

IN 1918. THE FINAL YEAR of the savage trench fighting of World War I, something else began felling the soldiers. No one knows for sure when or where the Spanish flu emerged, though it certainly wasn't in Spain. As a neutral country, Spain had no wartime censorship, and the flu apparently got its false pedigree from news reports about outbreaks there in May 1918. In fact the disease was already spreading on both sides of the European front, laying low entire divisions through the spring and early summer. Then it seemed to subside.

In late summer, though, the Spanish flu returned, and this time its virulence was unmistakable. The sick took to their beds with fever, piercing headache, and joint pain. Many were young adults, exactly the group that normally shrugs off the flu. About 5 percent of the victims died, some in just two or three days, their faces turning a ghastly purple as they essentially suffocated to death. Doctors who opened the chests of the dead were horrified: The lungs, normally light and elastic, were as heavy as water-logged sponges, clogged with bloody fluid.

After flashing through crowded military camps and troopships in Europe and the United States, the flu leaped out of uniform to ports and industrial cities. In Philadelphia, historian Alfred Crosby found, 12,000 people died of flu and pneumonia in October—759 in a single day. Schools and businesses were shut down and church services cancelled. Morgues overflowed.

By then the sickness had spread to the far corners of the planet, from the South Pacific to the Arctic. "Everybody on Earth breathed in the virus, and half of them got sick," says Jeffery Taubenberger of the Armed Forces Institute of Pathology in Maryland, who is trying to learn what made it such a killer. More than 50 million people died—at least three times as many as in the war. The best medical minds of the day could hardly believe that this was flu.

It was flu all right, but with a crucial difference that scientists are only beginning to understand. Scattered across Taubenberger's desk are translucent wax blocks the size of matchboxes. Borrowed from a pathology archive, they hold fingernail-size scraps of purplish tissue, sliced from the lungs of flu victims in U.S. military hospitals almost 90 years ago. In the mid-1990s Taubenberger and his colleagues realized that a sample from someone who died quickly, lungs still seething with virus, might still hold genetic traces of the killer. They were right: In 1996 lung tissue from a soldier who died in September 1918 at Fort Jackson, South Carolina, yielded pieces of the virus's genes.

The pickings soon got better. Inspired by Taubenberger's discovery, a retired pathologist named Johan Hultin traveled to a remote Alaska village and excavated a mass grave that had been hacked into the permafrost after the Spanish flu swept through in November 1918. One female body still contained intact lung tissue, preserved by the cold and sheer luck. Bit by bit, Taubenberger's group teased out the entire genetic sequence of the virus. They plan to finish publishing it this year.

So far this genetic blueprint hasn't revealed exactly what made the Spanish flu so deadly. No single gene or protein is the obvious culprit. But comparing the 1918 sequence to those of the flu viruses that wreak mild havoc each winter has confirmed what was long suspected:

The Spanish flu virus had recently crossed into people from some unknown animal, leaving victims with little immunity to this new threat.

1918 Flu
National Geographic
October 2005
"The Next Killer Flu"
page 12

