Cracking the Code of an Enzyme That Ultimately Helps HIV

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Researchers at Johns Hopkins University have discovered how to inhibit a key enzyme that plays an important yet complex role in HIV's life cycle. The investigators recently published a summary of their research in Proceedings of the National Academy of Sciences.

Known as SAMHD1, the enzyme in question depletes the genetic building blocks that HIV uses to convert its RNA into DNA and ultimately produce new copies of itself. When levels of SAMHD1 are low, HIV is more likely to thrive in immune cells. However, inhibiting the enzyme is a double-edged sword, because when HIV is thwarted by a lack of proper genetic building blocks, it winds up leaving small pieces of itself in CD4 cells. The consequence of this is an inflammatory response that causes neighboring CD4s to commit suicide. As a result, scientists have concluded that people with HIV may be better off without the enzyme, even though it does help fight other viruses.

After researching how SAMHD1 is turned on and kept functioning, the Johns Hopkins researchers have developed a small molecule that inhibits the enzyme.

"This is a nice starting place for further inhibitor design," James Stivers, PhD, a professor of pharmacology and molecular sciences at the Johns Hopkins University School of Medicine, said in a release.

Because SAMHD1 is rarely mutated, the researchers hope it will prove a better target for antiretroviral therapies.

To read the release, click here.

To read the study abstract, click here.