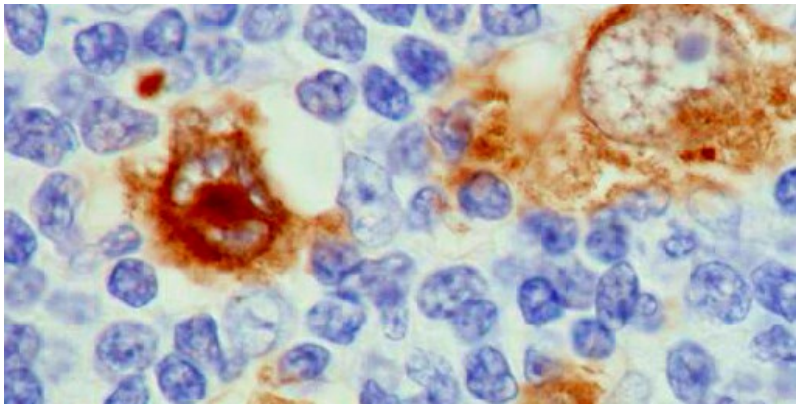
Pathogenesis of B cell lymphomas



Group leaders: **Professor Paul Murray (/staff/profiles/cancer/murray-paul.aspx)** , Professor Ciaran Woodman and **Professor Martin Rowe (/staff/profiles/cancer/rowe-martin.aspx)**

Overview

This research group is interested in understanding the pathogenesis of Hodgkin's lymphoma (HL) and diffuse large B cell lymphoma (DLBCL). It has a major interest in the contribution of the Epstein-Barr Virus (EBV) and its latent genes, as well as cellular events to the development of these lymphomas.

The research is centred around several themes;

1) EBV-induced aberrant B cell differentiation and its role in lymphoma development

2) Contribution of lipid signalling to the pathogenesis of lymphomas

3) The tumour micro environment of lymphomas

Our research group

Research in the B cell lymphoma group is investigating how the Epstein-Barr virus (EBV) contributes to the pathogenesis of lymphomas, particularly Hodgkin's lymphoma as well as diffuse large B cell lymphoma. The emphasis is on understanding those cellular signalling pathways that are aberrantly activated by EBV in B cells that could explain its transforming properties. With this knowledge we are developing and testing novel approaches to treat lymphoma patients.

The Murray group maintains important interactions with other research groups both within and outside the University of Birmingham These multidisciplinary arrangements include collaborations with epidemiologists, laboratory scientists and clinicians in the School of Cancer Sciences (Tanja Stankovic, Alan Rickinson, Martin Rowe, Pamela Kearns, Andrew Peet), Infection and Immunity (David Sansom) as well as in Public Health and Epidemiology (KK Cheng). Outside the University, there are also significant and productive long-term collaborations which include those with Professor Richard Ambinder (Johns Hopkins University School of Medicine, Baltimore), Professor Martin Allday (UCL, London), Professor Qian Tao, (Chinese University, Hong Kong) and Professor Kenneth Wright, University of Florida).

Open all sections

Current projects

- Regulation of B cell differentiation and EBV replication by B cell associated transcription factors (Vockerodt, Vrzalikova, Nagy)
- Modulation of EBV-induced transformation by the tumour microenvironment (Cader)
- Role of autotaxin and related molecules in driving EBV-induced B cell transformation(Lupino/Vrzalikova/Nagy)
- The contribution of oncogenic lipids to the microenvironment of Hodgkin's lymphoma (Vrzalikova)
- A study of cellular and virus gene expression in EBV-positive expanded germinal centres (Nagy)
- LPA and S1P and their roles in the development of primary CNS lymphoma (Lupino)
- New insights into the complex genetics of the Hodgkin/Reed-Sternberg cell (Wei/Mohamed/Kearns)

Recent publications

1. Vrzalikova K, Vockerodt M, Leonard S, Bell A, Wei W, Schrader A, Wright KL, Kube D, Rowe M, Woodman CB, Murray PG. Down-regulation of BLIMP1α by the EBV oncogene LMP1 disrupts the plasma cell differentiation program and prevents viral replication in B cells: implications for the pathogenesis of EBV-associated B cell lymphomas. *Blood* 2011; 117: 5907-17.

2. Leonard S, Wei W, Anderton J, Vockerodt M, Rowe M, Murray PG, Woodman CBJ. An investigation of the epigenetic and transcriptional changes which follow Epstein-Barr virus infection of germinal centre B cells and their relevance to the pathogenesis of Hodgkin's lymphoma. *J Virol* 2011; 85: 9568-77

3. Anderton JA, Bose S, Vockerodt M, Vrzalikova K, Wei W, Kuo M, Helin K, Christensen J, Rowe M, Murray PG, Woodman CB. The H3K27me3 demethylase, KDM6B, is induced by Epstein-Barr virus and over-expressed in Hodgkin's lymphoma *Oncogene*, 2011; 30: 2037-43.

4. Murray PG, Fan Y, Davies G, Ying J, Geng H, Ng KM, Li H, Gao Z, Kapatai G, Bose S, Anderton JA, Reynolds GM, Ito A, Woodman CBJ, Marafioti T, Ambinder RF, Tao Q. Epigenetic silencing of a proapoptotic cell adhesion molecule-the immunoglobulin superfamily member IGSF4 by promoter CpG methylation protects Hodgkin's lymphoma cells from apoptosis. *Am J Pathol* 2010; 177: 1480-90.

5. Bose S, Yap L-F, Fung M, Starzcynski J, Saleh A, Morgan S, Dawson C, Chukwuma MB, Maina E, Buettner M, Wei W, Arrand JR, Lim PVH, Young LS, Teo S-W, Stankovic T, Woodman CBJ, Murray PG. The ATM tumour suppressor gene is down-regulated in EBV-associated nasopharyngeal carcinoma. *J Pathol*, 2009; 217: 345-52.

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