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Dr Jo Parish

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About

Jo qualified with a B.Sc. (Hons) in Biochemistry from the University of Bristol in 1998. She obtained her Ph.D from the University of Bristol in 2002 studying virus-host interactions of human papillomavirus (HPV). She was then appointed as a Research Fellow at the University of Massachusetts Medical School, USA. In 2007, Jo was awarded a Royal Society University Research Fellowship and moved to the University of St Andrews. In 2012, she was appointed Senior Lecturer in Cancer Sciences in the University of Birmingham. Her research is focused on HPV-host interactions and the molecular mechanism of HPV persistence.

Qualifications

- Royal Society University Research Fellow 2007
- Ph.D. University of Bristol 2002
- B.Sc. (Hons) in Biochemistry University of Bristol 1998

Biography

Jo moved to Birmingham in 2012 having studied the molecular biology of human papillomaviruses (HPV) for over 14 years. HPV infections are the cause of benign and malignant lesions of differentiating cutaneous and mucosal epithelium. These infections are often persistent and hard to treat. At present effective non-invasive treatments are unavailable.

During her postdoctoral research at the University of Massachusetts USA, Jo became interested in HPV-host interactions that are required for the maintenance and persistence of HPV genomes in infected cells. She discovered an interaction between the HPV E2 protein and the cellular DNA helicase ChlR1 and went on the show that this interaction is necessary for HPV persistence. The interaction between ChlR1 and E2, and its importance in the persistence of HPV infections remains one of the focuses of Jo's research portfolio. The potential for this important virus-host interaction as a novel therapeutic target is currently being investigated.

Jo is also interested in the cellular pathways important for host cell genome stability that HPVs target to facilitate persistence. Her laboratory continues to study the functional role of ChlR1 in genome integrity and uses a variety of cellular and biochemical techniques to study the function of ChlR1 and key binding partners in the maintenance of genome stability.

Teaching

- **Clinical Science BMedSc - Intercalated Degree** (/undergraduate/courses/med/ClinicalScienceBMedSc-IntercalatedDegree.aspx)

Postgraduate supervision

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

- The study of novel HPV-host interactions important for the control of viral gene expression and completion of the viral life cycle.
- The study of HPV-virus interactions important for the maintenance of persistent HPV infections.

If you are interested in studying any of these subject areas please **contact Jo** (mailto:j.l.parish@bham.ac.uk) , or for any general doctoral research enquiries, please email: **dr@contacts.bham.ac.uk** (mailto:dr@contacts.bham.ac.uk) or call +44 (0)121 414 5005.

For a full list of available Doctoral Research opportunities, please visit our **Doctoral Research Programme** (http://www.bhamtest1.bham.ac.uk/students/courses/postgraduate/research/mdslisting.aspx) listings.

Research

Jo's research group is focused on the cellular pathways involved in maintaining host cell genome stability and long-term human papillomavirus (HPV) persistence.

HPV infection is associated with the development of benign lesions or warts, and several malignant lesions of the epidermal layer including cervical cancer. The majority of sexually active adults will become infected with a genital HPV type at least once in their lives, which can result in persistent, disfiguring and hard to treat genital warts or in the development of carcinoma, particularly in patients that maintain a latent 'high-risk' infection over many years. Although HPV infection causes carcinoma in a relatively small number of infected individuals, 99.7% of cervical carcinomas are associated with HPV infection and each year 240,000 cervical cancer related deaths are reported.

To complete their life cycle, all viruses must synthesise proteins in an ordered and concerted manner, copy their genomes (DNA), and partition genomes to new cells during cell division. This ensures long term or latent infection.

Work in the Parish laboratory is primarily focused on how HPV genomes are passed to daughter cells during mitotic division. To ensure the passage of viral genomes to new cells, papillomaviruses hijack some of the mechanisms that exist in host cells required the maintenance of genome stability. In particular, the HPV E2 protein targets several cellular proteins during DNA replication and mitosis to facilitate attachment of genomes to mitotic chromosomes. Work is on going to characterise these virus-host interactions and the cellular pathways hijacked by HPV and decipher their importance in the HPV life cycle.

Other activities

Memberships:

- British Society for Cell Biology (2009)
- Biochemical Society (2009)
- Society for General Microbiology (2010)

Peer reviewing:

- Reviewer of manuscripts submitted to numerous molecular cell biology and virology journals
- Reviewer for grant funding bodies such as the Medical Research Council, BBSRC, and CR-UK
- Editorial board for the journal 'Viruses' (April 2009)

External examination:

- External Ph.D. examiner in the areas of HPV molecular biology and cell cycle control

Outreach:

- STEM ambassador (**STEMNET (http://www.stemnet.org.uk/)** Science, Technology, Engineering and Mathematics Network)

Publications

Feeney K. M., McFarlane-Majeed L., and Parish J.L. Analyzing sister chromatid cohesion in mammalian cells. In: Noguchi E., and Gadaleta M. ed. *Cell Cycle Control: Mechanisms and Protocols, Second Edition*. Methods in Molecular Biology, Humana Press/Springer. In Press

Prystowsky M. B., Feeney K. M., Kawachi N., Montagne C., Willmott M. K. S., Wasson C. W., Antkowiak M., Loudig O. and Parish J. L. (2013) Inhibition of Plk1 and Cyclin B1 expression results in panobinostat-induced G2 delay and mitotic defects. *Scientific Reports*. 3:2640. DOI: 10.1038/srep02640

Feeney K. M., Saade A., Okrasa K. and Parish J. L., (2011) In vivo analysis of the cell cycle dependent association of the bovine papillomavirus E2 protein and ChlR1. *Virology*. 414:1-9.

Feeney K. M, Wasson C. and Parish J. L., (2010) Cohesin: Regulator of Genome Integrity and Gene Expression. *Biochemical Journal.* 428 (2):147-159.

Jolly C. E., Gray L. J., Parish J. L., Lain S. and Herrington C. S., (2009), Leptomycin B Induces Apoptosis in Cells Containing the Whole HPV 16 Genome. *The International Journal of Oncology.* 35:649-656.

Prystowsky M. B., Adomako A., Smith R. V., Kawachi N., McKimpson W., Atadja P., Chen Q., Schlecht N., Parish J. L., Childs G. and Belbin T., (2009), The histone deacetylase inhibitor LBH589 inhibits expression of mitotic genes causing G2/M arrest and cell death in head and neck squamous cell carcinoma cell lines. *Journal of Pathology*. 218 (4):467-477.

Feeney K.M. and Parish J. L., (2009), Targeting mitotic chromosomes: a conserved mechanism to ensure viral genome persistence. *Proceedings of the Royal Society B: Biological Sciences.* 276 (1662):1535-44.

Parish J. L., Bean A.M., Park R.B., Androphy E.J., (2006), ChlR1 is required for loading papillomavirus E2 onto mitotic chromosomes and viral genome maintenance. *Molecular Cell.* 24(6), 70-76.

Parish J. L., Rosa J., Wang X., Lahti J., Doxsey S. J., Androphy E. J., (2006), The DNA helicase ChlR1 is required for sister chromatid cohesion in mammalian cells. *Journal of Cell Science.* 119, 4857-4865.

Parish J. L., Kowalczyk A., Chen H-T., Roeder G., Sessions R., Buckle M. and Gaston K., (2006), The E2 proteins from high- and low-risk HPV type differ in their ability to bind p53 and induce apoptotic cell death. *Journal of Virology.* 80 (9), 4580-90.

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