Subject: Dissertation Defense - Amrita Haikerwal, PhD in Biosciences
Date: Thursday, April 16, 2026 at 9:45:13 AM Eastern Daylight Time
From: SSB Faculty List on behalf of Diane St. Germain
To: SSB-FACULTY-LIST-L@LISTSERV.GMU.EDU

Dissertation Defense Announcement
To: The George Mason University Community

Candidate: Amrita Haikerwal

Program: PhD in Biosciences

Date: April 27, 2026

Time: 10:00 A.M Eastern Time (US and Canada)

Location: In Person - Life Science Engineering Bldg, Science & Tech Campus, Manassas VA

and via Zoom

Join Zoom meeting

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Committee Chair: Dr. Yuntao Wu

Committee members: Dr. Aarthi Narayanan, Dr. Ramin Hakami, Dr. Mikell Paige

Title: CD34 Restricts HIV-1 Replication in Hematopoietic Stem and Progenitor Cells

Abstract:

Hematopoietic stem cells (HSCs) are multipotent, have self-renewal capability and can differentiate and produce all mature blood cells. Hematopoietic stem cells are generally resistant to viral infections as a protective mechanism to ensure their survival and function. These stem cells use restriction factors, RNA interference pathways and constitutively express interferon stimulated genes (ISGs) to protect themselves from viral infections. The relationship between HIV infection and hematopoietic stem and progenitor cells is a complex interplay of resistance, and low-level infections. However, integrated HIV proviral DNA copies are found within highly purified populations of human CD34+ hematopoietic stem and progenitor cells from HIV-infected patients. This suggests that HIV infection in CD34+ hematopoietic stem and progenitor cells is possible, but some antiviral mechanism prevents productive HIV replication in these cells. In this study, we identified that CD34, a marker of hematopoietic stem and progenitor cells, has antiviral activity against HIV-1. Mechanistically, the major phenotype is that in CD34 expressing HIV-1 producer cells, CD34 gets incorporated in the budding HIV-1 virion and prevents viral attachment to target cells; and CD34 disrupts viral protease processing and impairs virion maturation. CD34 does not block viral entry, integration, viral gene expression or viral release during primary infection. Collectively, our findings demonstrate CD34 as an antiviral host intrinsic protein which prevents spread of HIV-1 infection in hematopoietic stem and progenitor cells. Notably, our study also explains that HIV-1 infection in CD34+ hematopoietic stem and progenitor cells is limited due to the presence of CD34 on the cell surface, however, upon cellular differentiation and the concomitant loss of surface CD34, this restriction would be relieved, enabling more efficient production of infectious viruses.

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