Agency for Science, Technology and Research
SINGAPORE

p53 Laboratory Seminar Announcement

- All Are Welcome -



Speaker: **Dr Christopher Kemp**
Full Member, Division of Human Biology,
Fred Hutchinson Cancer Research Center

Title: ***"Personalized cancer models for target development and precision oncology"***

Date: **7 May 2018 (Monday)**

Time: **11.00am – 12.00pm**

Venue: **Breakthrough Theatrette, Matrix Level 4, Biopolis**

Host: **Prof Sir David Lane**
*Chief Scientist, A*STAR & Director, p53Lab*

Abstract:
A major goal of precision oncology is to utilized genomic information to inform patient care. While there are spectacular examples of success, for the great majority of cancer patients, genomic information is insufficient to guide patient care or select effective therapeutic options. We have developed an approach that employs high-throughput functional testing with both siRNA and drugs using patient tumor cell cultures. This functional data combined with genomic analysis is used to distinguish driver from passenger mutations and identify novel drug targets and potentially effective drugs specific to a given patient. This approach led to identification of WEE-1 as a targetable vulnerability of p53 mutant cancers. Results from a clinical trial of the WEE1 inhibitor AZD1775 in p53 mutant head and neck squamous cell carcinoma will be presented.

About the Speaker:
Dr. Kemp received a BA in Biology from Case Western Reserve University, an MS in Fisheries Toxicology from Oregon State University and PhD in Experimental Oncology from the University of Wisconsin, Madison. His PhD thesis focused on the genetic and hormonal basis of hepatocellular carcinoma using mouse models. He did postdoctoral research with Allan Balmain at the Beatson Institute for Cancer Research in Glasgow Scotland studying the role of Hras and p53 in multistage cancer using mouse models. He has been at the Fred Hutchinson Cancer Research Center for over 23 years where he studied the role of tumor suppressor genes including p19/Arf, Atm, Cdkn1b/p27 and Ctcf in cancer progression. He is currently using functional genomic approaches to identify new cancer drug targets using a range of patient derived tumor models.
Highlights of Dr. Kemp's 30 year research career in studying the environmental and genetic basis of cancer using mouse models include the following. 1.Discovery of a causal role for p53 in tumor progression and metastasis (Kemp et al., 1993). 2) Discovery that p53 is a major barrier to ionizing radiation induced carcinogenesis (Kemp et al., 1994). 3) Discovery of synthetic lethality between DNA repair genes DNA-PK and Atm, the first such example in mammals and predating the current interest in synthetic lethality in cancer research by many years (Gurley and Kemp, 2001). 4) Discovery that p27 is a tumor suppressor gene (Fero et al., 1998). 5) Discovery of the first example of a haploinsufficient tumor suppressor, thus modifying one of the central dogmas in cancer research (e.g. Knudson's two hit hypothesis). The concept of tumor suppressor haploinsufficiency is now firmly integrated in the field of cancer genetics (Payne and Kemp, 2005). 6) Discovery of a novel p53 independent apoptotic pathway that is regulated by DNA-PK (Gurley et al., 2009). 7) Discovery that the DNA binding protein CTCF is a haploinsufficient tumor suppressor gene (Kemp et al., 2014). 8) Pioneering the application of proteomics to mouse models of cancer for early detection research (Pitteri et al., 2011; Whiteaker et al., 2011; Taguchi et al., 2011). 9) Development and application of a functional genetics platform to both human and mouse derived tumor cells to identify new cancer drug targets (Moser et al., 2014, Xu et al, 2018).