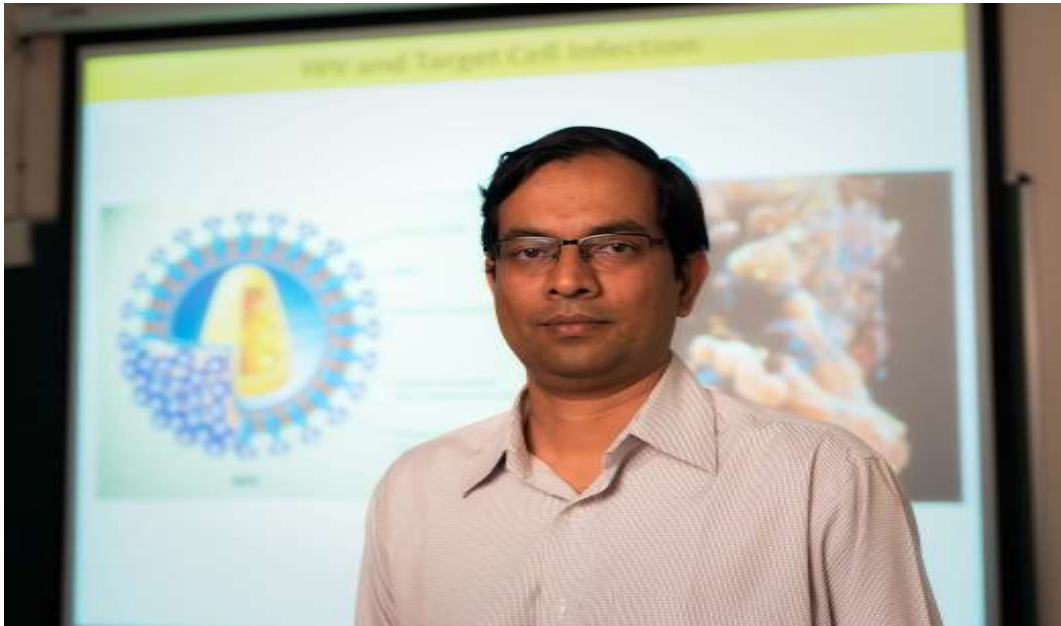


NARENDRA DIXIT (Associate Professor, Department of Chemical Engineering)

BEATING HIV AT ITS OWN GAME

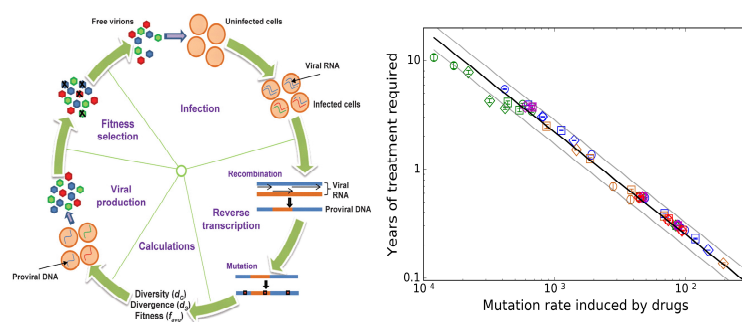


Though the symptoms of Acquired Immuno Deficiency Syndrome (AIDS) can be controlled with a cocktail of anti-retroviral drugs, the disease, caused by the Human Immunodeficiency Virus, or HIV, does not have a permanent cure as yet. The reason researchers have not been successful in this endeavour is because of the high rate at which the virus mutates, causing it to overcome the selection pressures imposed by drugs and potential vaccines.

From the virus's point of view, however, there is a flipside to having very high mutation rates. If it increases beyond a tipping point, called error threshold, then it leads to low fitness in the virus, making it an ineffective pathogen.

Narendra Dixit and his team have been studying the process of infection caused by HIV and its evolution using stochastic simulation models. These models allow them to

closely track the fitness levels of the genetically diverse populations within a host and the factors that contribute to it. A recent study by Dixit and his student, Vipul Gupta,



has demonstrated that if the mutation rate of HIV is increased over six times its natural mutation rate then the virus can be rendered inactive against its host.*

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The finding also has implications for how we treat AIDS. High mutation rates can be induced in HIV using a special class of anti-retroviral drugs. If the mutation rate can be raised to the error threshold, then, in principle, the virus can be prevented from causing infection. The research by Dixit's team shows that, in fact, such a benign state can be achieved by using mutation-inducing drugs for a period of about 10 years. Some of these drugs are already undergoing clinical trials. Dixit believes that in the coming years, with better drug combinations, the treatment window could be reduced further.



* Gupta, V, Dixit, N. 2015. Scaling law characterizing the dynamics of the transition of HIV-1 to error catastrophe. *Phys. Biol.* 12(5): 054001