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Regulation of Cellular and Virion APOBEC3G (A3G) Complexes

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A3G is detectable in both high molecular mass (HMM) and low molecular mass (LMM) complexes in different cells. Enzymatically active LMM A3G complexes are present in resting CD4 T-cells and blood derived monocytes. These cells are not permissive for HIV infection because LMM A3G functions as a potent post-entry restriction factor for HIV and possibly other retroviruses (Chiu et al. Nature 435:108–114, 2005). The antiviral activity of LMM A3G is exerted at the level of reverse transcription but does not appear to involve extensive cytidine deamination of nacent minus strand HIV DNA. When T-cells are activated by mitogens or naïve T cells enter lymphatic tissues where IL-2 and IL-15 are produced, LMM A3G is recruited into an enzymatically inactive HMM ribonucleoprotein complex. This change in A3G complex size is associated with the acquisition of permissiveness to HIV infection. Interestingly, HIV DVif virions incorporate the HMM form of A3G assembled with HIV genomic RNA. Accordingly, a mechanism for activation of this latent A3G complex must come into play. Recently, we have assembled preliminary evidence supporting a key role for Rnase H in the activation of the latent HMM A3G complex. Thus, Rnase H not only prepares the substrate for mutagenesis, but also activates the enzyme.