

Dr Sally Roberts DPhil

Senior Lecturer

[School of Cancer Sciences \(/schools/cancer/index.aspx\)](/schools/cancer/index.aspx)

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About

Sally qualified with a B.Sc. (Hons) in Biochemistry from the University of Warwick in 1981. She gained a D.Phil. from the Laboratory of Molecular Biophysics in the University of Oxford in 1987 and was then appointed as a Research Fellow in the Department of Cancer Studies in the University of Birmingham. In 1999, she was appointed Lecturer in Cancer Studies and in 2002 promoted to Senior Lecturer. Whilst in Birmingham her research has focussed on the molecular biology of human papillomavirus – host interactions.

Qualifications

- DPhil University of Oxford 1987
- BSc (Hons) in Biochemistry University of Warwick 1981

Biography

Sally came to Birmingham in 1989 with a background in cell and molecular biology and she has since developed a research portfolio studying the molecular biology of human papillomaviruses (HPV), small DNA tumour viruses that cause a range of clinical manifestations ranging from anogenital warts and laryngeal papillomas to cancers at anogenital sites (e.g. cervical cancer), on the skin and in the oropharynx.

The molecular basis for such diverse pathogenesis lies in virus-host interactions necessary to support productive infection and Sally's programme of research focuses on understanding the virus life cycle and the contribution of virus-host interactions to the development of cancer.

Teaching

- BMedSc
- Clinical Oncology MSc/PG Dip Module Leader
- MBChB

Postgraduate supervision

Sally is interested in supervising doctoral research students in the following areas:

- The molecular biology of the human papillomavirus life cycle
- The role of human papillomavirus in the pathogenesis of anogenital and oropharyngeal cancers: host - virus interactions that contribute to malignancy

Research

Sally's HPV Research Group is interested in virus-host interactions that are important in the pathogenesis of human papillomavirus (HPV), a small DNA tumour virus that is the recognised cause of cancers within the anogenital tract, and more recently as contributing to the aetiology and pathogenesis of a subset of head and neck cancers. Sally's research is driven by the need to improve the understanding of the interactions between HPV and its human host with a view to identifying new therapeutic targets for the treatment of HPV associated disease.

Central to the programme of work is the use of cell models based on the host cell of the virus – the keratinocyte, whose stratification in organotypic raft culture enables recapitulation of the full replication cycle of the virus. Moreover, manipulation of these cell-based models reflects virus changes observed in the progression of HPV infections to cancer and thus allows investigation of virus-host interactions that contribute to the development of malignancy.

Molecular biology of the HPV life cycle

An important regulator of the HPV life cycle is the E4 protein and Sally's long-standing interest in E4 has revealed that the viral protein has diverse effects on cell behaviour and cellular organization, ranging from suppression of host cellular DNA synthesis and cell cycle dysregulation to the induction of reorganization of sub-nuclear ND10 domains – the potential sites for HPV replication. Her team's work has also shown that distinct biological actions of E4 arise through posttranslational modification of the protein and that this is likely to be a key mechanism to modify the E4 function during the virus life cycle. Emphasis is now on gaining insight into how these multiple actions of E4 contribute to its role in the virus life cycle since the development of a strategy to target E4 function may enable abrogation of virus propagation.

She is also using the cell-based models to understand the importance of the function of E6, one of the viral oncoproteins, to target cellular PDZ domain-containing proteins that regulate cell proliferation and cell polarity, to the replication cycle of high-risk HPV types.

HPV and cancer

HPV induced carcinogenesis is dependent upon interactions between the viral oncoproteins and a number of key cellular pathways controlling cell proliferation and survival. These include those regulated by a subset of cellular PDZ domain-containing proteins that are targets of the E6 oncoprotein. Sally's studies of this E6 function have been influential in understanding the contribution of E6 to cellular immortalization and to how regulation of this E6 function by cellular signalling pathways might contribute to an enhancement of the oncogenicity of the viral protein.

As well as using the cell-based models to understand the contribution of virus-host interactions to cervical carcinogenesis Sally is also developing cell-based systems that recapitulate the full spectrum of stages of oropharyngeal carcinogenesis; providing us with a unique opportunity to investigate the impact of HPV-host interactions on the development of cancer at this body site.

Epithelial cell biology

Sally's studies on cellular PDZ domain containing substrates of E6 such as the human homologue of the Drosophila tumour suppressor discs large (hdlg) has given insights into the post-transcriptional regulation of hdg function during terminal differentiation of squamous epithelia. A better understanding of the function of the alternative-splice variants that arise as a consequence of this process is now an important goal.

Publications

Knight, G. L., Pugh, A. G., Yates, E., Bell, I., Wilson, R., Moody, C. A., Laimins, L. A., Roberts, S. (2011) A cyclin-binding motif in human papillomavirus type 18 (HPV18) E1^{E4} is necessary for association with CDK-cyclin complexes and G2/M cell cycle arrest of keratinocytes, but is not required for differentiation-dependent viral genome amplification or L1 capsid protein expression. *Virology*, 412: 196-210 [cover article].

Collins, S. I., Constandinou-Williams, C., Roberts, S., Young, L.S., Woodman, C. B. J., Murray, P. G. (2010) Is human papillomavirus viral load a clinically useful predictive marker: a longitudinal study. *Cancer Epi. Bio. & Preven.* 19: 832-837.

Collins, S. I., Constandinou-Williams, C., Wen, K., Young, L.S., Roberts, S., Murray, P. G., Woodman, C. B. J. (2009) Disruption of the E2 gene is a common and early event in the natural history of cervical human papillomavirus infection: a longitudinal cohort study. *Cancer Research* 69: 3828-3832.

Roberts, S., Kingsbury, S. R., Stoeber, K., Knight, G. L., Gallimore, P. H., Williams, G. H. (2008) Identification of an arginine-rich motif in human papillomavirus type 1 E1^{E4} protein necessary for E4 mediated inhibition of cellular DNA synthesis in vitro and in cells. *J. Virol.* 82: 9056-9064.

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Wilson, R., Ryan, G. B., Knight, G. L., Laimins, L. A., Roberts, S. (2007) The full-length E1^{E4} protein of human papillomavirus type 18 modulates differentiation-dependent viral DNA amplification and late gene expression. *Virology* 362: 453-460.

Roberts, S., Calautti, E., Vanderweil, S., Nguyen, H. O., Foley, A., Baden, H. P., Viel, A. (2007) Changes in localization of human discs large (hdlg) during keratinocyte differentiation are associated with expression of alternatively spliced hdg variants. *Exp. Cell Res.* 313: 2521-2530.

Bell, I., Martin, A., Roberts, S. (2007) The E1^{E4} protein of human papillomavirus interacts with the serine-arginine specific protein kinase SRPK1. *J. Virol.* 81: 5437-5448.

