EBOLA

8

MORMONISM

WE ARE taking quite a leap. We are leaving the "Isms" which have been more or less directly influenced from the East. We are bidding farewell to the out and out pantheistic cults (although there are many more). And we are turning to "Isms" of a simon-pure American make. Among these Mormonism, Seventh-Day Adventism, and Russellism are perhaps the most persistent propagandists; hence we intend to devote considerable attention to these three.

Mormonism is a marvelously composite faith. It has developed over a period of time, and as it went along it took over some of the most divergent elements from other sects and groups. They brewed, to speak with Mr. Ferguson, a synthetic religion in Utah.

HISTORY

The Mormon "prophet" Joseph Smith, Jr., was born on December 23, 1805, in Sharon, Vermont. He was reared in ignorance, poverty and superstition. Moreover, he was indolent in his youth. However, quite in keeping with the superstitious atmosphere in which he breathed, he claimed to have visions and divine revelations as early as 1820 and 1823. In the latter year the angel Moroni revealed to him the spot where golden plates lay buried containing the history of ancient America in "reformed-Egyptian *caractors*." Smith undoubtedly meant *characters*, but, unlike Mother Eddy, he had never known enough grammar for it to be "eclipsed" by a divine revelation; hence he made occasional grammatical errors.

In 1830 "Joe," as he was known, organized the "Church of Jesus Christ of Latter-Day Saints" at Fayette, N. Y. This

he accomplished after having convinced a few friends that his "translation" of the Golden Plates—afterward duly returned into the hands of the angel Moroni—had been done, not as was maliciously slandered, with the aid of "a peepstone in a hat," but with the assistance of the proper "Urim and Thummim" which the obliging angel had provided. The plates are stated to have been hidden in the earth from the year 420 of our era until September 22, 1823, when "Joe Smith" discovered them in the "Hill Cumorah"; and yet the Book of Mormon, being a faithful rendering of the said plates, gives extensive quotations from the Bible in —: the King James Version! It contains modern phrases and ideas that could not have been known to its supposed author in 420 A. D. It puts the words of Jesus (though often twisted) into the mouths of men alleged to have lived centuries before Christ. It was not only written in a poor imitation of Biblical style; it also undermines the Bible by declaring it *insufficient*, by *adding to and changing many Biblical passages*, "by divine revelation." For such reasons as these it could hardly have been revealed by an angel. Its story of the ancient inhabitants of America, the supposed ancestors of the "Latter Day Saints," contains twelve historical errors.

The Book of Mormon is officially recognized by both branches of Mormonism as of equal authority with the Bible, and practically receives honor far beyond the Bible. But there is an abundance of incontestable evidence that the origin of the Book of Mormon must be sought in Solomon Spaulding's unpublished and stolen novel, *The Manuscript Found*. The Mormons try to obliterate this evidence by referring to another manuscript, *The Manuscript Story*, by the same Spaulding; they prove that the Book of Mormon is not a copy of the latter manuscript. The unknowing are thus convinced that Joseph Smith did not copy from "*the* Spaulding manuscript"; but the real argument, that the "Golden Bible" is the work of copying and embellishing by Rigdon and Smith, remains unanswered.

In June, 1831, a "revelation" commanded the Saints to settle in Missouri, the "land of Zion." Kirtland, Ohio, and

Emerging Viruses

Hemorrhagic fever viruses are among the most dangerous biological agents known. New ones are discovered every year, and artificial as well as natural environmental changes are favoring their spread

by Bernard Le Guenno

In May 1993 a young couple in New Mexico died just a few days apart from acute respiratory distress. Both had suddenly developed a high fever, muscular cramps, headaches and a violent cough. Researchers promptly started looking into whether similar cases had been recorded elsewhere. Soon 24 were identified, occurring between December 1, 1992, and June 7, 1993, in New Mexico, Colorado and Nevada. Eleven of these patients had died.

Bacteriological, parasitological and virological tests conducted in the affected states were all negative. Samples were then sent to the Centers for Disease Control and Prevention (CDC) in Atlanta. Tests for all known viruses were conducted, and researchers eventually detected in the serum of several patients antibodies against a class known as hantaviruses. Studies using the techniques of molecular biology showed that the patients had been infected with a previously unknown type of hantavirus, now called Sin Nombre (Spanish for "no name").

New and more effective analytical techniques are identifying a growing number of infective agents. Most are viruses that 10 years ago would probably have passed unnoticed or been mistaken for other, known types. The Sin Nombre infections were not a unique occurrence. Last year a researcher at the Yale University School of Medicine was accidentally infected with Sabià, a virus first isolated in 1990 from an agricultural engineer who died from a sudden illness in the state of São Paulo, Brazil.

Sabià and Sin Nombre both cause illnesses classified as hemorrhagic fevers. Patients initially develop a fever, followed by a general deterioration in health during which bleeding often occurs. Superficial bleeding reveals itself through skin signs, such as petechiae (tiny releases of blood from vessels under the skin surface), bruises or purpura (characteristic purplish discolorations). Other cardiovascular, digestive, renal and neurological complications can follow. In the most serious cases, the patient dies of massive hemorrhag-

PATRICK ROBERT SYGMA

es or sometimes multiple organ failure.

Hemorrhagic fever viruses are divided into several families. The flaviviruses have been known for the longest. They include the Amaril virus that causes yellow fever and is transmitted by mosquitoes, as well as other viruses responsible for mosquito- and tick-borne diseases, such as dengue. Viruses that have come to light more recently belong to three other families: arenaviruses, bunyaviruses (a group that includes the hantaviruses) and filoviruses. They have names like Puumala, Guanarito and Ebola, taken from places where they first caused recognized outbreaks of disease.

All the arenaviruses and the bunyaviruses responsible for hemorrhagic fevers circulate naturally in various populations of animals. It is actually uncommon for them to spread directly from person to person. Epidemics are, rather, linked to the presence of animals that serve as reservoirs for the virus and sometimes as vectors that help to transfer it to people. Various species of rodent are excellent homes for these viruses, because the rodents show no signs when infected. Nevertheless, they shed viral particles throughout their lives in feces and, particularly, in urine. The filoviruses, for their part, are still a mystery: we do not know how they are transmitted.

Hemorrhagic fever viruses are among the most threatening examples of what are commonly termed emerging pathogens. They are not really new. Mutations or genetic recombinations between existing viruses can increase virulence, but what appear to be novel viruses are generally viruses that have existed for millions of years and merely come to light when environmental conditions change. The changes allow the virus to multiply and spread in host organisms. New illnesses may then sometimes become apparent.

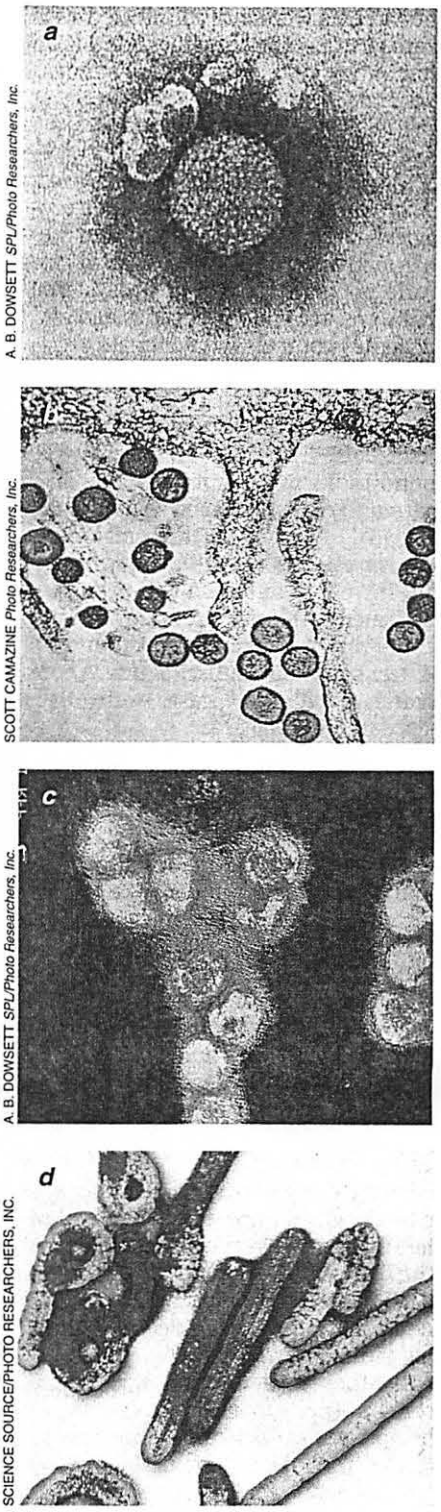
Improvements in Diagnosis

The seeming emergence of new viruses is also helped along by rapid advances in the techniques for virological identification. The first person diagnosed with Sabià in São Paulo (called the index case) was originally thought to be suffering from yellow fever. The agent actually responsible was identified only because a sample was sent to a laboratory equipped for the isolation of viruses. That rarely happens, because most hemorrhagic fever viruses circulate in tropical regions, where hospitals generally have inadequate diagnostic equipment and where many sick people are not hospitalized. Even so, the rapid identification of Sin Nombre was possible only because of several years of work previously accumulated on hantaviruses.

Hantaviruses typically cause an illness known as hemorrhagic fever with renal syndrome; it was described in a Chinese medical text 1,000 years ago. The West first became interested in this illness during the Korean War, when more than 2,000 United Nations troops suffered from it between 1951 and 1953. Despite the efforts of virologists, it was not until 1976 that the agent was identified in the lungs of its principal reservoir in Korea, a field mouse. It took more than four years to isolate the virus, to adapt it to a cell culture and to prepare a reagent that permitted a diagnostic serological test, essential steps in the study of a virus. It was named Hantaan, for a river in Korea. The virus also circulates in Japan and Russia, and a similar virus that produces an illness just as serious is found in the Balkans.

