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About

Victoria currently is driving new areas of paediatric acute leukaemia research, teamed with Dr Pam Kearns, Director of Birmingham Cancer Research Clinical Trials Unit (CRCTU).

Specifically, she is interested in molecular mechanisms underlying chemo-resistance and relapse in acute myeloid leukaemia and acute lymphoblastic leukaemia, with the goal of informing and testing new targeted therapeutic approaches. She has received funding from Leukaemia Lymphoma Research Fund, Experimental Cancer Medicine Centre and British Society for Haematology for her research.

Her previous work in adult chronic lymphocytic leukaemia led to a national clinical trial for the DNA repair inhibitor, Olaparib, in ATM null CLL which is currently open in Birmingham. She won the Catovsky prize for CLL for this work in 2010.

Qualifications

- PhD Medicine and Dentitry 2002
- BSc (Hons) Genetics First Class 1998

Biography

Victoria qualified from Cardiff University in 1998 with a first class honours BSc degree in Genetics. During her time in Cardiff, she spent a year working at the Regional West Midlands Genetics Laboratory under the care of Val Davison and Simon Larkins learning techniques in chromosome analysis.

She also undertook research with Lesley Jones at the Huntington's Research group at Heath Hospital, Cardiff. In 1998, Victoria joined Tanja Stankovic and Carmel McConville to study, in collaboration with Birmingham Children's hospital, clonal evolution in childhood ALL. She stayed with Tanja Stankovic for several years, working on to drive the development of research into childhood leukaemia.

Teaching

- Medicine - SGT
- BMedSci - Tutorials
- MSc in Clinical Oncology (Animal Models in Translational Research; Cancer Stem Cells)

Summer student (2011) '*AML stem cells*'

BMedSci final year project student (2011) '*Manipulating a cell cycle defect to selectively kill T-ALL cells*'

Postgraduate supervision

- PhD student (2008-2012) *'Investigating pro-survival pathways in apoptosis resistant paediatric B-precursor ALL'*
- *Clinical PhD student (2008-2012) 'Receptor tyrosine kinases in paediatric ALL – novel therapeutic approaches'*
- MRes Toxicology BBRSC-funded (£5K) *in vivo* project student (2011) '*An in vivo murine xenograft model to determine drug sensitivity of subpopulations of ALL cells*'

Research

Currently, Victoria is working closely with Pam Kearns to develop and establish a National Centre for Leukaemia Xenograft Models which is currently funded by the LLR. They wish to expand this existing service to facilitate basic, translational and pre-clinical research both locally and nationally. Currently, a number of exciting collaborations are underway.

Victoria's main research interests focuses on chemo-resistance in AML and ALL in children. In AML, she has identified a population of highly chemo-resistant cells which may contain the leukaemia stem cells responsible for relapse in a subgroup of high risk patients. These are currently being characterised further to identify new potentially effective therapeutic targets. Another major area of interest which has arisen from her earlier research into clonality in ALL is patterns of clonal evolution in different leukaemia cell populations in AML and the association with relapse.

In ALL, the role of microRNAs in poor responses to chemotherapy is a major area of her research and is funded by the LLR. Specific microRNAs may be targeted to improve clinical outcome in some patients. A second, ECMC funded project is addressing the molecular basis for a synergistic effect of inhibition of two specific survival pathways which can chemo-sensitise ALL cells. This could offer an exciting new therapeutic approach for ALL and other tumours.

Previous research included the ability of the DNA repair inhibitor, Olaparib, to target ATM mutant CLL in adults. Victoria won the Catovsky prize for CLL in 2010 for this work. A Birmingham-based national trial is now open (under Tanja Stankovic and Guy Pratt) for 11qdel CLL and Olaparib.

Other activities

Victoria is currently a member of the childhood leukaemia research UK (CLR-UK) group and the I-BFM biology and diagnostics and resistant disease committees.

