with mutated immunoglobulin genotypes: implications for virus biology.** (http://www.ncbi.nlm.nih.gov/pubmed/22589726) PLoS Pathog. 2012 May;8(5):e1002697.
- Fox CP, **Shannon-Lowe C**, Rowe M. Deciphering the role of Epstein Barr virus in the pathogenesis of T and NK cell lymphoproliferations Herpesviridae. 2011 Sep 7;2:8.

- **Shannon-Lowe C** and Rowe M. Epstein-Barr virus infection of polarized epithelial cells via the basolateral surface by memory B cell-mediated transfer infection. PLoS Pathogens 2011 May;7(5):e1001338.
- Heather M. Long, Alison M. Leese, Odette L. Chagoury, Shawn R. Connerty, Jared Quarcoopome, Laura L. Quinn, **Claire Shannon-Lowe**, Alan B. Rickinson. Cytotoxic CD4+ T cell responses to Epstein-Barr virus contrast with CD8 responses in breadth of lytic cycle antigen choice and in lytic cycle recognition. J.Immunol 2011 Jul 1;187(1):92-101.
- Fox CP, Haigh TA, Taylor GS, Long HM, Lee SP, **Shannon-Lowe C**, O'Connor S, Bollard CM, Iqbal J, Chan WC, Rickinson AB, Bell AI, Rowe M. A novel latent membrane 2 transcript expressed in Epstein-Barr virus-positive NK and T cell lymphoproliferative disease encodes a target for cellular immunotherapy. Blood. 2010 Nov 11;116(19):3695-704.
- Fox CP, **Shannon-Lowe C**, Gothard P, Kishore B, Neilson J, O'Connor N, Rowe M.
- Epstein-Barr virus-associated hemophagocytic lymphohistiocytosis in adults characterized by high viral genome load within circulating natural killer cells. Clin Infect Dis. 2010 Jul 1;51(1):66-9.
- **Shannon-Lowe C**, Adland E, Bell AI, Delecluse HJ, Rickinson AB, Rowe M. Features distinguishing Epstein-Barr virus infections of epithelial cells and B cells: viral genome expression, genome maintenance, and genome amplification. J Virol. 2009 Aug;83(15):7749-60
- Croft NP, **Shannon-Lowe C**, Bell AI, Horst D, Kremmer E, Ressing ME, Wiertz EJ, Middeldorp JM, Rowe M, Rickinson AB, Hislop AD. Stage-specific inhibition of MHC class I presentation by the Epstein-Barr virus BNLF2a protein during virus lytic cycle. PLoS Pathog. 2009 Jun;5(6):e1000490