The genetic scripts written in DNA code constantly undergo changes, or mutations. At times, these mistakes in a gene's message can be harmful; often, they have no significant effect. Occasionally, though, a mutation confers a survival advantage in the face of an environmental change. Most of the non-carriers of the mutation die, and those with the mutation are able to reproduce. With that powerful evolutionary selection force, the gene can become common in a population.

Recently, scientists were astonished to find that some individuals did not become infected with HIV, even after repeated exposure to the deadly virus.

For some reason, they were immune. A long and difficult scientific search, using blood samples from hundreds of HIV-resistant patients, finally teased out the genetic explanation. Resistant individuals had in their cells two copies of a mutation that disrupted the entryway through which HIV viruses entered white blood cells. People who inherited just one copy of the change could become infected, but their disease progressed more slowly.

With this being such a recent epidemic, where did peoples' immunity come from?

Another puzzle was the way this resistance is distributed throughout the world. In some Northern European populations it is relatively common. In Southern Europeans it is more rare, and it is almost entirely absent in Africans, Asians, and Native Americans. Logically, the mutation must have occurred in the past, acting as a defense against a different, previous epidemic caused -- like the AIDS epidemic -- by a pathogen that also targeted white blood cells.

Reading a chronological history, biologists traced the HIV-resistance gene mutation back about 700 years. That was the time at which the Black Death -- bubonic plague -- swept like a deadly scythe through Europe, killing one-third of the population. Then, as now, there were individuals who survived the lethal organism, perhaps because it could not enter their white blood cells. The areas that were hardest hit by the Black Plague match those where the gene for HIV resistance is the most common today.

At present, scientists are trying to infect such resistant cells with bubonic plague bacteria to test the hypothesis that the mutation in the CCR-5 receptor gene could have thwarted the plague in the Middle Ages, as it does HIV today. If it turns out that this mutation does protect against the plague, this coincidence will be yet another illustration of what scientists are finding over and over in the human genome: Nature's past successes often remain part of our genetic toolbox.
Questions for Discussion

1. A single gene mutation offers some European Caucasians immunity to human immunodeficiency virus (HIV). How does the gene mutation change cells to block HIV from entering them?

2. From your perspective, can you think of any general method O'Brian's team could determine if Europeans who died during the black plague were missing the genetic mutation for HIV immunity?

3. Do you think a genetic test should be developed so people can find out whether they are immune to HIV? What might be the pros and cons of such a test?