TWINCORE

TWINCORE – Seminar

Monday September 10th, 2018, 5 p.m.
TWINCORE Lecture Hall 0.020

Innate Immune Activation in HIV Infection

Hisashi Akiyama, Ph.D.

Systemic chronic immune activation and T cell dysfunction are hallmarks of HIV-1 infection. Despite long-term viral suppression by combination antiretroviral therapy (cART), immune activation and inflammation persist in the majority of HIV-infected individuals on cART, and is associated with excess risk of mortality and morbidity. Low-levels of type I interferon (IFN-I) are thought to be a driving force for immune activation and T cell exhaustion in HIV-1 infected individuals on cART. Since tissue-resident myeloid cells can remain persistently infected with HIV-1 even in individuals on cART, we hypothesized that persistent infection of myeloid cells may contribute to immune activation and T cell dysfunction.

Here we demonstrate that expression and Rev–CRM1-dependent nuclear export of intron-containing HIV-1 RNA activates host sensing mechanisms and IFN-I-dependent pro-inflammatory responses via MAVS in productively infected macrophages. Furthermore, HIV-1 infection-induced activation of macrophages, in turn, leads to exhaustion of co-cultured autologous T cells. This study suggests that use of HIV RNA expression inhibitors as adjunct therapy might abrogate aberrant inflammation and restore immune function in HIV-infected individuals on cART.

Who is Hisashi Akiyama?

- Research Assistant Professor, Department of Microbiology, Boston University School of Medicine

Before:

- 1/2014-1/2017: Senior Research Scien., Boston University School of Medicine, Boston, MA
- 1/2009-1/2014: Research Associate, Boston University School of Medicine, Boston, MA
- 5/2006-12/2008: Postdoctoral Fellow, University of Heidelberg, Germany
- 4/2004-3/2006: Postdoctoral Research Fellowship, Japanese Foundation for AIDS Prevention

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