The year was 1996. Drug companies were rushing to the market with the most effective drugs yet against the dreaded AIDS virus. These protease inhibitors, despite their side effects and high cost, would be the first to ease symptoms dramatically and improve the prognosis for infected patients. During that same year, competing scientific groups made a series of discoveries that could open a new chapter in treating -- and preventing -- infection by the lethal organism. For the first time, the treatments would be aimed not at the virus itself, but at a portal through which HIV enters white blood cells and attacks the immune system.

Scientists in 1996 identified the portal: It was a large protein called Chemokine Receptor 5, or CCR-5, which is a normal part of the cell produced by a gene having the same name. When this gene mutates, it produces the protein incorrectly. In fact, when people inherit two copies of the mutant CCR-5 gene, the cell fails to make the portal protein at all. It simply does not exist, and the cell is like a house with no front door.

The finding was "pretty stunning," said Stephen O'Brien of the National Cancer Institute. He had been searching for years for genetic changes that conferred HIV resistance, and here was a powerful one.

Perhaps best of all, people can get along fine without a functioning CCR-5 gene or protein. That makes CCR-5 an excellent "target" for drugs. A compound that blocks the CCR-5 protein might prevent HIV infection as if a person had naturally inherited the mutation.

This discovery has not yet translated into new treatments. A dozen pharmaceutical companies, however, are developing drugs designed to block HIV's entrance into the white blood cell by creating a non-functional CCR-5 receptor. Already, some of them are in clinical trials in HIV patients, perhaps laying the groundwork for the most effective treatments yet.

Questions for Discussion

1. How did Dr. David Ho confirm that Steve Crohn's cells were immune to HIV infection?

2. How does the human immunodeficiency virus (HIV) get inside cells? What kind of cells does HIV infect?

3. What gene underlies natural immunity to HIV? How does the mutant form of the gene prevent HIV from getting inside cells?