Researchers find how natural killer cells regulate protective HIV antibodies

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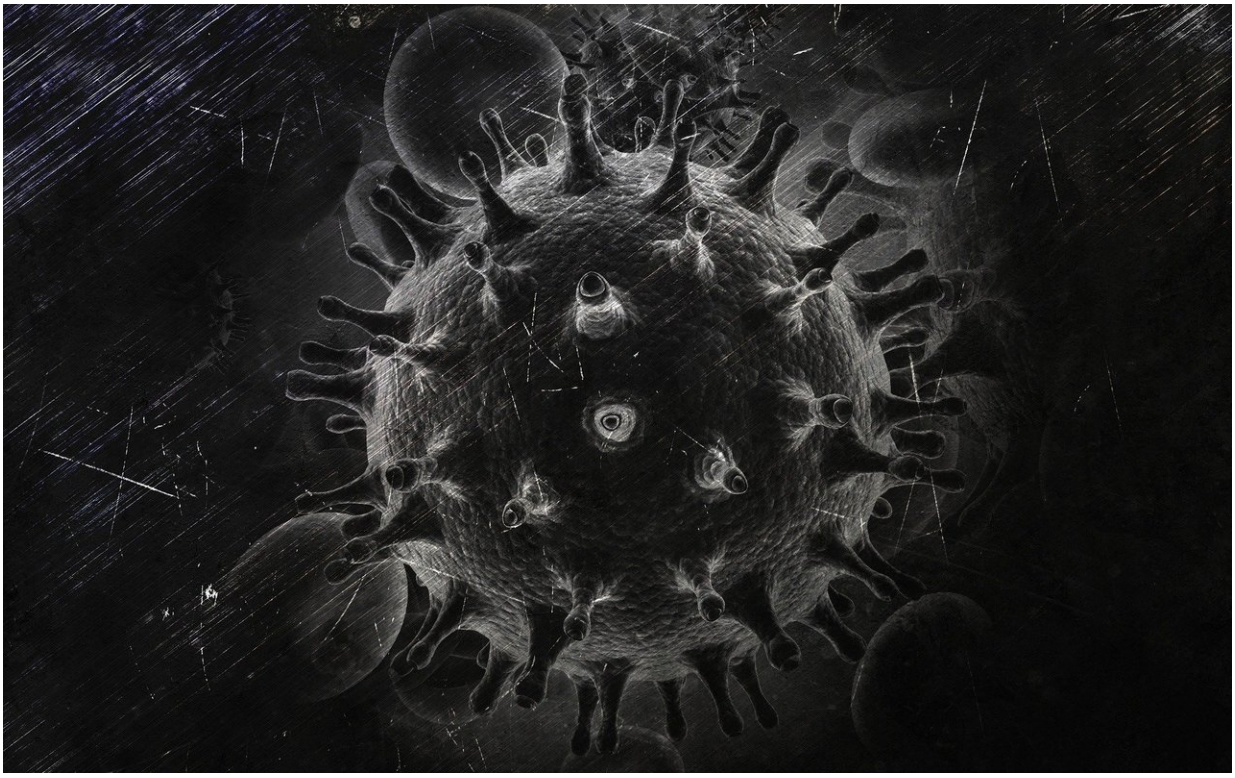

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In the quest to develop a vaccine that triggers the immune system to prevent HIV infection, researchers have focused on identifying and eliciting a particular type of antibody that is capable of neutralizing the virus.

These broadly neutralizing antibodies, or bnAbs, eventually arise naturally in about half of HIV-infected people, but they develop too late to be effective, long after the virus has repeatedly mutated and inserted itself into the genome of host cells.

Searching for a way to elicit bnAbs before HIV infection so they can block the virus if they encounter it, a research team led by the Duke Human Vaccine Institute identified an important protein that is highly active in people who develop bnAbs compared to those who don't.

The protein, called RAB11FIP5, appears to be involved in changing the distribution and function of natural-killer cells, which are among the immune system's early responders during a viral infection. Natural killer cells also play a role in autoimmune diseases, when the body's immune system turns on itself.

"This type of immune cell wasn't previously known to regulate bnAbs," said Barton Haynes, M.D., director of the Duke Human Vaccine Institute and senior author of a study published online Sept. 27 in the journal *Cell*. "We found a new natural killer cell cargo-carrying pathway that appears to be important in regulating the bnAb production."

Haynes and colleagues, including lead author Todd Bradley, Ph.D., designed the study to analyze the molecular differences between HIV-infected people who make bnAbs, and those who don't. They identified 239 infected people, and screened them to find approximately 50 on each extreme—those with the highest numbers of bnAbs, and those with the lowest.

The researchers used RNA sequencing analyses to determine the molecular differences that distinguished between those who produce bnAbs and those who do not, finding a marked discrepancy in the RAB11FIP5 gene expression.

"These data suggest that natural killer cell dysfunction permits bnAb development, implicating Rab11 as a modulator of the HIV antibody response," Bradley said. "This is a new pathway that we hope to modulate during vaccination to generate a better HIV antibody response."

Provided by Duke University Medical Center

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