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Major breakthrough in understanding how HIV interferes with infected cell division

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Dr. Éric A. Cohen, a researcher at the IRCM (Institut de recherches cliniques de Montréal), and his team will publish on Friday, July 13, in *PLoS Pathogens* a discovery that could lead to the development of a new class of drugs to combat HIV.

Human immunodeficiency virus type 1 (HIV-1) causes AIDS by depleting essential immune cells called CD4+T lymphocytes in infected individuals, resulting in a compromised immune system. At the center of this process is the HIV protein, viral protein R (Vpr), which stops infected CD4+T cells from dividing and as a consequence compromises their immune function. In addition, by arresting cell division, Vpr helps HIV to harness the infected cell's resources to enhance viral replication. The way Vpr exerts this effect is by interacting with cellular proteins that control cell division.

Dr. Cohen and his team have identified a novel cellular protein complex targeted by HIV-1 Vpr to stop infected cell division. This protein complex, designated DDB1-CUL4-VprBP, is involved in a process called ubiquitination. Ubiquitination is a mechanism by which a small protein called ubiquitin is conjugated to cellular proteins in order to modulate their biological activity or induce their degradation.

The researchers demonstrated that association of Vpr with this ubiquitinating complex, also called an E3 ubiquitin ligase complex, is essential for the defect in cell division induced by Vpr. Further characterization of this protein complex as well as the elucidation of the

mechanism by which it affects cell division may open new avenues for therapeutic intervention against HIV.

Source: Institut de recherches cliniques de Montreal

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