Cologne Evolution Colloquium

SFB 680 Molecular Basis of Evolutionary Innovations

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HIV - Tropism switch is one of the results of the evolutionary Dynamics of this chronically persisting RNA virus in Humans

HIV is an RNA virus with a high mutation rate. This results in a high genetic divergence so that a patient harbors a swarm of different variants in his body, called quasispecies. These variants have different properties: Under antiretroviral drug pressure HIV can develop resistant strains to each single drug. In addition to this well known event in HIV, the HIV tropism came into clinical focus after the discovery of drugs that can block the CCR5 receptor as one of the two possible coreceptors of HIV. The coreceptor use also termed tropism is determined through the virus. If the HIV variants in the host use the CCR5-receptor, the blocking of CCR5 prevents viral replication. It is widely described that HIV can use either CCR5 or CXCR4 but also that they can switch tropism during the course of disease. Shortly after infection, almost exclusively only CCR5-tropic strains (R5-viruses) can be detected, while 50% of the untreated HIV patients die with a CXCR4-tropic strain (X4-virus).

We developed a cell culture system in which we could selectively modify the two counter actors driving the viral evolution, namely APOBEC (host) and Vif (HIV). We could find that APOBEC enhances the genetic diversity and appears to be one of the possible driving forces of tropism switch/viral evolution. In our future work we would like to verify our so far generated hypothesis in further cell culture system with APOBEC activity on HIV-2 Vif.

Wednesday, December 9, 2015, 17:00
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Seminar Room 0.46

Hosted by Michael Lässig