Department of Biomedical Sciences & School of Veterinary Medicine present Seminar

"Roles for the 9-1-1 DNA damage response complex during mammalian development and tumorigenesis"

By

Prof. Robert S. Weiss

Professor
Department of Biomedical Sciences
Cornell University

Date: 14 April 2015 (Tuesday)

Time: 11:00 am – 12:30 pm

Venue: CSE Conference Room
Room B6605 *(near Lift 3)*
Level 6, Blue 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 ~

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About the speaker

Robert Weiss is an Associate Professor of Molecular Genetics in the Department of Biomedical Sciences, within the College of Veterinary Medicine at Cornell University. He received an A.B. in Biology *summa cum laude* from Wabash College in 1992 and a Ph.D. in Molecular Virology from Baylor College of Medicine in 1997. He then trained as a postdoctoral fellow in the Department of Genetics at Harvard Medical School with support from an American Cancer Society postdoctoral fellowship. In 2002, Dr. Weiss joined the faculty of Cornell University. He lectures on Cell Biology and Genetics in the pre-clinical veterinary curriculum and additionally teaches a graduate course on genome maintenance mechanisms. His National Institutes of Health-funded research group studies how mammalian cells maintain chromosomal integrity and investigates roles for genomic instability in cancer and other diseases, using mouse models and an array of genetic, molecular, cell biological, and genomic technologies. Dr. Weiss is Director of the Comparative Cancer Biology Program at the College of Veterinary Medicine.

Abstract

Eukaryotic cells continuously experience DNA damage that originates from both intrinsic and extrinsic sources. If left uncorrected, the resulting genomic lesions can result in developmental defects, premature aging, and increased cancer risk. Cells protect against these undesirable outcomes using a variety of mechanisms, including DNA damage checkpoint pathways that promote DNA repair and coordinate repair processes with cell cycle progression. The Weiss laboratory uses mouse models to investigate mammalian genome maintenance mechanisms and the physiological consequences of genomic instability. A primary research objective is to understand the functions of the essential checkpoint protein Hus1, a component of the heterotrimeric 9-1-1 complex that promotes DNA damage-induced checkpoint signal transduction and also directly participates in DNA repair by recruiting repair proteins to DNA lesions. The presentation will describe new findings related to the molecular mechanisms of the 9-1-1 complex as well as the use of mouse models to investigate the impact of checkpoint dysfunction on organismal development, tumorigenesis, and DNA damage responses in adult mice.