A nonfatal form exists in Europe. It was described in Sweden in 1934 as the "nephritic epidemic," but its agent was not identified until 1980, when it was detected in the lungs of the bank vole. Isolated in 1983 in Finland, the virus was named Puumala for a lake in that country. Outbreaks occur regularly in northwestern Europe. Since 1977, 505 cases have been recorded in northeastern France alone. The number of cases seems to be increasing, but this is probably because doctors are using more biological tests than formerly, and because the tests in recent years have become more sensitive.

Thus, it is only for about a decade that we have had the reagents necessary to identify hantaviruses. Thanks to these reagents and a research technique that spots antibodies marking recent infections, scientists at the CDC in 1993 were quickly on the track of the disease. The presence of specific antibodies is not always definite proof of an infection by the corresponding pathogen, however. False positive reactions and cross-reactions caused by the presence of antibodies shared by different viruses are possible. A more recent technology, based on the polymerase chain reaction, permits fragments of genes to be amplified (or duplicated) and sequenced. It provided confirmation that



HEMORRHAGIC FEVER VIRUSES vary greatly in appearance under the electron microscope. Lassa (*a*), found in Africa, is an arenavirus, a kind that is typically spherical. Hantaviruses (*b*) cause diseases of different varieties in many regions of the world. Tick-borne encephalitis virus (*c*) is an example of a flavivirus, a group that includes yellow fever and dengue. Ebola (*d*) is one of the filoviruses, so called because of their filamentous appearance. The images have been color-enhanced.

ZAIREAN RED CROSS members bury victims of the Ebola virus in Kikwit earlier this year. At least 190 died in the epidemic. Poor medical hygiene and unsafe funeral practices helped to propagate the infection.

PATRICK ROBERT SYGMA

the patients were indeed infected with hantaviruses. The identification of Sin Nombre took no more than eight days.

The Infective Agents

The primary cause of most outbreaks of hemorrhagic fever viruses is ecological disruption resulting from human activities. The expansion of the world population perturbs ecosystems that were stable a few decades ago and facilitates contacts with animals carrying viruses pathogenic to humans. This was true of the arenavirus Guanarito, discovered in 1989 in an epidemic in Venezuela. The first 15 cases were found in a rural community that had started to clear a forested region in the center of the country. The animal reservoir is a species of cotton rat; workers had stirred up dust that had been contaminated with dried rat urine or excrement—one of the most frequent modes of transmission. Subsequently, more than 100 additional cases were diagnosed in the same area.

Other arenaviruses responsible for hemorrhagic fevers have been known for a long time—for example, Machupo, which appeared in Bolivia in 1952, and Junín, identified in Argentina in 1958. Both those viruses can reside in species of rodents called vesper mice; the Bolivian species enters human dwellings. Until recently, an extermination campaign against the animals had prevented any human infections with Machupo since 1974. After a lull of 20 years, however, this virus has reappeared, in the same place: seven people, all from one family, were infected during the summer of 1994.

Junín causes Argentinian hemorrhagic fever, which appeared at the end of the 1940s in the pampas west of Buenos Aires. The cultivation of large areas of maize supported huge populations of the species of vesper mice that carry this virus and multiplied contacts between these rodents and agricultural workers. Today mechanization has put the operators of agricultural machinery on the front line: combine harvesters not only suspend clouds of infective dust, they also create an aerosol of infective blood when they accidentally crush the animals.

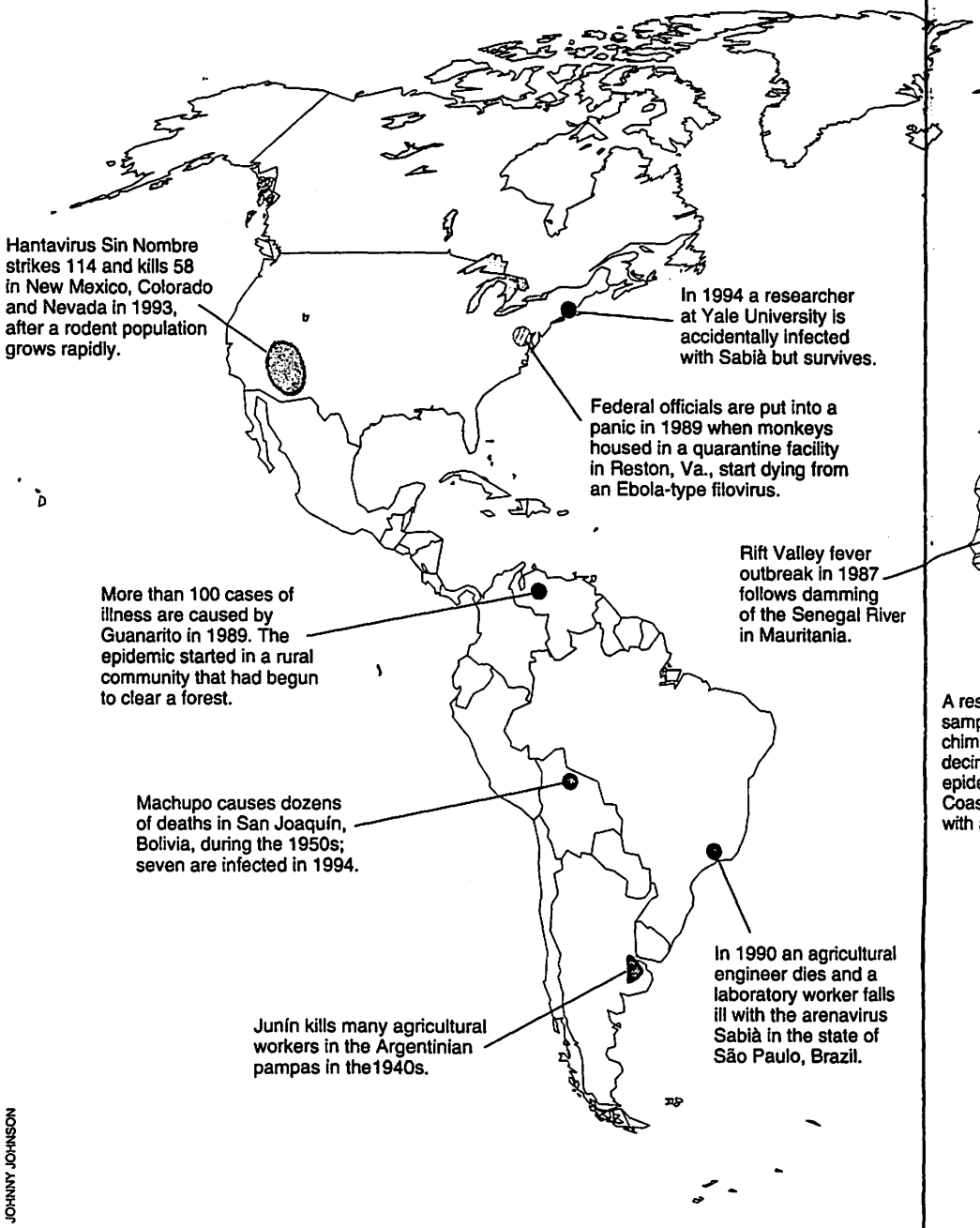
The arenavirus Sabià has, so far as is known, claimed only one life, but other cases have in all probability occurred in Brazil without being diagnosed. There is a real risk of an epidemic if agricultural practices bring the inhabitants of São Paulo into contact with rodent vectors. In Europe, the main reservoirs of the hantavirus Puumala—the bank vole and yellow-necked field mouse—are woodland animals. The most frequent route of contamination there is inhalation of contaminated dust while handling wood gathered in the forest or while working in sheds and barns.

Humans are not always the cause of dangerous environmental changes. The emergence of Sin Nombre in the U.S. resulted from heavier than usual rain and snow during spring 1993 in the mountains and deserts of New Mexico, Nevada and Colorado. The principal animal host of Sin Nombre is the deer mouse, which lives on pine kernels: the exceptional humidity favored a particularly abundant crop, and so the mice proliferated. The density of the animals multiplied 10-fold between 1992 and 1993.

