1116-92-1264 **Jessica M Conway***, jmconway@psu.edu, and **Alan S Perelson**. *Residual viremia in treated HIV+ patients: simple model insights.*

Antiretroviral therapy (ART) effectively controls HIV infection, suppressing HIV viral loads. However, in HIV-infected patients on ART, some residual virus remains, below the level of detection. The source of this viremia is an area of debate: does it derive from ongoing rounds of viral replication, activation of infected cells in the latent reservoir, or some combination of the two? Observations support both sides. For example, emergent drug resistance, which results from mutation during viral replication, is rare, implying that viremia derives HIV archived in the latent reservoir, but evidence of short-term evolution remains, implying ongoing viral replication. We will discuss a simple deterministic model with its stochastic, branching process, analogue to gain insight into residual viremia dynamics in HIV-infected patients. We show that the contribution of viral replication to residual viremia may be non-negligible, permitting some short-term viral evolution. But even if that contribution is significant, long-term evolution can still be limited: results suggest de novo emergence of drug resistance is rare. Thus our simple modeling suggests reconciles the seemingly contradictory observations on residual viremia. (Received September 18, 2015)