SARS: A Pandemic Prevented

In retrospect, it almost seems like a bad dream. When severe acute respiratory syndrome (SARS) roared onto the world scene in March, the fatal, contagious disease seemed unstoppable. Experts warned that it could ignite a pandemic. And then, in early July … gone. The nightmare never became a reality.

The outbreak was a chilling reminder that new infectious diseases are always lurking in the woodwork—and they needn't sicken a lot of people to bring economies to their knees. The episode gave the World Health Organization (WHO) new prominence and showcased public health and science at their best in response to a new challenge—and occasionally at their worst.

In the line of duty. WHO's Carlo Urbani died from SARS after warning the world.

SARS made its debut in November 2002, in the Chinese province of Guangdong, but the Chinese government remained silent about it for months, contributing to its spread. Officials remained in a state of denial even when SARS had started its march across the world from a hotel in Hong Kong. By sticking to a reassuring but false hypothesis—that the disease was caused by more familiar *Chlamydia* bacteria—and discouraging alternative views, the Chinese government prevented its scientists from playing what could have been a pivotal role in the discovery of the new virus.

On the upside, SARS inspired intense collaboration among laboratories worldwide hunting for the real culprit. There was plenty of rivalry and the occasional squabble behind the scenes. But sharing information, specimens, and reagents definitively nailed the agent, a member of the coronavirus family, only 5 weeks after WHO had sounded a global alarm. By that time, the virus's entire genome had already been sequenced.

But in the end it wasn't science that brought SARS under control. Centuries-old measures, such as strict isolation of patients, eventually cornered the virus after more than 8000 reported cases and 774 reported deaths. For doctors and nurses charged with that task, simply going to work took a great deal of courage. Some, including Carlo Urbani, a WHO official in Hanoi who first sounded the alarm, paid with their lives.

Six months later, many questions remain. Perhaps the biggest ones are "Where did SARS come from?" and "Will it come back?" Apart from a Singapore scientist who became infected in the lab in August, no new cases have been reported since July, and most scientists agree that it's unlikely that SARS is going around unnoticed. But the virus's natural reservoir is still unknown, and it's unclear whether its leap to humans was caused by an extremely unlikely event—say, a freak set of mutations—or something that could happen again any day.

Even if SARS does not return, its impact will last. The hunt for the virus set a new standard for the next emergency and helped assert WHO's leadership. The agency is fortifying its Global Outbreak Alert and Response Network—a loose affiliation of labs and institutions that have agreed to respond to emerging threats—and it's pushing hard for the adoption of the Global Health Regulations, a treaty that would oblige member states to act quickly and speak up about dangerous outbreaks within their borders. Meanwhile, the United States and several other countries are taking a critical look at often-antiquated quarantine laws and rethinking the balance between public health and civil liberties.

Few doubt that there's more to come. An Institute of Medicine report released in March concluded that with ecological disruption increasing, populations expanding, public health breaking down, and travel and trade booming, the risk of new SARS-like outbreaks, if not of SARS itself, is only increasing.

–MARTIN ENSERINK

somes come in pairs. When a gene on one partner goes bad, it can be replaced with a copy of the other partner's good gene. A genomic loner, Y appeared to have no way to prevent mutations from gradually destroying its genes. That's where the palindromes come in: Genes on one end of a palindrome can replace mutated twins on the other end.

By sequencing parts of the Y chromosomes of other primates, researchers now know that at least six of the palindromes predate the evolution of humans and arose more than 5 million years ago. Thus it seems that gene swapping between palindrome arms keeps the Y chromosome's genetic makeup stable.

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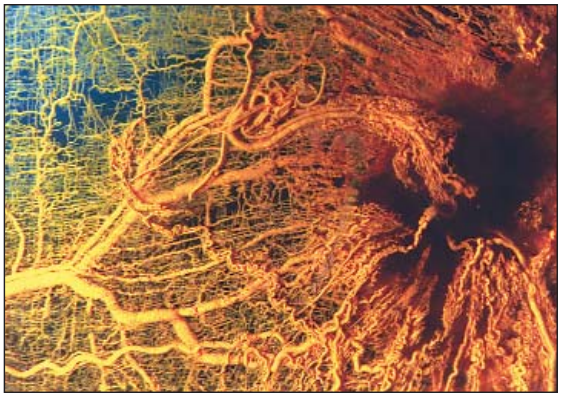
Starving cancer. It's been a roller-coaster ride for researchers working on anticancer drugs that block development of the blood vessels that feed tumor growth. They've seen their field bounce from obscurity and skepticism to superhype—after a 1998 article in *The New York Times* suggested that antiangiogenesis drugs, as they are called, would cure cancer in 2 years—and then back to skepticism when early clinical trials produced unimpressive results. But this year, they've finally begun to see their efforts pay off.

The drugs' premise is simple. As a cancerous tumor grows, it must chemically induce the growth of new blood vessels to supply it with nutrients. Antiangiogenic agents starve tumors by preventing this blood vessel growth. Numerous agents, both naturally occurring proteins and synthetic drugs, shrink tumors in lab animals, but they had not been able to meet the "gold standard" of clinical cancer trials: extending the lives of patients.



Angiogenesis in action. Blood vessels grow toward a dark sarcoma tumor.

But this June, researchers announced that an antiangiogenesis drug, given with conventional chemotherapy drugs in a large clinical trial, prolonged the lives of patients with advanced colon cancer. The drug had failed a similar test with breast cancer patients, possibly because advanced breast tumors produce more angiogenesis-promoting factors than colon tumors do and are thus harder to control. This suggests that antiangiogenesis therapies will have to be tailored to their targets to be effective.

Researchers have also learned that antiangiogenesis drugs work most effectively in combination, either with each other or with conventional chemotherapeutic drugs or radiation. And clinicians will have plenty of drugs to choose from. Some 60 different antiangiogenesis drugs are currently in clinical trials against a wide variety of cancers, and many more are in preclinical testing.

–THE NEWS AND EDITORIAL STAFFS

CREDITS: (TOP TO BOTTOM) GUIDO PICCHIO/AP; L. HEUSER AND R. ACKLAND/UNIVERSITY OF LOUISVILLE SCHOOL OF MEDICINE