SOUTHWESTERN NEWS

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STUDY OF MONKEY SPECIES THAT FIGHTS OFF AIDS MAY LEAD TO NEW TREATMENTS FOR HUMANS

DALLAS – March 17, 2003 – A deactivation of the immune system in patients infected with HIV could be one way to inhibit progression to the immunodeficiency diseases associated with AIDS, researchers from UT Southwestern Medical Center at Dallas and Emory University report.

A study comparing the effects of immunodeficiency virus in humans to its effects in sooty mangabey monkeys, which do not become ill when infected, revealed two major differences in the monkeys' responses to the infection. The findings could open the door to groundbreaking approaches to AIDS treatments, said Dr. Donald Sodora, an assistant professor of internal medicine at UT Southwestern who contributed to the study.

The findings are being published online today at www.immunity.com and will appear in a future edition of *Immunity*.

"The mangabeys have just as much virus in their system as during pathogenic HIV infection of humans. The riddle is, they don't get sick," Sodora said. "The idea is, you look at the monkeys and you try to unravel that riddle. And as you unravel it, you can begin to develop new and innovative ideas that have not been explored by others for preventing the progression of AIDS in HIV-infected patients."

Mangabeys exposed to long-term, high-levels of SIV – the version of immunodeficiency virus found in primates – remain healthy and free of any sign of immune deficiency. Researchers found that this lack of symptoms occurred because, unlike humans, these primates have only low-level immune system responses when infected with SIV and do not lose their ability to make new T cells.

T cells, a cornerstone of the immune system, assist other immune cells in eliminating infection. SIV infection in primates and HIV infection in humans both cause a depletion of these cells.

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In contrast to the mangabeys, humans infected with HIV respond with an aberrant activation of the immune system that leads to further destruction of these cells. This depletion is then compounded by the inability of the infected individual to adequately replace the T cells, Sodora said.

"What we call AIDS is actually a combination of the direct effects of HIV replication and the indirect effects brought about by the immune system dysregulation," he said. "In contrast, the absence of the indirect effects in the SIV infected mangabeys can at least be partially attributed to a reduced activation of the immune system, and an ability to maintain continued renewal of T cells."

These findings influence the way in which one might think about treating an AIDS patient or developing a therapy for AIDS, Sodora said.

"It has relevance with regard to how we think about people getting sick with AIDS," he said. "One potential treatment might be an approach to deactivate the immune system, in a very strategic and careful way."

HIV and AIDS began to be identified in the mid-1980s. The virus had been transmitted to humans from primates among which SIV is prevalent. The two types of HIV that exist today originated from two variations of SIV present in chimpanzees (HIV-1) and mangabeys (HIV-2). In both species of primates, the host becomes infected with SIV and replicates the virus in its own T cells, but does not become ill.

Dr. Richard Koup, former chief of infectious diseases at UT Southwestern who is now at the National Institutes of Health Vaccine Research Center, and researchers Guido Silvestri and Mark Feinberg of Atlanta's Emory University School of Medicine also contributed to the NIH-funded study.

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