

Dr Neil Hotchin BSc PhD

Senior Lecturer in Molecular Cell Biology
Director of Graduate Research, College of Life and Environmental Sciences

[School of Biosciences \(/schools/biosciences/index.aspx\)](/schools/biosciences/index.aspx)

Contact details

Telephone **+44 (0)121 41 45412** (tel: **+44 121 41 45412**)

Fax +44 (0)121 41 45925

Email n.a.hotchin@bham.ac.uk (mailto: n.a.hotchin@bham.ac.uk)

School of Biosciences
University of Birmingham
Edgbaston
Birmingham
B15 2TT
UK



About

Dr Hotchin is a cell biologist with an interest in normal epithelial cell function and in understanding the processes that contribute to diseases such as cancer. He has published a number of high impact papers on the role of small GTP-binding proteins in control of epithelial cell function. In addition to his research and teaching activities, Dr Hotchin is also currently Director of Graduate Research for the College of Life and Environmental Sciences.

Qualifications

BSc (University of York)

PhD (University of London)

Biography

Dr Neil Hotchin was born in Lincolnshire and did his first degree in Biology at the University of York. His PhD at the Royal Postgraduate Medical School (now part of Imperial College) was on the role of Epstein Barr Virus in the development of Burkitt's Lymphoma. After his PhD he worked as a postdoctoral Research Fellow at the Imperial Research Cancer Research Fund (now the Cancer Research UK London Research Institute) where he first became interested in how cell adhesion to extracellular matrix regulates cell function. He continued this work in the MRC Laboratory for Molecular Cell Biology at University College London before moving to Birmingham to set up his own research group.

Teaching

Dr Hotchin teaches cell biology on a number of first and second year courses and runs a final year module on Cancer Biology. He also teaches on a number of postgraduate level courses.

Postgraduate supervision

For a list of possible PhD projects offered by Dr Hotchin:

www.findaphd.com/search/customlink.asp?inst=birm-Biol&supersurname=Hotchin (<http://www.findaphd.com/search/customlink.asp?inst=birm-Biol&supersurname=Hotchin>)

Research

Research Theme within School of Biosciences: [Molecular and Cell Biology \(/research/activity/cellbiology/index.aspx\)](/research/activity/cellbiology/index.aspx)

The role of Rho family GTPases in regulation of cell function

Throughout the life of an organism cells must 'sample' their environment and take decisions accordingly. Cellular interactions with extracellular matrix proteins such as fibronectin, via integrin adhesion receptors, play an essential role in both developing and adult organisms. Many cellular functions require the integration of adhesion mediated signals with those received via growth factor receptors. Identifying the nature of these signals and the mechanism by which they are integrated is clearly essential to the understanding of how normal cellular function is regulated.

The Rho family of small GTP-binding proteins regulate both cell adhesion and growth factor mediated signal transduction. As such, they are believed to play a pivotal role in integration of adhesion and growth factor dependent signals. The main focus of our research is directed towards understanding how Rho family proteins regulate adhesion-mediated signal transduction events.

1. Regulation of Epithelial Cell Function by RhoFamily GTPases

Normal epithelial cell function is dependent on both cell-cell interactions mediated by cadherin adhesion receptors and interaction with extracellular matrix proteins via integrin adhesion receptors. There is good evidence that both of these adhesion events are regulated by members of the Rho family of small GTP-binding proteins. We are particularly interested in the role played by Rho-family members in regulating integrin-dependent signalling events in human epidermal keratinocytes.

2. The Role of Syntenin-1 in Trafficking of Frizzled Receptors in Breast Cancer

Frizzled (Fz) receptors are a group of seven-pass transmembrane receptors that play a key role in the transduction of signals from secreted ligands known as Wnts. Wnt signaling plays a central role in development, regulating proliferation, stem cell maintenance and cell fate decisions, as well as coordinating cell movements and the establishment of tissue polarity. Deregulated Wnt signaling is also a major contributing factor to epithelial carcinogenesis, including breast and colon cancer. Syntenin-1 is a membrane-associated adapter protein that we, and others, have shown interacts with a number of transmembrane and cytosolic proteins.