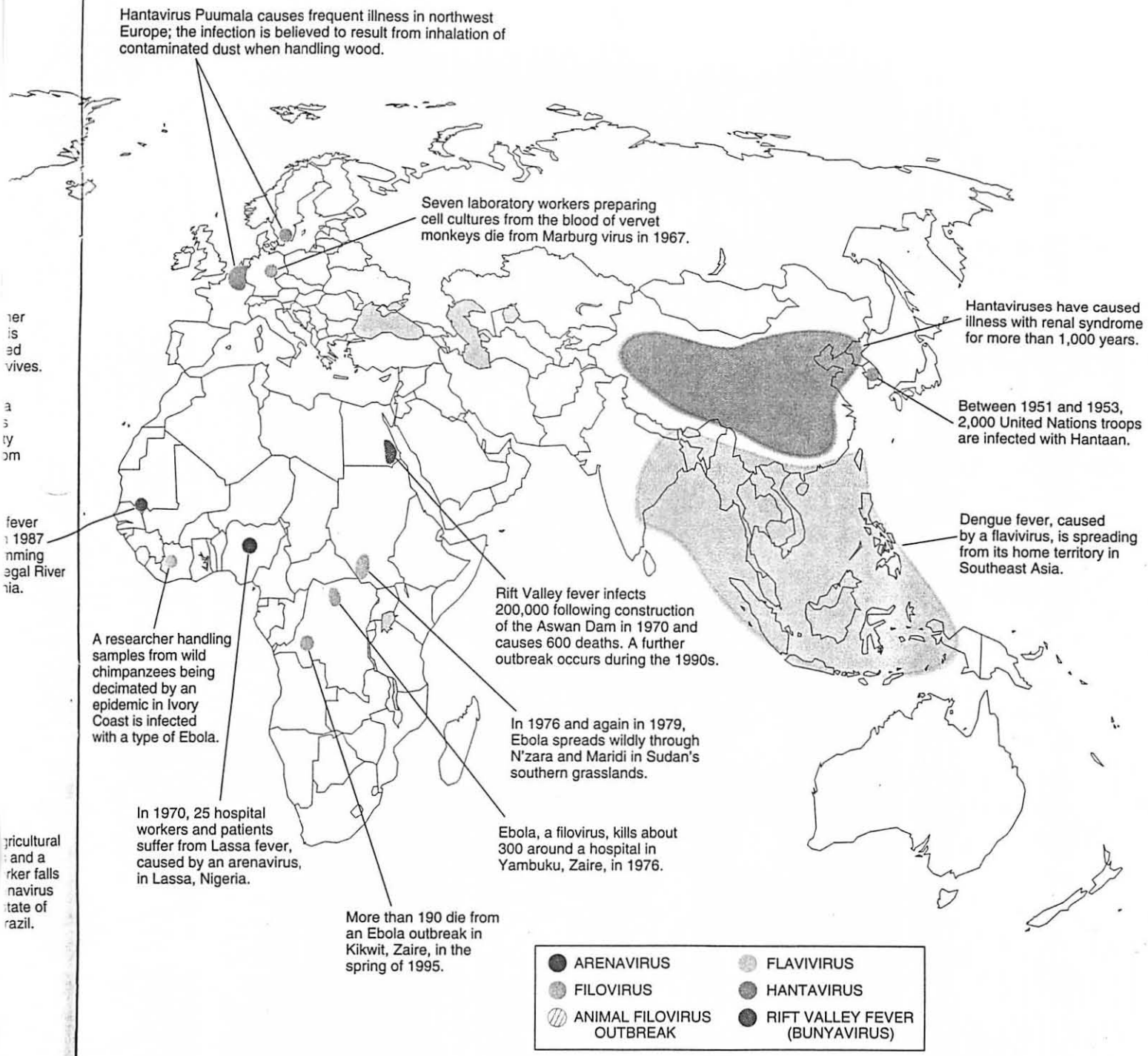
Transmission by Mosquitoes

Some bunyaviruses are carried by mosquitoes rather than by rodents. Consequently, ecological perturbations such as the building of dams and the expansion of irrigation can encourage these agents. Dams raise the water table, which favors the multiplication of

Global Reach of Hemorrhagic Fever Viruses



JOHNNY JOHNSON



the insects and also brings humans and animals together in new population centers. These two factors probably explain two epidemics of Rift Valley fever in Africa: one in 1977 in Egypt and the other in 1987 in Mauritania.

The virus responsible was recognized as long ago as 1931 as the cause of several epizootics, or animal epidemics, among sheep in western and South Africa. Some breeders in contact with sick or dead animals became infected, but at the time the infection was not serious in humans. The situation became more grim in 1970. After the construction of the Aswan Dam, there were major losses of cattle; of the 200,000 people infected, 600 died. In 1987 a minor epidemic followed the damming of the Senegal River in Mauritania.

Rift Valley fever virus is found in several species of mosquitoes, notably those of the genus *Aedes*. The females transmit the virus to their eggs. Under dry conditions the mosquitoes' numbers are limited, but abundant rain or irrigation allows them to multiply rapidly. In the course of feeding on blood, they then transmit the virus to humans, with cattle acting as incubators.

Contamination by Accident

Although important, ecological disturbances are not the only causes of the emergence of novel viruses. Poor medical hygiene can foster epidemics. In January 1969 in Lassa, Nigeria, a nun who worked as a nurse fell ill at work. She infected, before dying, two other nuns, one of whom died. A year later an epidemic broke out in the same hos-

pital. An inquiry found that 17 of the 25 persons infected had probably been in the room where the first victim had been hospitalized. Lassa is classed as an arenavirus.

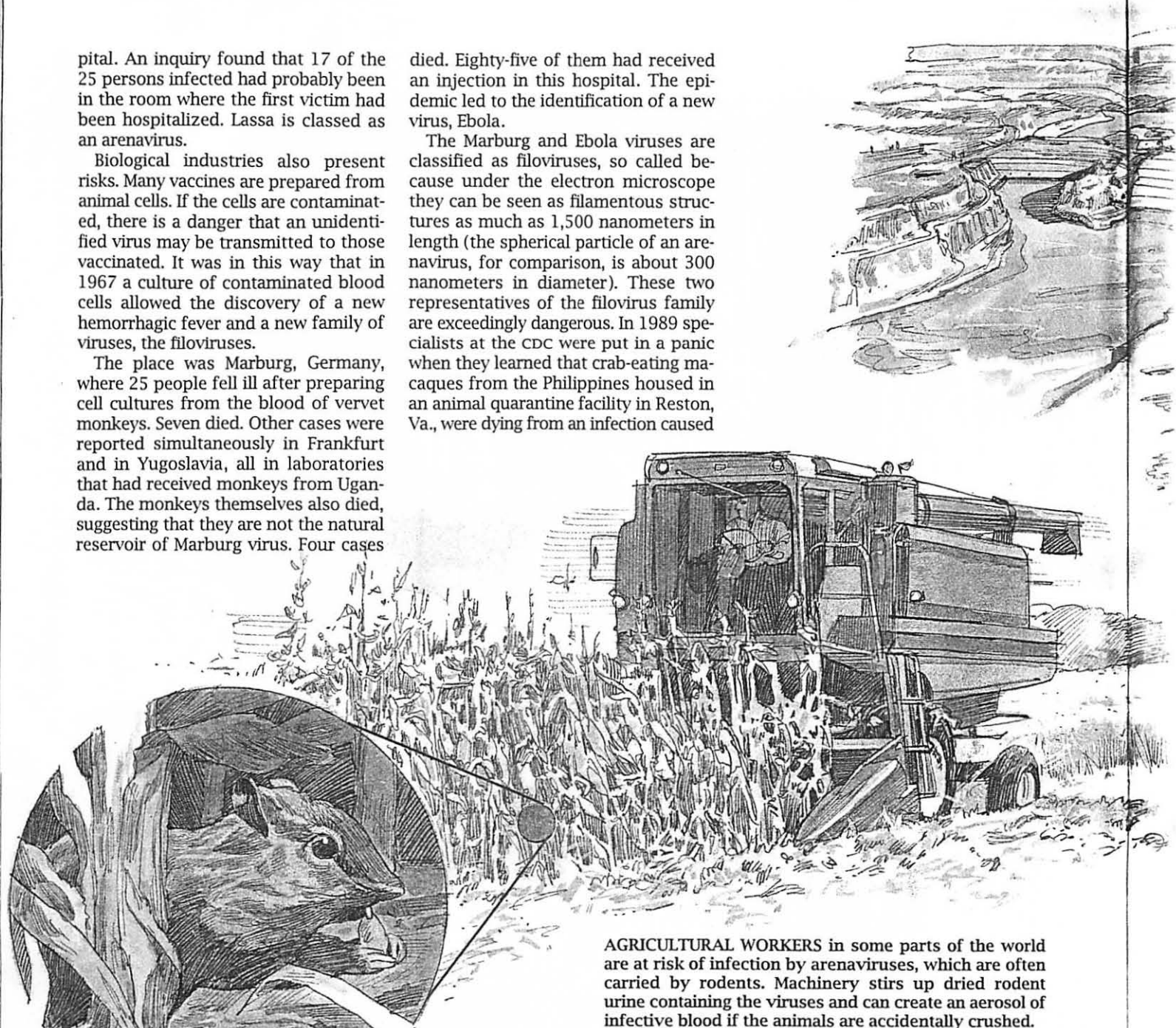
Biological industries also present risks. Many vaccines are prepared from animal cells. If the cells are contaminated, there is a danger that an unidentified virus may be transmitted to those vaccinated. It was in this way that in 1967 a culture of contaminated blood cells allowed the discovery of a new hemorrhagic fever and a new family of viruses, the filoviruses.

The place was Marburg, Germany, where 25 people fell ill after preparing cell cultures from the blood of vervet monkeys. Seven died. Other cases were reported simultaneously in Frankfurt and in Yugoslavia, all in laboratories that had received monkeys from Uganda. The monkeys themselves also died, suggesting that they are not the natural reservoir of Marburg virus. Four cases of natural infection with Marburg have been reported in Africa, but neither the reservoir nor the natural modes of transmission have been discovered. What is clear is that Marburg can propagate in hospitals: secondary cases have occurred among medical personnel.

In 1976 two epidemics of fever caused by a different virus occurred two months apart in the south of Sudan and in northern Zaire. In Zaire, around Yambuku Hospital, by the Ebola River, 318 cases were counted, and 280 persons died. Eighty-five of them had received an injection in this hospital. The epidemic led to the identification of a new virus, Ebola.

The Marburg and Ebola viruses are classified as filoviruses, so called because under the electron microscope they can be seen as filamentous structures as much as 1,500 nanometers in length (the spherical particle of an arenavirus, for comparison, is about 300 nanometers in diameter). These two representatives of the filovirus family are exceedingly dangerous. In 1989 specialists at the CDC were put in a panic when they learned that crab-eating macaques from the Philippines housed in an animal quarantine facility in Reston, Va., were dying from an infection caused by an Ebola-type filovirus. The virus was also isolated from other animal facilities that had received monkeys from the Philippines. No human illnesses were recorded in the wake of this epizootic, however, which demonstrates that even closely related viruses can vary widely in their effects.

