1155-92-420 **Maria Emelianenko***, memelian@gmu.edu, and **Dan Anderson**, **Fatah Kashanchi**, **Catherine DeMarino**, **Tin Phan** and **Yang Kuang**. *Three-state mathematical HIV model reveals differences in transcriptional dynamics between T-cells and macrophages.*

HIV-1 viral transcription persists in patients despite antiretroviral treatment, potentially due to intermittent HIV-1 LTR activation. While several mathematical models have been explored in the context of LTR-protein interactions, in this work for the first time HIV-1 LTR model featuring repressed, intermediate, and activated LTR states is integrated with generation of long (env) and short (TAR) RNAs and proteins (Tat, Pr55, and p24) in T-cells and macrophages using both cell lines and primary cell infection. This type of extended modeling framework allows us to compare and contrast behavior of these two cell types. We demonstrate that they exhibit unique LTR dynamics, which ultimately results in differences in the magnitude of viral products generated. Incorporation of a transcription inhibitor (F07#13) into the governing equations demonstrates how the model can be used to assess drug efficacy. Theoretical results concerning model stability and numerical sensitivity analysis on the model parameters are presented. (Received January 20, 2020)