BIOENGINEERING
Spring 2021 Seminar

Date: **Thursday, April 29, 2021**
Time: **12:00 pm - 1:00pm**
Location: **Virtual**
Join Zoom Meeting
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Meeting ID: 988 0549 4005 Passcode: 454698

Fatah Kashanchi, Ph.D.

Biography: Dr. Kashanchi received his Ph.D. in 1990 under the supervision of Dr. Charles Wood who also worked with the Nobel Laurite, Dr. Susumu Tonegawa at MIT. He then moved to National Cancer Institute at NIH's intramural program and continued his work on RNA viral infections. He is currently a Tenured Faculty in the department of Systems Biology at the Prince William Campus of George Mason University. He has obtained independent funding of more than $17.3 M in funding (NIH, DOD, DOE, and Keck) since his departure from NIH in 2000. He has published 236 peer-reviewed manuscripts (h index = 62), and served as an editorial board and reviewer for number of journal including Cell, Molecular Cell, Nature, Nature Medicine, Science Translational Medicine, Retrovirology, JBC, J. Virol, Virology, NAR, and 4 PLoS journals. He is a regular NIH study section member and has served on 161 panels and chaired 17 since 2000.

Title: ***Exosomes and Viruses: A Tale of Two Overlapping Worlds***

Abstract: Extracellular vesicles (EVs) play a significant role in intercellular communication by serving as a carrier for the transfer of membrane and cytosolic proteins, lipids, and RNA between cells. In recent years, using state of art technologies such as RNA seq, RPMA, and single cell omics, we have found that virally infected cells including HIV-1, HTLV-1, Rift Valley Fever, Zika, Ebola, and Coronavirus infected cells secret exosomes that contain biomarker of these infections in urine, saliva, CSF, and blood. We have been able to separate and characterize EVs from several different viruses including HIV-1. These EVs are not infectious and have a different density than infectious virions using gradients. They contain various viral RNAs including TAR (non-coding RNA), Nef, gp120/160 and Tat. The origin of these EVs are infected cells, especially when treated with cART or Interferons. They are present in patient samples tested (plasma and CSF, 33%-95%) to date (4 cohorts of 5-20 patients each). The EVs contribute to pro-inflammatory signals in the naïve recipient cells using TLR3 signaling. Recently, we have asked about the timing difference between EV and virus release from infected cells using serum starvation experiments from cells followed by release. Results from supernatants of uninfected cells showed a peak of tetraspanin proteins (CD63, CD81, and CD9) at 6 hours and a gradual decrease of all EV associated proteins by 24 hours. However, the EV from HIV-1 infected cells showed all three tetraspanins present at 3 hours and expression gradually increased up to 24 hours. HIV-1 viral proteins (p24, gp120, Nef) expression was present at 6 hours and continued to increase and peaked at 24 hours. HIV-1 supernatant 6- hour sample was found not to be infectious. However, infectious HIV-1 was successfully rescued from 24-hour sample. We will discuss the implications of these findings in infection spread and finally how to repair damaged cells post infection using stem cell exosomes.