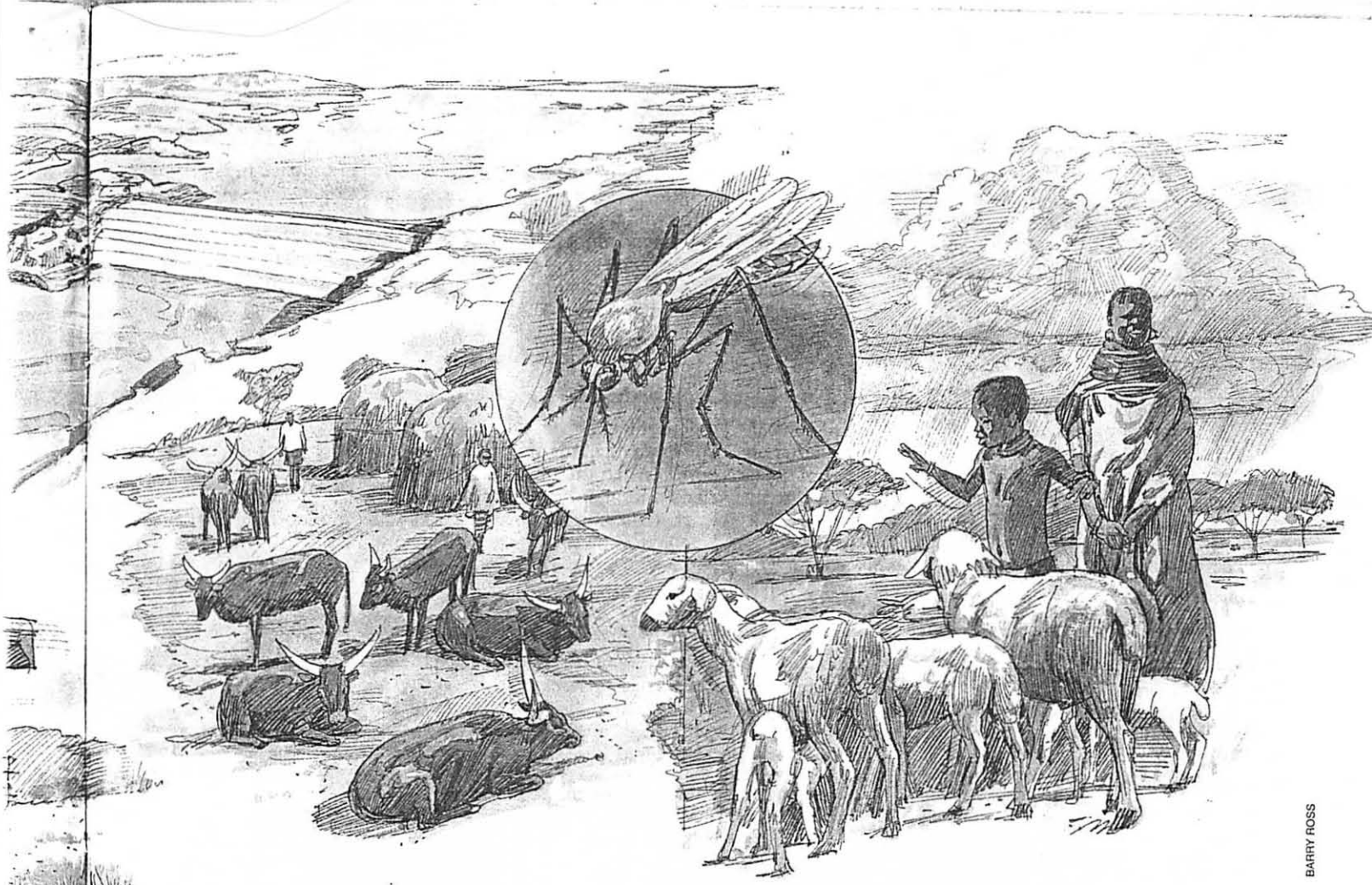
In January of this year we isolated a previously unknown type of Ebola from a patient who had infected herself handling samples from wild chimpanzees that were being decimated by a strange epidemic. That the chimpanzees, from Ivory Coast, succumbed is further evidence that primates are not filoviruses' natural reservoir, which has not yet been identified. Although Marburg has infected few people, Ebola surfaced again to cause a human epidemic in Zaire this past May [*see box on pages 62 and 64*].



AGRICULTURAL WORKERS in some parts of the world are at risk of infection by arenaviruses, which are often carried by rodents. Machinery stirs up dried rodent urine containing the viruses and can create an aerosol of infective blood if the animals are accidentally crushed.

A Shifting, Hazy Target

The extreme variability and speed of evolution found among hemorrhagic fever viruses are rooted in the nature of their genetic material. Hemorrhagic fever viruses, like many other types, generally have genes consisting of ribonucleic acid, or RNA, rather than the DNA employed by most living things. The RNA of these viruses is "negative stranded"—before it can be used to make viral proteins in an infected cell, it must be converted into a positive



BARRY ROSS

RIFT VALLEY FEVER VIRUS, a bunyavirus, is transmitted by mosquitoes from cattle and sheep to humans. Dams allow multiplication of the insects by raising the water table and bring people and animals together in new locations, causing epidemics.

strand by an enzyme called RNA polymerase. RNA polymerases cause fairly frequent errors during this process. Because the errors are not corrected, an infected cell gives rise to a heterogeneous population of viruses resulting from the accumulating mutations. The existence of such "quasispecies" explains the rapid adaptation of these viruses to environmental changes. Some adapt to invertebrates and others to vertebrates, and they confound the immune systems of their hosts. Pathogenic variants can easily arise.

There is another source of heterogeneity, too. A characteristic common to arenaviruses and bunyaviruses is that they have segmented genomes. (The bunyaviruses have three segments of RNA, arenaviruses two.) When a cell is infected by two viruses of the same general class, they can then recombine so that segments from one become linked to segments from the other, giving rise to new viral types called reassortants.

Although we have a basic appreciation of the composition of these entities, we have only a poor understanding of how they cause disease. Far beyond the limited means of investigation in local tropical hospitals, many of these viruses are so hazardous they cannot be handled except in laboratories that conform to very strict safety requirements. There are only a few such facilities in the world, and not all of them have the required equipment. Although it is relatively straightforward to handle the agents safely in culture flasks, it is far more dangerous to handle infected monkeys: researchers risk infection from being scratched or bitten by sick animals. Yet the viruses cannot be studied in more common laboratory animals such as rats, because these creatures do not become ill when infected.

We do know that hemorrhagic fever viruses have characteristic effects on the body. They cause a diminution in the number of platelets, the principal cells of the blood-clotting system. But this diminution, called thrombocytopenia, is not sufficient to explain the hemorrhagic symptoms. Some hemorrhagic fever viruses destroy infected cells directly; others perturb the immune system and affect cells' functioning.

Among the first group, the cytolytic viruses, are the bunyaviruses that cause a disease called Crimean-Congo fever and Rift Valley fever; the filoviruses Marburg and Ebola; and the prototype of hemorrhagic fever viruses, the flavivirus Amaril. Their period of incubation is generally short, often less than a week. Serious cases are the result of an attack on several organs, notably the liver. When a large proportion of liver cells are destroyed, the body cannot produce enough coagulation factors, which partly explains the hemorrhagic symptoms. The viruses also modify the inner surfaces of blood vessels in such a way that platelets stick to them. This clotting inside vessels consumes additional coagulation factors. Moreover, the cells lining the vessels are forced apart, which can lead to the escape of plasma or to uncontrolled bleeding, causing edema, an accumulation of fluid in the tissue, or severely lowered blood pressure.

The arenaviruses fall into the noncytolytic group. Their period of incubation is longer, and although they invade most of the tissues in the body, they do not usually cause gross lesions. Rather the viruses inhibit the immune system, which delays the production of antibodies until perhaps a month after the first clinical signs of infection. Arenaviruses

Ebola's Unanswered Questions

by Laurie Garrett

Last spring in Kikwit, Zaire, Ebola proved once again that despite the agonizing and usually fatal illness it provokes, the microbe cannot in its present incarnation spread far—unless humans help it to do so. The virus is too swiftly lethal to propagate by itself. In the early waves of an epidemic, it kills more than 92 percent of those it infects, usually within a couple of weeks. Such rapidity affords the microbe little opportunity to spread unaided, given the severity of the illness that it causes.

In each of the four known Ebola epidemics during the past 19 years, people have helped launch the virus from its obscure rain forest or savanna host into human populations. In 1976 in Yambuku, an area of villages in Zaire's northern rain forest, the virus's appearance was multiplied dozens of times over by Belgian nuns at a missionary clinic who repeatedly used unsterilized syringes in some 300 patients every day. One day someone arrived suffering from the then unknown Ebola fever and was treated with injections for malaria. The syringes efficiently amplified the viral threat.

In both 1976 and 1979, humans helped the virus spread wildly in N'zara and Maridi, in the Sudan's remote southern grasslands. Improper hospital hygiene again played a key role, and local burial practices, which required the manual removal of viscera from cadavers, compounded the disaster.

Medical and funeral settings were likewise crucial in Kikwit earlier this year. Infections spread via bodily fluids among those who tended the dying and washed and dressed the cadavers. The major amplification event that seems to have started the epidemic, early in the new year, was an open casket funeral. The deceased, Gaspard Menga, probably acquired his infection gathering firewood in a nearby rain forest. The virus spread rapidly to 13 members of the Menga family who had cared for the ailing man or touched his body in farewell, a common practice in the region, or cared for those who got Ebola from Menga.

