



***Department of Biomedical Sciences***

***Departmental Seminar***

**“Epigenetic Mechanisms underlying Male  
Predominance of Liver Cancer”**

By

**Prof Alfred Sze Lok Cheng**

***School of Biomedical Sciences***

***Faculty of Medicine***

***The Chinese University of Hong Kong***

**Date: 17 June 2015 (Wednesday)**

**Time: 11:00 am – 12:30pm**

**Venue: Room P4701 (*near Lift 1*),  
Level 4, Purple Zone,  
Academic 1,  
City University of Hong Kong,  
Tat Chee Avenue, Kowloon Tong**

***For abstract, please refer to the attachment.***

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***~ All are Welcome ~***

# “Epigenetic Mechanisms underlying Male Predominance of Liver Cancer”

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## **About the speaker**

Alfred Cheng is an Associate Professor in the School of Biomedical Sciences at the Chinese University of Hong Kong (CUHK). He completed his PhD under the supervision of Prof. Joseph Sung in the Department of Medicine and Therapeutics at CUHK in 2002 and went on postdoctoral training characterizing the roles of cyclooxygenase-2 in hepatitis B-induced hepatocarcinogenesis. From 2004-2007, he was trained as a postdoctoral researcher in Prof. Tim Huang's lab in the Ohio State University, USA, where he developed an integrated genome-wide and bioinformatics approach to interrogate gene regulatory network in cancer. He has received several scientific awards including three American Association of Cancer Research (AACR) Scholar-in-Training Awards (2004-2006) and Travel Grants/Oral Free Paper Prize from the United European Gastroenterology for three consecutive years (2011-2013). He was also a recipient of the Most Promising Young Investigator Award by the Food and Health Bureau, The Government of Hong Kong SAR at 2014. Dr. Alfred Cheng published in international journals including *Molecular Cell*, *Nature Genetics*, *Journal of Clinical Investigation*, *Cancer Cell*, *Cancer Research*, *Gastroenterology*, *Gut* and *Journal of Hepatology*. His current research focuses on the transcriptional and epigenetic mechanisms of gastrointestinal carcinogenesis and is supported by funding from RGC-TBRS, CRF, GRF, HMRF and NSFC.

## **Abstract**

Hepatocellular carcinoma (HCC) is one of the commonest and deadliest cancers worldwide, with a striking epidemiological characteristic of male predominance. Androgen receptor (AR) is a ligand-activated nuclear receptor that regulates the development and maintenance of male sexual phenotype. Aberrant AR signaling, however, has detrimental consequences in the development of cancers. The Polycomb protein Enhancer of zeste homolog 2 (EZH2) plays a key role in oncogenesis by epigenetically silencing tumor-suppressor genes. Both AR and EZH2 contribute to HCC, yet whether the two oncogenic factors have functional crosstalk has been elusive. Our previous genome-wide location analysis has identified cell cycle-related kinase (CCRK) as an AR direct target in HCC. More recently, we found that AR/CCRK signaling up-regulates EZH2 expression and phosphorylation, which in turn remodel the chromatin environment for gene silencing and activation, respectively. These findings not only advance our fundamental understanding of the gender disparity in HCC, but also provide a new paradigm for nuclear receptor regulation of cancer epigenome.