One of the main functions of syntenin-1 is the trafficking of membrane receptors to and from the plasma membrane. Increased expression of syntenin-1 is also associated with invasive behaviour in a number of breast cancer cell lines. In collaboration with Fedor Berditchevski and Chris Tselepis we are using a combination of biochemistry and cell biology to analyse the role of syntenin-1 in post-endocytic trafficking of Frizzled receptors in breast cancer cells.

3. The role of Rho GTPases in Glioblastoma

Glioblastoma is a common and aggressive brain tumour which is generally resistant to currently used therapies. In collaboration with Francesco Falciani and Roy Bicknell we are using a combination of bioinformatics and cell biology to analyse the role of Rho family GTPases in the development and spread of this tumour.

Other activities

Dr Neil Hotchin has been an Editor for PLoS ONE since 2007.

Publications

Hotchin NA, Cover TL and Akhtar N (2000). Cell vacuolation induced by the VacA cytotoxin of *Helicobacter pylori* is regulated by the Rac1 GTPase. *J. Biol. Chem.* **275**, 14009-14012.

Akhtar N and Hotchin NA (2001). RAC1 regulates adherens junctions through endocytosis of E-cadherin. *Mol. Biol. Cell* **12**, 847-862.

Sawada S, Yoshimoto M, Odintsova E, Hotchin NA and Berditchevski F (2003). The tetraspanin CD151 functions as a negative regulator in the adhesion-dependent activation of Ras. *J. Biol. Chem.* **278**, 26323-26326.

McMullan R, Lax S, Robertson VH, Radford DR, Broad S, Watt FM, Rowles A, Croft DR, Olson MF and Hotchin NA (2003). Keratinocyte differentiation is regulated by the Rho and ROCK signaling pathway. *Curr. Biol.* **13**, 2185-2189.

Turner FE, Broad S, Khanim FL, Jeanes A, Talma S, Hughes S, Tselepis C, Hotchin NA (2006). Slug regulates integrin expression and cell proliferation in human epidermal keratinocytes. *J. Biol. Chem.* **281**, 21321-21331.

Latysheva N, Muratov G, Rajesh S, Padgett M, Hotchin NA, Overduin M, Berditchevski F (2006). Syntenin-1 is a new component of tetraspanin-enriched microdomains: mechanisms and consequences of the interaction of syntenin-1 with CD63. *Mol. Cell Biol.* **20**, 7707-7718.

Brookes MJ, Boulton J, Roberts K, Cooper BT, Hotchin NA, Matthews G, Iqbal T, Tselepis C (2008). A role for iron in Wnt signaling. *Oncogene* **27**, 966-975.

Lock FE and Hotchin NA (2009). Distinct roles for ROCK1 and ROCK2 in the regulation of keratinocyte differentiation. *PLOS ONE* **4**, e8190.

Lim J, Hotchin NA, Caron E (2011). Ser756 of $\beta 2$ integrin controls Rap1 activity during inside-out activation of $\alpha M\beta 2$. *Biochem. J.* **437**, 461-467.

Lock FE, Ryan KR, Poulter NS, Parsons M, Hotchin NA (2012) Differential Regulation of Adhesion Complex Turnover by ROCK1 and ROCK2. *PLOS ONE* **7**, e31423.

Ryan KR, Lock FE, Heath JK, Hotchin NA. (2012). Plakoglobin-dependent regulation of keratinocyte apoptosis by Rnd3. *J. Cell Sci.* **125**, 3202-3209.

Lim J and Hotchin NA (2012). Signalling mechanisms of the leukocyte integrin $\alpha M\beta 2$: Current and future perspectives. *Biol. Cell* doi: 10.1111/boc.201200013.

Scales TM, Jayo A, Obara B, Holt MR, Hotchin NA, Berditchevski F and Parsons M (2012). $\alpha 3\beta 1$ integrins regulate CD151 complex assembly and membrane dynamics in carcinoma cells within 3D environments. *Oncogene* doi:10.1038/onc.2012.415.

Lim J, Thompson J, May RC, Hotchin NA and Caron E. (2013). Regulator of G-Protein Signalling-14 (RGS-14) regulates the activation of $\alpha M\beta 2$ integrin during phagocytosis. *PLOS ONE* **8**, e69163.