A second amplification event occurred in March inside Kikwit General Hospital. Overrun by cases of incurable bloody diarrhea, hospital officials thought they were facing a new strain of bacteria. The doctors ordered a laboratory technician to draw blood samples from patients and analyze them for drug resistance.

When he took ill, the hospital staff thought that his enormously distended stomach and high fever were the results of typhus infection and performed surgery to stave off damage. The first procedure was an appendectomy. The second was a horror. When the physicians and nurses opened the technician's abdomen again for what they expected to be repair work, they were immediately drenched in blood. Their colleague died on the operating table from uncontrolled bleeding. The contaminated surgical team became the second wave of the epidemic.

The virus's reliance on unintended help from humans forces attention to the common thread that runs through the known Ebola epidemics: poverty. All the outbreaks have been associated with abysmal medical facilities in which poorly paid (or, in the case of Kikwit, unpaid) medical personnel had to make do with a handful of syringes, minimal surgical equipment and intermittent or nonexistent running water and electricity.

It seems quite possible that Ebola (and other hemorrhagic fever viruses) might successfully exploit similar conditions occurring anywhere in the world. As air transportation be-

(continued on page 64)

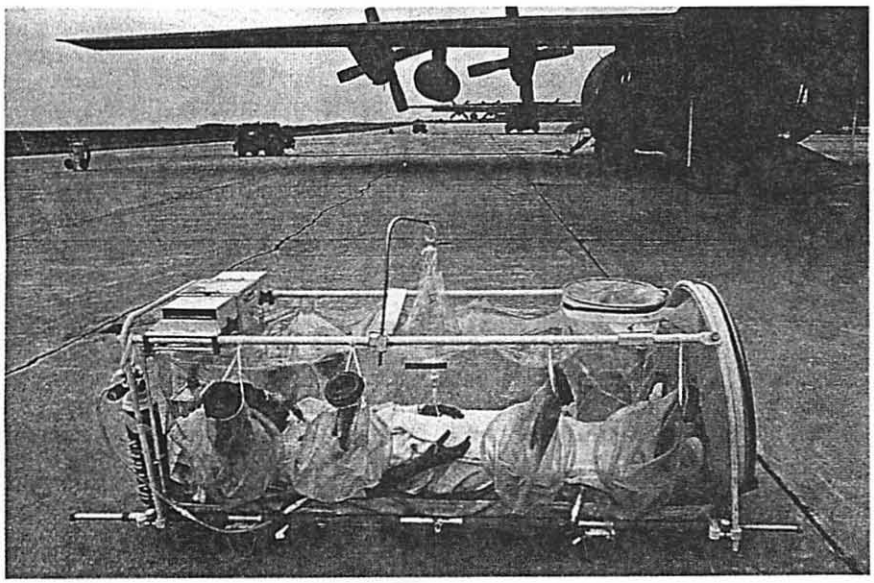
suppress the number of platelets only slightly, but they do inactivate them. Neurological complications are common.

Hantaviruses are like arenaviruses in that they do not destroy cells directly and also have a long period of incubation, from 12 to 21 days. They target cells lining capillary walls. Hantaan and Puumala viruses invade the cells of the capillary walls in the kidney, which results in edema and an inflammatory reaction caused by the organ's failure to work properly. Sin Nombre, in contrast, invades pulmonary capillaries and causes death by a different means: it leads to acute edema of the lung.

Prospects for Control

Several research groups are trying to establish international surveillance networks that will track all emerging infectious agents. The World Health Organization has established a network for tracking hemorrhagic fever viruses and other insect-borne viruses that is particularly vigilant.

Once a virus is detected, technology holds some promise for combating it. An antiviral medication, ribavirin, proved effective during an epidemic of hantavirus in China. A huge effort is under way in Argentina to develop a vaccine to protect people against Junín.



KAREN KASMAUSKI *Matrix*

PORTABLE ISOLATOR UNITS equipped with air filters have been maintained by the U.S. Army since 1980 for evacuating personnel carrying suspected dangerous pathogens. The equipment would be used to bring patients needing specialized care to an isolation facility at Fort Detrick, Md., but has never been called on for this mission.

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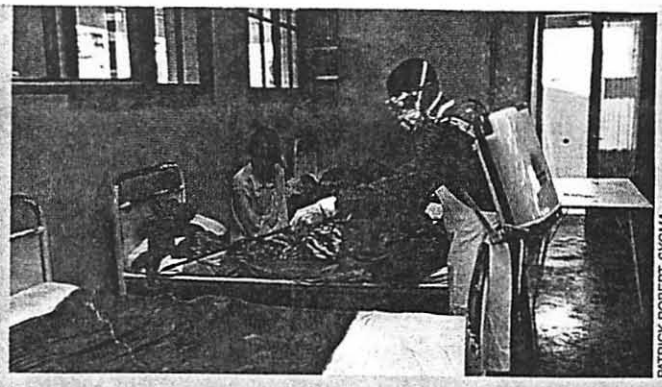
comes more readily available and affordable, viruses can be more easily moved around the planet. The rapid deterioration in public health and medical facilities in the former Soviet Union and other regions should therefore be cause for concern.

The exact nature of the risk, of course, depends on the Ebola virus's biology, much of which remains mysterious. Throughout the summer, researchers from the University of Kinshasa, the U.S. Centers for Disease Control and Prevention, the Pasteur Institute in Paris, the National Institute of Virology in Johannesburg and the World Health Organization combed Kikwit for answers to questions that have puzzled scientists since the first Yambuku epidemic: What are the precise constraints on Ebola's transmission? And where does it hide between epidemics?

The two Sudanese epidemics started among cotton factory workers. At the time scientists scoured the N'zara complex for infected insects or bats, but although the animals were plentiful, none carried the virus. In Yambuku, suspicions fell on a range of rain-forest animals, including monkeys. Again, however, no trapped animals tested positive for infection. Surveys conducted during the late 1970s in conjunction with a WHO effort to control monkeypox found no infected primates or large animals in central Africa.

The rain forest frequented by Gaspard Menga contained abundant rats, bats, mice and snakes. Trapping efforts in the region may eventually reveal Ebola's hideout. For the present, though, the virus's reservoir remains unknown. Also unknown is whether shared drinking water, foods and washing facilities can transmit infection.

Because all outbreaks to date have involved transmission by fluids, control has consisted of fairly straightforward, low-cost efforts. Patients were isolated, and the citizenry instructed to turn over their unwashed dead to authorities.



PATRICK ROBERT SYGMA

MASKED AND GLOVED health worker disinfects a bed used by a patient stricken by the Ebola virus in Kikwit, Zaire.

Once residents appreciated the links between tending the sick, washing a cadaver and dying of Ebola, epidemics quickly ground to a halt.

One way that Ebola could escape such controls would be through a major mutational event that made it more easily transmissible. Were Ebola, or any hemorrhagic fever virus, to acquire genetic characteristics suitable for airborne transmission, an outbreak of disease anywhere would pose a threat to all humanity.

As far as is known, nobody has ever acquired the microbe from inhaled droplets coughed into the air (although it can certainly be passed in saliva during a kiss). There are usually many genetic differences between fluid-borne microbes and airborne ones, so it seems unlikely that the jump could be made easily. But the question has never been specifically studied in the case of Ebola, because research on microbes that are found primarily in developing countries has for many years been poorly funded.

LAURIE GARRETT is a reporter for Newsday *and the author of* The Coming Plague: Newly Emerging Diseases in a World Out of Balance *(Penguin USA, 1995).*

Indeed, vaccines against the Rift Valley fever in animals, and against yellow fever in humans, are already approved for use. Yet despite the existence of yellow fever vaccine, that disease is now raging in Africa, where few are vaccinated.

Other approaches are constrained because it is difficult or impossible to control animals that are natural reservoirs and vectors for the viruses or to predict ecological modifications that favor outbreaks of disease. There was an effective campaign against rodent vectors during the Lassa and Machupo arenavirus outbreaks, but it is not usually possible to sustain such programs in rural regions for long periods.

Precautions can be taken in laboratories and hospitals, which have ironically served as amplifiers in several epidemics. In the laboratory, viruses responsible for hemorrhagic fevers must be handled in maximum confinement conditions (known in the jargon as biosafety level 4). The laboratory must be kept at lowered pressure, so that no potentially infectious particle can escape; the viruses themselves should be confined in sealed systems at still lower pressure. In hospitals, the risk of infection from a patient is high for some viruses, so strict safety measures must be followed: hospital personnel must wear masks, gloves and protective clothing; wastes must be decontaminated. A room with lowered pressure is an additional precaution.

Since penicillin has been in widespread use, many people had started to believe that epidemics were no longer a threat. The global pandemic of HIV, the virus that causes AIDS, has shown that view to be complacent. Hemorrhagic fever viruses are indeed a cause for worry, and the avenues to reduce their toll are still limited.

The Author

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Further Reading

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THIS MONTH: CLOSE ENCOUNTER WITH A NEW COMET

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ARTICLE

THE KILLING FIELDS: LATTER-DAY PLAGUES

AIDS may be just the beginning. Experts fear a rash of worldwide epidemics

BY KATHLEEN McAULIFFE

"An entire hospital has been wiped out and we still don't know what's behind the outbreak. Can you find out for us?"

It was not the sort of phone call Karl Johnson relishes, but as a world-renowned expert on tropical infections at the U.S. Centers for Disease Control (CDC), he stoically bid farewell to his wife and boarded the next plane for Central Africa. There, near the fetid banks of the Ebola River in northwestern Zaire, a horrible fever had sprung seemingly from nowhere. The year was 1976, and as Johnson arrived with an international team of investigators, fleeing villagers were being turned back at gunpoint by government authorities ordered to quarantine the entire province. None of the community would go near the bush hospital where the outbreak began. So the party of foreigners—with only surgical gowns, gloves, and face masks for protection—set off in jeeps to visit the sick in scattered villages.

