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Toward a Better Understanding of…

Interaction of Two HIV Protein Paralogues with DCAF1 and Cyclin L2

Austin Niu

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This research focuses on HIV and associated retroviruses. HIV-2 causes a very mild disease in humans while HIV-1 is responsible for the current pandemic. Both viruses have a paralogue of Vpx called Vpr which is believed to be important in HIV pathogenicity. At the cellular level, Vpr and Vpx interact with a DCAF1 (DDB1-CUL4 ASSOCIATED FACTOR 1) which is believed to be essential for their functions. Little is known about these two proteins; hence a screen was performed to look for DCAF1 interacting proteins. The result showed a protein called Cyclin L2 as a novel DCAF1 interacting protein. In response to the screen, a hypothesis was generated that Cyclin L2 would interact with Vpx in the cell. This was tested by creating a cellular environment that would include both Cyclin L2 and Vpx (not the full virus). Cyclin L2 (in plasmid form) was co-transfected with Vpx (also in plasmid form) in increasing dosages into 293T cells which were recovered after 2 days. The Western blot showed that Vpx is degraded by Cyclin L2 in a dose dependent manner. Since DCAF1 is associated with proteasomal degradation of other proteins, we hypothesized that Vpx may be degraded by the proteasome. To test this idea, we did another co-transfection with the addition of the proteasome inhibitor, MG132. The Western blot revealed that MG132 did indeed rescue the degradation of Vpx by Cyclin L2 and showed that the mechanism used by Cyclin L2 to degrade Vpx is the proteosome pathway. Following this, this test will be applied to Vpr during the fall semester to test if Vpr participates in a similar biochemical pathway. Knowledge of the difference between the features of these two proteins is vital to understanding the extreme pathological differences between two nearly identical retroviruses, HIV-1 and HIV-2.