Through her previous work on the DNA repair inhibitor, Olaparib, in CLL with Professor Stankovic, Victoria won the *Catovsky prize for CLL* in 2010.

Publications

- Britt-Compton, B., Lin, T.,T., Ahmed, G., Weston, V., Jones, R.,E., Fegan, C., Oscier, D.G., Stankovic, T., Pepper, C., Baird, D.M. (2012). **Extreme telomere erosion in ATM-mutated and 11q-deleted CLL patients is independent of disease stage (http://www.ncbi.nlm.nih.gov/pubmed/21986843)**. Leukemia;26(4):826-30.
- Skowronska, A., Austen, B., Powell, J.E., Weston, V., Oscier, D.G., Dyer, M.J.S., Matutes, E., Pratt, G., Fegan, C., Moss, P., Taylor, A.M.R., Stankovic, T. (2012). ATM germline heterozygosity does not play a role in CLL initiation but influences rapid disease progression through loss of the remaining ATM allele. Haematologica;97(1):146-6.
- Weston, V.J., Oldreive, C.E., Skowronska, A., Oscier, D.G., Pratt, G., Dyer, M.J.S., Smith, G., Powell, J.E., Zbigniew, R., Kearns, P., Moss, P.A.H., Taylor, A.M.R., Stankovic, T. (2010).The novel PARP inhibitor, AZD2281 (KU-0059436), selectively targets ATM-deficient cycling B-CLL cells for killing by a mechanism independent of p53-induced apoptosis. Blood;116(22):4578-87.
- Marston, E., Weston, V., Jesson, J., Maina, E., McConville, C., Agathanggelou, A., Skowronska, A., Mapp, K., Sameith, K., Powell, J.E., Lawson, S., Kearns, P., Falciani, F., Taylor, M., Stankovic, T. (2009). Stratification of paediatric ALL by in vitro cellular response to DNA double-strand breaks provides insight into the molecular mechanisms underlying clinical response. Blood; 113(1):117-26.
- Alvi, A.J., Austen, B., Weston, V.J., Fegan, C., MacCallum, D., Gianella-Borradori, A., Lane, D.P., Hubank, M., Powell, J.E., Wei, W., Taylor, A.M., Moss, P.A., Stankovic, T. (2005). A novel CDK inhibitor, CYC202 (R-roscovitine), overcomes the defect in p53-dependent apoptosis in B-CLL by down-regulation of genes involved in transcription regulation and survival. Blood; 105(11): 4484-91.
- Weston, V.J., Austen, B., Wei, W., Marston, E., Alvi, A., Lawson, S., Darbyshire, P.J., Griffiths, M., Hill, F., Mann, J.R., Moss, P.A.H., Taylor, A.M.R., and Stankovic, T. (2004). Apoptotic resistance to ionising radiation in paediatric B-precursor acute lymphoblastic leukaemia frequently involves increased NF-□B survival pathway signalling. Blood; 104(5); 1465-73.

- Marston, E., Weston, V., Jesson, J., Maina, E., McConville, C., Agathanggelou, A., Skowronska, A., Mapp, K., Sameith, K., Powell, J.E., Lawson, S., Kearns, P., Falciani, F., Taylor, M., Stankovic, T. (2009). Stratification of paediatric ALL by in vitro cellular response to DNA double-strand breaks provides insight into the molecular mechanisms underlying clinical response. Blood; 113(1):117-26.
- Alvi, A.J., Austen, B., Weston, V.J., Fegan, C., MacCallum, D., Gianella-Borradori, A., Lane, D.P., Hubank, M., Powell, J.E., Wei, W., Taylor, A.M., Moss, P.A., Stankovic, T. (2005). A novel CDK inhibitor, CYC202 (R-roscovitine), overcomes the defect in p53-dependent apoptosis in B-CLL by down-regulation of genes involved in transcription regulation and survival. Blood; 105(11): 4484-91.

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