"For two to three weeks, we really held our breath," says Johnson, who now works at National Biosys-

PAINTINGS BY GEORGE TOOKER

tems, Inc., in Rockville, Maryland. "We saw very rapidly that the disease had an eighty to ninety percent fatality and we had no idea how it was being transmitted." Compounding their fears, members of the team—all of whom had volunteered for the mission—kept getting splattered with blood while collecting medical samples. Meanwhile the villagers were unwittingly inviting death by participating in funerary rites that involved intimate contact with the deceased.

To Johnson's relief, the tribal chiefs awoke to the gravity of the threat, banned this ritual, and reinstituted stringent disease control practices used since antiquity in Africa to thwart the ravages of smallpox. The infected were isolated in a hut, where their only contact with the outside world was through food and water slipped under the door. "If they walked out," says Johnson, "fine. If not, the hut was set on fire."

Several hundred deaths later, the disease vanished as mysteriously as it had appeared. The researchers eventually determined that it was a blood-borne viral infection—unprecedented in medical history—precipitated at the hospital by the use of a few unsterilized syringes to administer hundreds of injections, and possibly spread by sex with infected individuals.

Such an outbreak would be unthinkable at a modern, well-equipped hospital in the United States or Europe. But that hasn't stopped Johnson from pondering alternative outcomes—frightening "what if" scenarios—that haunt him like a recurrent nightmare more than a decade later. "What would we have done if the virus was spread by cough droplets in the air?" he asks. "If that were the case, there's no doubt in my mind that Ebola fever could have qualified as the Andromeda strain and we'd all have died."

Such a disaster may not be as remote as commonly thought. As Nobel laureate Joshua Lederberg of Rockefeller University warns in a leading medical journal: "Most people today are grossly overoptimistic with respect to the means we have available to forfend global epidemics comparable with the Black Death of the fourteenth century or on a lesser scale the influenza of 1918, which took a toll of millions of lives."

In a span of less than 15 years, the United States alone has been stricken by a wave of new infections. Toxic shock syndrome, Legionnaires' disease, AIDS, Lyme disease—all have emerged from obscurity to become household names. And while no one has a crystal ball, infectious disease experts fear an escalation of new—and possibly more deadly—epidemics in the future. Indeed, some candidates already loom on distant horizons—isolated in tiny geographical pockets. Others—notably an insect-borne infection that can be fatal to children—are rapidly encroaching on American territory.

Why the explosion of pestilence? If these were the Dark Ages rather than the Space Age, we would undoubtedly wonder if God was punishing us for our sins. To be sure, changing mores associated with sex and drug use have contributed to the spread of highly lethal diseases—AIDS being a leading example. But many other global forces have also conspired against us.

With air travel becoming increasingly affordable, infectious agents that would have been confined to one corner of the world just a few decades ago are now jumping across continents at near-supersonic speeds. This places modern man at a historical crossroads—much like New World inhabitants at the dawn of the Age of Discovery—that makes us uniquely vulnerable to novel plagues. As William McNeill, a retired professor of history at the University of Chicago and author of *Plagues and Peoples*, points out, "Cortés did not conquer the Aztecs—it was the smallpox that the Spaniards brought with them." Whenever an unexposed population comes into contact with a new, lethal infectious agent, the death toll is notoriously high, leaving only a tiny subgroup of survivors composed largely of resistant individuals. Just 130 years after the conquistadores arrived in central Mexico, up to 90 percent of the indigenous population had perished from smallpox and other European diseases—a frightening reminder of the threat posed by today's globe-trotting microbes.

IT'S BIZARRE: A DISEASE NEVER SEEN BEFORE STRIKES WITH THE SAME VENGEANCE, IN THE SAME SEASON, 600 MILES APART.

Our encroachment on rain forests and other wilderness areas is also bringing humans into direct contact for the first time with potentially dangerous pathogens harbored by wild animals. In the U.S. Northeast, for example, new housing developments infringing on natural deer habitats have been implicated in the explosion of Lyme disease—a debilitating arthritic condition transmitted by a deer-borne tick.

Just as ominous, a favorite breeding ground of microorganisms—the steamy tropics—now sustains the densest human population ever. Of particular concern is the unprecedented emer-

gence of "mega-cities" of 5 million or more people in developing parts of the tropics, where malnutrition and poor sanitation give microbes a further edge. Says McNeill, "Swelling numbers at the top of the food chain constitute a magnificent feeding ground for microbes."

Adding to this peril, the United States and other nations are withdrawing support for infectious disease surveillance around the world. Such biological "listening posts" are critical for detecting epidemics early on—especially in primitive cultures at high risk for emerging plagues.

Before beating a fast retreat to civilization, however, be forewarned that technology is no safeguard against the rising tide of infection—and may even increase our chances of succumbing. Ironically, inventions designed to ease our lives can sometimes backfire, providing microbes with more lethal routes of entry into the body. Consider *Staphylococcus epidermidis*, a bacterium long viewed as harmless, which is now a leading cause of infection in intensive care units. Tough new strains that don't respond to antibiotics routinely find their way into patients' veins through intravenous tubes and sometimes even colonize artificial-heart valves. "It's a terrible problem and can be fatal for patients who are very ill to begin with," says Claire Broome, chief of the CDC's special pathogens branch.

The bacterium that killed 29 people who attended a 1976 American Legion convention in Philadelphia may also have gained a technological boost. Recent outbreaks of the disease have been traced to faulty ventilating systems, which permit the pathogen to proliferate. This is not to imply—public perceptions to the contrary—that the bacterium sprang out of thin air. After isolating the culprit at the convention, the CDC found similar samples in its vaults that came from people presumed to have died from pneumonia. "In the past," says Broome, "Legionnaires' probably occurred and was mistaken for viral pneumonia."

Likewise, toxic shock syndrome owes its notoriety to a more absorbent tampon introduced by Rely in 1980. Before then, according to CDC epidemiologist Benjamin Schwartz, the bacterial infection probably struck infrequently—and hence was likely mistaken for scarlet fever, which has similar symptoms.

As AIDS and Ebola fever clearly demonstrate, however, some diseases are true originals, emerging unheralded in man. As such, they pose a formidable challenge to medical science—and tracing their evolution involves detective work of the highest order. Although there are often more clues than answers, high on the suspect list are pathogens that appear to have jumped from an animal to a human host.

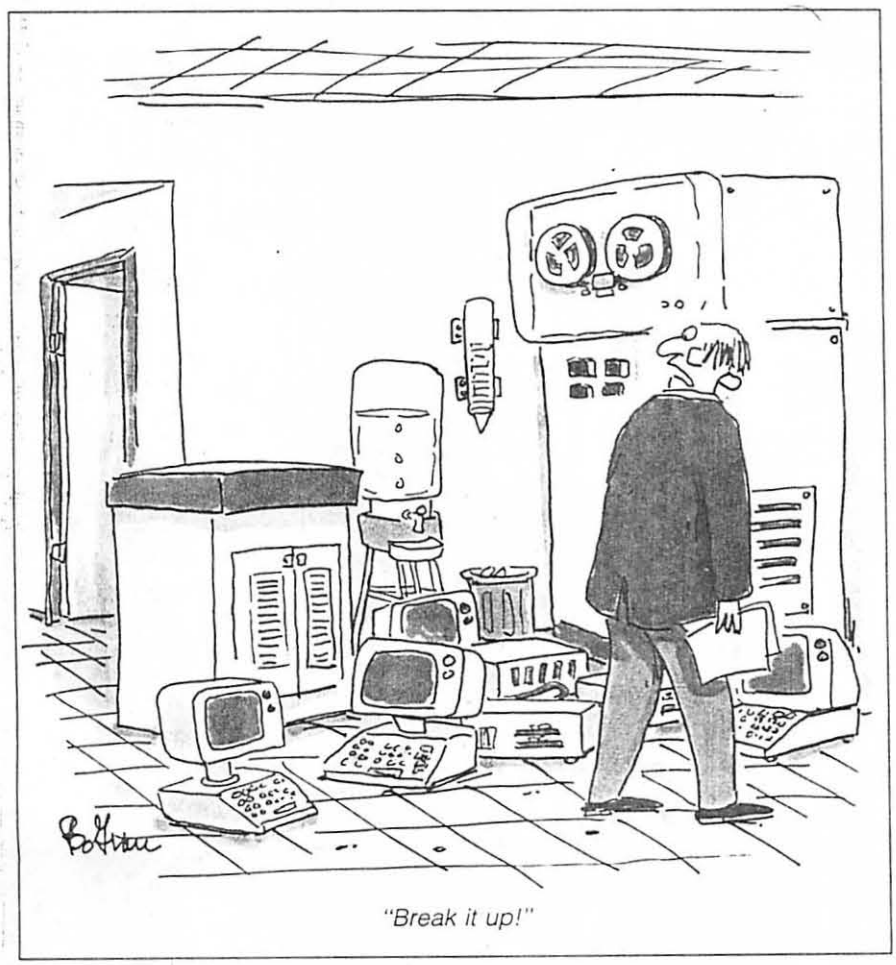
An intriguing example from the past: The oldest documented case of syphilis has been tentatively traced to a bear that lived 11,500 years ago in what is now Indiana. That is more than 5,000 years before there is clear-cut historical evidence of the first outbreak in man. The bear's bones, uncovered in 1987, had holes and other signs of the venereal disease—a possibility further supported by an antibody test. Although more studies of both human and animal remains could alter the picture, researchers now speculate that a bear bite, or contact with a contaminated carcass, could have unleashed the scourge of syphilis upon mankind.

