# Department of Biological Sciences

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***Venue: L3, LHC***

## SERINC: A novel family of antiretroviral genes

As a result of its coevolution with viral parasites, the host has developed sophisticated mechanisms that interfere with virus replication. Viruses have, on the other hand, evolved countermeasures to elude such antiviral barriers in order to adapt to its natural host environment. Primate lentiviruses encode Nef, a multifunctional regulatory protein which plays a crucial role in virus replication and the development of AIDS. Nef is required for the biogenesis of fully infectious retrovirus particles, an activity which remained so far unexplained. We have recently identified novel cellular inhibitors of HIV-1, SERINC5 and SERINC3, which are highly expressed in blood-derived cells and are counteracted by Nef. SERINC5 is efficiently incorporated into retrovirus particles and acts by undermining an early stage of the infection process of target cells, preceding reverse transcription. When retrovirus producer cells express Nef, SERINC5, otherwise predominantly localized at the cell surface, is targeted to endosomal compartments and fails to incorporate into virions. We have demonstrated that not only HIV, but also MLV and EIAV have developed factors capable of antagonizing SERINC5. Interestingly, all three retroviruses establish life-long, slow-progressing persistent infection in their host by infecting blood cells which highly express SERINC5. The necessity for SERINC5 counteraction by HIV-1 and other related retroviruses therefore advocates its fundamental role in pathogenesis. Importantly, ectopically overexpressed SERINC5 can abolish the infectivity of progeny HIV-1, indicating that this is a powerful inhibitor of retrovirus infectivity and a potential anti-HIV therapeutic gene.

[*Chande A et al, PNAS, in press; Rosa A*, Chande A*, Ziglio S* et al, Nature 2015; 526:212-7)*