On the Cure Front, More Drugs Appear to Spur Latent HIV

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Researchers have identified an existing class of investigational cancer drugs that may help spur back into action immune cells latently infected with HIV. Publishing their findings in Cell Host & Microbe, researchers studied the ability of a drug class called Smac mimetics to reverse HIV latency in cells.

Standard antiretroviral treatment cannot attack HIV that effectively hides within latent cells, because ARVs only work when a cell is replicating. This essentially makes HIV inside a latent cell invisible to standard treatment for the virus. (Latently infected cells are a key part of what is known as the viral reservoir.) Consequently, ARVs on their own are unlikely to ever cure HIV. The apparent exception is a dozen or so documented cases of people treated very soon after infection who later went into what French scientists studying them call “viral remission”: Even after going off ARVs for an extended, years-long period, their virus is still under control.

The agents considered in this new study may one day become a part of a cure strategy known as shock and kill, or kick and kill. This method is still in the early phase of the investigational process. One drug, or combination of drugs, causes the latently infected cells to replicate. Another process is also required to prompt an adequate, safe immune response to kill off the HIV. Thus far, researchers have made more progress on the shock part of the approach than the kill part.

Other cancer drugs, known as HDAC inhibitors, are also under investigation by many researchers as latency-reversal agents. So far the results of studies of these drugs’ effect on the HIV reservoir have not been stellar.

In their recent research on Smac mimetics, scientists found promising results by combining a drug called SBI-0637142 with the HDAC inhibitor panobinostat. The combination showed signs of awakening latent HIV without prompting an immune response (which can actually be harmful). There were similarly positive results from combining the Smac mimetic LCL161-a with panobinostat.

The researchers hope to move these experiments into human testing.

To read a press release about the study, click here.

To read the study abstract, click here.