In a similar vein, a primate is now suspected of having played a key role in the eruption of AIDS. The evidence: Large numbers of seemingly healthy African green monkeys living in the wild carry antibodies against a closely related virus. "Possibly facilitating viral transmission from animal to man," says Gerald Myers of Los Alamos National Laboratory in New Mexico, "there was a sharp rise in the exportation of monkeys from Africa into the United States in the Sixties for medical research."

The origin of Ebola fever is more perplexing. While Johnson struggled to stamp out the epidemic in Zaire, an identical disease broke out in another rural hospital in the southern Sudan—600 miles away. Initially, it was assumed that the same virus had caused both epidemics. But to everyone's shock, laboratory analysis later revealed that two distinct—though related—strains of viruses were involved. "It's a bizarre biological coincidence," says Johnson. "A disease never before encountered in recorded time strikes with the same vengeance, in the same season, six hundred miles apart. It almost makes you think that some environmental factors were just right for this family of viruses to explode on the scene."

The plot thickens. Last January a Swedish tourist returning from a vacation in Kenya mysteriously contracted another related virus—and, after hovering at the edge of death for two weeks, appears (as of this writing) to be recovering in the intensive care unit of a hospital south of Stockholm. Only a month earlier, Ebola fever made an even odder appearance, this time striking monkeys imported into the United States from the Philippines. This makes Johnson wonder whether a primate is a key link in the propagation of the disease in Africa. But, as he cautions, an exhaustive search for infected animals in the vicinity of the stricken villages turned up negative. "Frankly, we're scratching our heads about this," he says. "Ebola fever is as lethal to monkeys as it is to man, and you'd

CONTINUED ON PAGE 94



"Break it up!"

KILLING FIELDS

CONTINUED FROM PAGE 54

normally expect the animal that harbors this virus in nature to be resistant to it."

The evolution of a new disease agent cannot always be traced to a microbe expanding its range of target hosts, however. Sometimes the precipitating event may be an internal transformation—a genetic mutation that turns a once-benign microorganism into a powerful foe. It appears to be just such a fluke that brought tragedy to a small Brazilian town in 1984. Ten children were rushed to an emergency ward after developing a high temperature and huge purple blotches on their skin—a syndrome that came to be known as Brazilian purpuric fever. As doctors puzzled over how to treat their strange symptoms, all of the youngsters perished. A second outbreak in 1986, involving 14 more children, eventually yielded an important clue: Many of the victims of Brazilian purpuric fever had earlier suffered from conjunctivitis, a bacterial infection that causes symptoms no more serious than runny eyes. But in this Brazilian town, says CDC epidemiologist Bradley Perkins, it looks as though the bacterium "underwent a genetic change that made it more virulent."

Viruses are a still more prolific source of new mutants in nature. That, coupled with the fact that they are impervious to antibiotics, makes them a daunting threat to public health. We have slain the tiger and speared the mighty whale, but we are still at the mercy of the world's smallest creatures. As Rockefeller's Lederberg proclaimed at a conference last year in Washington, DC, on emerging viruses, they are "our only real competitors for dominion of the planet.... We shall have to be very nimble indeed to keep up with them."

Unlike bacteria and the cells of higher organisms, many viruses lack "proofreading" mechanisms for correcting genetic errors during replication. Since their sheer numbers are staggering—many billions of times the entire human population could easily fit inside a test tube with a good source of bacteria for food—genetic copying mistakes are commonplace. According to recent estimates by John J. Holland, a virologist at the University of California at San Diego, viral mutations occur in about 1 in 10,000 replications—a figure much higher than previously suspected and a full six orders of magnitude greater than occurs in human cells.

To be sure, most of these mutations are deleterious—and even when the organism is rendered more potent, it must still contend with immune cells in the body. But there is always the risk that a rare mutant will be able to crush the host's defense system. Such gangbusters typically go on a killing spree until they have virtually exhausted their food supply.

That is exactly what happened in a Pennsylvania chicken farm in 1983. A mild avian flu that normally infects the chickens' lungs suddenly turned killer and attacked their brains. Every chicken died, and the virus spread to three states before it was finally brought under control. To do so, the U.S. Department of Agriculture spent $70 million and eradicated 17 million chickens, burying them in a mass grave. "The virus nearly wiped chickens off U.S. menus," says Robert Webster, a virologist at St. Jude's Children's Research Hospital in Memphis. "It was that serious."

Remarkably, a single point mutation wrought all this mayhem—and there's no ruling out a repeat episode. A close relative of the mutant germ is now broadly disseminated in the wild duck population, where it coexists in their guts and is excreted into the water. "Several hundred million chickens are just waiting to be infected," says Webster. And he's not just worried about a cheap source of protein evaporating. The situation of the chickens, he warns, "is very similar to humans who live in urban environments. What if this occurred to us? We can't dig trenches and bury everyone [suspected of being infected]."

Actually, an evolving strain of the influenza virus may be the least of our worries. What might happen, for example, if the already lethal AIDS virus were to undergo further genetic change? Could this formidable opponent become a more efficient multiplier, one day enabling it to populate the bloodstream in sufficient quantities for an insect to transmit it? Or could the virus grow more readily in the skin, intestines, lungs, or mouth—paving the way for transmission by casual contact, ingestion, or inhalation? At the Washington conference on emerging viruses, even Nobel laureates clashed in their assessment of these risks.

One of them, Howard M. Temin of the University of Wisconsin, remained skeptical. Sure, he acknowledges, a mutation could permit the virus to grow more efficiently in other tissues—such as the respiratory tract. But if that were the case, he argues, the pathogen would have to alter its external coat so much that it would lose the capacity to infect immune cells. "So it would no longer cause AIDS," says Temin. "It might be just another cold virus."

Lederberg, on the other hand, was not so sanguine. "There will be surprises," he says, "because our fertile imagination does not begin to match all the tricks that nature can play."

Doctors tracking the spread of a novel human disease agent called a viroid

Even so, the American Red Cross isn't taking any chances with the nation's blood supply. In December 1988 it began screening for the two viruses. "With so many uncertainties surrounding their health effects," says CDC epidemiologist Rima Khabbaz, "it's hard to know how to counsel infected people who are sexually active or who want to have children."

The behavior of rodents rather than humans is primarily responsible for yet another viral plague that has quietly infiltrated this country. Called Seoul virus, it causes an acute disease of the kidneys in Asia. The pathogen is believed to have been brought to our shores by adventurous vermin that climbed aboard ships carrying goods from South Korea and is now harbored by rats and mice in Philadelphia, Houston, New Orleans, and Baltimore. The rodents themselves are unaffected by the virus, but humans who inhale dust contaminated by their urine or feces may not fare as well.

At Johns Hopkins Hospital in Baltimore, tests showed that 15 out of 1,148 patients with acute kidney failure had been infected by the Seoul virus. In addition to chronic renal problems, they often suffered from hypertension and strokes. None had traveled outside the United States, ruling out the possibility of exposure to the virus overseas.

"This is a very suggestive finding," says James Childs of the Johns Hopkins University School of Hygiene and Public Health. As he points out, hypertension and strokes are much more common among inner-city residents—particularly blacks—than among rural inhabitants of the same race. He suspects that rat infestations in urban settings could be one reason why. "I don't mean to imply that the Seoul virus is the predominant or only cause of hypertension in inner-city blacks," says Childs, "but it's an intriguing link that we certainly want to explore further." If the association holds up, he predicts city health departments will make the eradication of rats a top priority.

Or one would hope. So far the nation's response to the threat of dengue hemorrhagic fever does not inspire much confidence. A viral infection transmitted by insects, dengue has been around in a mild form for centuries in Asia, causing flulike symptoms and aching joints in adults. In the Fifties, however, the virus suddenly became much more virulent—especially in children. Young victims typically break out in a rash and begin bleeding from the nose and ears. Many of them then go into shock and die. More than 600,000 cases of this severe type of dengue were reported in Southeast Asia in 1987, compared with 2,060 in 1967—a 300-fold increase in 20 years.

As if that were not bad enough, one type of mosquito that transmits the disease has been entering America since the early Eighties aboard tires imported from Japan for retreading. The insect's eggs, explains entomologist Bruce Eldridge of the University of California at Davis, hatch in water that collects inside the tires when it rains. This highly successful invader is known as the Asian tiger mosquito and is now found in Texas, Missouri, and everywhere east of the Mississippi. So far it does not appear to be transmitting the deadly dengue virus—at least not within the continental USA. But there are plenty of mosquito carriers throughout the tropics—including Puerto Rico and Mexico, where the hemorrhagic fever attacked more than 30,000 people in 1986. "The disease is literally knocking at our back door," warns Eldridge.

The Asian tiger mosquito is an extremely aggressive biter, and it is difficult to eradicate. On the outskirts of almost every major metropolitan area are tire dumps that stretch for acres. Since pesticides can't penetrate to the inside of the tires, they are of little use in controlling the insect. The obvious solution is to conduct a massive cleanup—but

would undoubtedly share Lederberg's awe at nature's inventiveness. Smaller than any known virus, this odd entity lacks a protein coat, being little more than a collection of free-roaming genetic material. Viroids have been implicated in many plant diseases, but they are exceedingly rare in the animal kingdom. Or so everyone thought until an Italian researcher in the late Seventies discovered a viroidlike particle in man. Referred to as the delta viroid, or agent, it is the ultimate parasite: To replicate, it requires not only a human cell but one infected by hepatitis B virus (whose outer coat provides protection for the viroid). If these two conditions are satisfied, the viroid can cause a far more devastating form of liver disease than hepatitis B alone. Called delta hepatitis, it now kills about 850 Americans each year. The majority are IV drug addicts and their intimate partners because the delta agent, like hepatitis B, is spread by needle sharing and sex.

