Biomedical Sciences Institutes
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p53 Laboratory Seminar Announcement

- All Are Welcome -

Speaker: **Professor Ron Hay**
Professor of Molecular Biology
College of Life Sciences, University of Dundee

Title : ***"Therapeutic benefits of dialogue between SUMO and ubiquitin"***

Date : **14 November 2011 (Monday)**

Time : **11.00am – 12.00pm**

Venue : **Aspiration Theatrette, Matrix Level 2M**

Host : **Professor Sir David Lane**
(Tel: 64070553, e-mail: dplane@p53lab.a-star.edu.sg)

Abstract:

Protein modification with ubiquitin and the ubiquitin-like protein SUMO is carried out by enzymatic pathways that, while mechanistically similar, are accomplished by unique enzymes. The physiological consequences of ubiquitin and SUMO modification are mediated by effector proteins that contain sequence motifs or small domains that specifically recognise either ubiquitin or SUMO. It was believed that these modifications resulted in quite different biological consequences but recent work is showing that there is considerable cross-talk between the ubiquitin and SUMO systems driven by SUMO targeted ubiquitin E3 ligases. In vertebrates the RING domain containing, ubiquitin E3 ligase Rnf4, targets poly-SUMO conjugated proteins for ubiquitin mediated degradation. Multiple SUMO Interaction Motifs in Rnf4 allow it to selectively recognise and ubiquitylate poly-SUMO chains. In Acute Promyelocytic Leukaemia, Promyelocytic Leukaemia (PML) protein is fused to the Retinoic Acid Receptor and the condition can be ameliorated by arsenic treatment. Arsenic induces SUMO modification and proteasomal degradation of PML. In the absence of Rnf4, arsenic cannot induce degradation of PML and SUMO modified PML accumulates in the nucleus. Recent work indicates that RNF4 plays a critical role in the DNA-damage response and repair pathways. I will discuss the likely substrates of RNF4, how it is recruited to sites of DNA damage and it's mechanism of action as a ubiquitin E3 ligase.

About the Speaker:

Ron Hay obtained a B.Sc. in Biochemistry at Heriot-Watt University, Edinburgh in 1975. He obtained his PhD in 1979 from the Medical Research Council Virology Unit in Glasgow and was a Damon Runyon-Walter Winchell Cancer Fund postdoctoral fellowship in the laboratory of Dr Mel DePamphilis at Harvard Medical School, Boston. Returning to the MRC Virology Unit in 1982, he established an independent laboratory working on the initiation of adenovirus DNA replication. In 1985 he moved to the University of St. Andrews where he held Lecturer and Reader positions before taking up the Chair in Molecular Biology and becoming Deputy Director of the new Centre for Biomolecular Sciences. In 2005 he joined the College of Life Sciences at the University of Dundee where he is Professor of Molecular Biology in the Wellcome Trust Centre for Gene Regulation and Expression and is an Honorary member of the Scottish Institute for Cell Signaling (SCILLS). Ron's research has established conjugation with the Small Ubiquitin-like Modifier (SUMO) as an important regulatory mechanism in eukaryotes. He recently uncovered a key role for SUMO and ubiquitin in mediating the therapeutic effects of arsenic for the treatment of Acute Promyelocytic Leukaemia. Ron has been elected as a Fellow of the Royal Society, a Fellow of the Royal Society of Edinburgh, a Fellow of the Academy of Medical Sciences and as a Member of the European Molecular Biology Organisation.

Lab website: http://groups.www.lifesci.dundee.ac.uk/ron_hay/