Shutting Down an HIV Protein May Lead to Brain-Protecting Drugs

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Researchers have discovered a new way that an HIV protein called Tat harms the brain, according to a study published in the online journal PLoS One and reported by *Science Daily*. The authors were able to block this damage in the laboratory, potentially opening up new avenues for developing the first drugs that could directly protect—and possibly heal—the brain from damage.

Scientists have known for some time that HIV's Tat protein plays a role in damaging the brain, in addition to helping HIV infect CD4 cells. Tat causes the brain's immune cells to produce chemicals that cause other cells to gather and clump together. Tat also harms mitochondria, the energy producing part of cells that allows them to function. Such damage in the brain can cause problems in movement, concentration and memory in people with HIV.

Now, Harris Gelber, MD, PHD, of the University of Rochester Medical Center in New York, and his colleagues have discovered that Tat inhibits a brain cell receptor called the ryanodine receptor. This receptor folds proteins that help transport chemical signals in the brain. Protein folding consumes a lot of cellular energy. Because Tat also damages the energy-producing mitrochondria, it delivers a one-two punch to brain cells when it inhabits the ryanodine receptor.

Perhaps most exciting, Dr. Gelber's team was able to use the drug dantrolene, which binds to the ryanodine receptor, to block Tat's damage to brain cells. While dantrolene is too toxic to use outside laboratory studies, Gelber's discovery does open new avenues for research into drugs that could not only protect, but also possibly heal HIV-related brain damage. The authors write that their research also has implications for Alzheimer's and Parkinson's diseases.

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