Department of Biological Sciences

Ph.D. Open Seminar

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Date & Time: **Friday, August 11, 2017 at 04:00 PM** Venue: **L8, LHC**

Essential Role of HCMV in Oncogenesis through evasion of Host Innate-Immunity

Cancer is a multifactorial disease and virus–mediated carcinogenesis is one of the crucial factors, which is poorly understood. The DNA and RNA viruses are known to cause cancer. The DNA oncogenic viruses such as Human-herpesvirus, Papilloma-virus, and Hepatitis-B virus are extensively studied, however, role of Human cytomegalovirus (HCMV) in cancer remains unknown, although HCMV is a human-herpesvirus and its structural components have been evidenced to be associated with cancer of different tissue origin. Here, herpesviral tegument protein known as pUL48 of HCMV, encoding deubiquitinase enzyme has been identified playing a key role in carcinogenesis [1]. Using deubiquitinase sufficient- and deficient-HCMV, it has been shown that HCMV deubiquitinase is a key in inducing enhanced cellular metabolic activity through upregulation of several anti-apoptotic genes and downregulation of several pro-apoptotic genes. Further investigation reveal that HCMV-deubiquitinase acquires pro-tumor functions through inhibiting PRR-mediated type I interferon synthesis via deubiquitination of key signaling molecules of innate-immune pathway. This new role of HCMV deubiquitinase in inhibition of innate-immunity is found in accordance with deubiquitinases of many viruses [2] and other HCMV proteins [3]. Taken together, the results suggest that HCMV infection may promote oncogenesis by inhibiting innate-immunity and the inhibitor for the viral deubiquitinases may help in combating virus-mediated oncogenesis.

References:

1. P Kumari et al. (**2017**), Cell Death & Disease, In Press.

2. P Kumari et al. (**2017**), Critical Reviews in Microbiology, Under Review.

3. P Kumari et al. (**2015**), Reviews in Medical Virology, **25**(3), p. 187-201.