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UCLA scientists discover immune response to HIV differs, even in identical twins

In findings illustrating the difficulty of developing an AIDS vaccine, UCLA AIDS Institute researchers report the immune systems in two HIV-positive identical twins responded to the infection in different ways.

Detailed in the Dec. 5 issue of the peer-reviewed Journal of Virology (http://jvi.asm.org/), the findings show that the body's defenses against the virus are random rather than genetically determined.

The researchers followed the cases of male twins who were infected shortly after their 1983 births in Los Angeles by blood transfusions administered from the same donor at the same time. Infected with the same strain of the virus, the twins continue to live in the Los Angeles area and grew up exposed to the same environmental forces.

Yet their T-cell receptors (TCR) reacted differently in each twin, showing that the body's defense response was random--and unpredictable. TCRs play an important role in the immune system by binding viruses and other antigens to receptors on their surface, killing the invader. HIV escapes this action by changing shape so that it does not fit into those receptors.

"These boys are as similar as two humans can be, yet we see differences in how they fight the virus," said Dr. Paul Krogstad, professor of pediatrics and pharmacology, and one of the researchers. "That's one more thing that makes it difficult to develop a vaccine for everyone."

When a virus invades a body, the cellular immune response targets small parts of proteins in the virus. This targeting mechanism itself is genetically determined. ". The virus tries to escape that immune response by mutating and changing shape.

The twins' targeting of the HIV was remarkably similar 17 years after infection yet their overall TCR characteristics were highly divergent. The finding, demonstrates that

the interaction between their immune systems and the virus was random and unpredictable--indicating that a "one size fits all" vaccine may not be possible.

"If the goal is to develop a vaccine, our findings suggestthis may not be so straightforward," said Dr. Otto Yang, associate professor of infectious diseases at the David Geffen School of Medicine at UCLA, and the study's lead researcher.

According to the UCLA researchers, the results of this study have broader implications, and could apply to other viruses such as cytomegalovirus (CMV), a herpes virus that causes opportunistic infections in immunosuppressed individuals, and hepatitis C, the latter being similar to HIV in both its changeable and chronic nature.

The study represented collaboration with other UCLA investigators and with Joseph Church of Children's Hospital Los Angeles and the Keck School of Medicine at the University of Southern California. Other researchers from the UCLA AIDS Institute and the David Geffen School of Medicine who contributed to the study are Ryan Kilpatrick, Ayub Ali, Yongzhi Geng, M. Scott Killian, Rachel Lubong Sabado, Hwee Ng, Jeffrey Suen, Yvonne Bryson, Beth D. Jamieson; and Christina M.R. Kitchen, associate professor of biostatistics, UCLA School of Public Health.

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