The good news is that all these deaths can now be prevented with the newly developed hepatitis B vaccine. The bad news is that the vaccine comes too late for much of the developing world, where hepatitis B has already reached epidemic proportions. Explains Stephen Hadler of the CDC's immunology branch, "Hepatitis B can be transmitted through festering skin wounds. So wherever people sleep several to a bed—as happens in poor communities around the globe—the virus is commonplace." More than 200 million people worldwide are estimated to be chronic carriers of the hepatitis B virus—and thus under grave threat from the delta agent. Indeed, delta hepatitis has recently caused devastating outbreaks in South America and is beginning to make inroads into Asia's vast population. "Over there it could do major damage," says Hadler, "but here it's likely to remain mostly in IV drug users."

Also insidiously spreading among IV drug users in the United States is a virus that was once largely confined to southwestern Japan and the Caribbean. Called HTLVI, it causes a rare, highly lethal type of lymphoma and, less frequently, a degenerative nerve disease that resembles multiple sclerosis. Adding to concerns, it has a mysterious cousin, HTLVII, which is also spreading among addicts—but so far without causing any illness. Although needle sharing is the most common route of propagation for these viruses, they can be transmitted by sex, blood transfusions, and from mother to baby through breast milk. Depending upon the part of the United States, 2 to 40 percent of IV drug users carry HTLVI or HTLVII. Long-term studies in Japan, however, indicate that less than 5 percent of infected individuals will actually develop disease symptoms.

it is also a costly solution, and so it has been abandoned. "I'm deeply concerned about this," says Eldridge. "If we don't commit the resources today, it could harm us in the long run."

Despite abundant evidence that we live in times most favorable to microbes that prey on man, the invention of antibiotics and several vaccines since World War II has lulled many people into a false sense of security. As the Institute of Medicine recently documented, there is now a tremendous shortage of specialists in infectious disease control both in the United States and in developing countries. Owing to budgetary pressures, the National Institutes of Health was forced to close the last of its laboratories for tropical virology in 1973. More recently, an important tropical medicine laboratory in Hawaii shut down, and the U.S. military has scrapped a key surveillance unit for new diseases in Kuala Lumpur, Malaysia. As for the World Health Organization, it has only a handful of people manning its viral disease unit at its headquarters in Geneva and a single regional office for all of Africa. Warns Donald A. Henderson, dean of the Johns Hopkins University School of Hygiene and Public Health, "We are not well structured or staffed on a global level to detect and investigate new diseases."

Like many of his colleagues at the DC conference, Henderson pleaded for greater funding of infectious disease control programs, especially in tropical regions where microbes thrive. For $150 million a year, he estimates, a global consortium could finance 15 tropical medical centers and ten U.S. research facilities, with a remaining $25 million available for special projects.

Given the current budget crunch, however, scientists are skeptical that politicians will be persuaded to cough up even that modest sum. In all likelihood, the early warning detection system will be postponed—until another disaster on the scale of AIDS jolts us into action.

Although dengue is an obvious contender for the title of microbial menace number one, scientists are quick to point out that the threat could come from almost anywhere. Brazilian purpuric fever, for example, has so far accounted for only a few sporadic cases in small, rural towns. But as the CDC's Perkins observes, "If the disease gets to São Paulo, with its population of fourteen million, it could be catastrophic." Even with intensive antibiotic treatment, he reports, the bacterial infection claims the lives of half of its victims.

In this era of fiscal shortsightedness, it is well to recall that germs have far-flung reaches. As Nobel laureate Lederberg stresses, "The microbe that felled one child on a distant continent can reach yours today and seed a global pandemic tomorrow." ⊠

COMMUNICATIONS

CONTINUED FROM PAGE 25

speaking countries, and your edition will certainly help to fill in the gap. That's why I'm sure *Omni* magazine will be extremely popular with the Soviet readers.

Wishing you a success on the Soviet market, I want to warn you of some difficulties that you may encounter. One of them is the correct use of the Russian language, which presents lots of traps for foreign learners.

Even those who think that they have mastered Russian to the full extent can find themselves in such a trap. And that's exactly what happened with you (or rather with your linguistic staff) on the very front page of the first issue, circulated in the Soviet Union in September 1989. Your cover persuades:

Выиграйте американские роскошные продукты на сто тысяч долларов.

This sounds alien to the Russian ear, because it contains three incorrect usages of modern Russian. Two of the mistakes can be regarded as not very bad, as they do not hinder understanding (only show that the text was made by a foreigner), but one is rather serious, because it affects sense. I mean the use of the word «продукты». Perhaps your translators were misled by the meaning of the English word *products*, but in Russian, «продукты» (plural of «продукт») has only one meaning, and that is "something one can eat": foodstuffs, food products. That is why the first natural reaction which the title page causes in the Soviet reader is: How on Earth can one eat up so many foodstuffs? The word you should have used instead of «продукты» is certainly «товары» (goods).

I'm not going to tire you with lengthy linguistic analysis because that's not what I'm aiming at. I can only offer you a better Russian variant, which won't sound alien to the Russian readers:

Вы можете выиграть роскошные американские товары на сто тысяч долларов.

While making these comments, I by no means intended to blame your linguistic staff for their inadequate knowledge of Russian. Being a linguist myself and having a special interest in the comparative study of English–Russian word combinations, I know perfectly well how difficult it is to create a flawless text in a foreign language. Please regard this letter as a friendly sign of appreciation of your and Kathy Keeton's attempts to make our life better and a sincere wish of success to *Omni* in the future in the Soviet Union.

It's always better to do things with a little help from one's friends, isn't it? And you've got them in the Soviet Union.

With best regards,
Tatiana Dobrosklonskaya
Doctor of Philology
Moscow State University
Moscow, USSR

For Good Measure

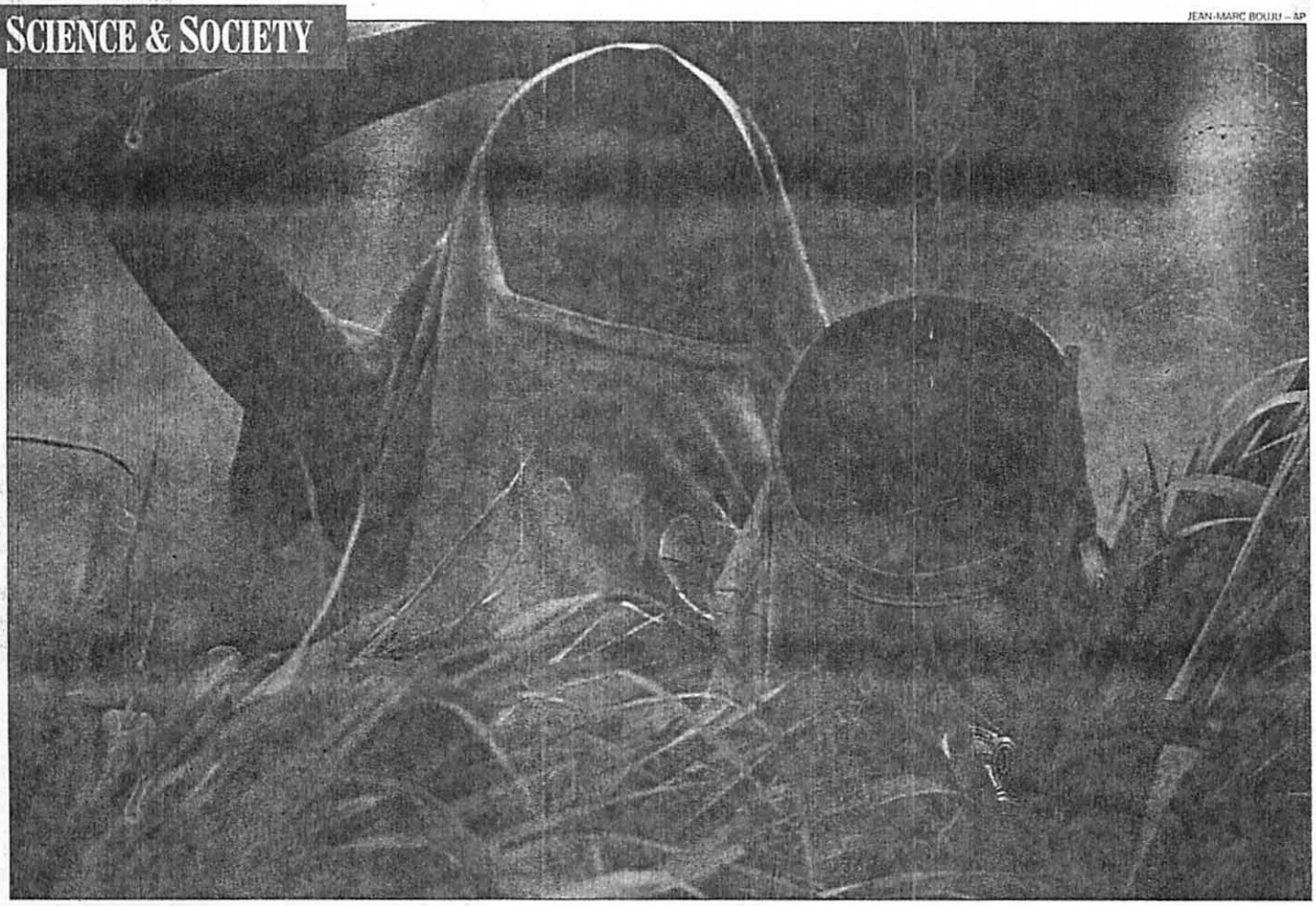
I was quite surprised to find an apparent lack of sense of speed and distance in "Adventure Capital" [February 1990]. You used the term *knots per hour* twice. A knot is a speed of nautical miles per hour. We are also informed that 100 meters is "less than a mile." While true, this is misleading, since 100 meters is less than one tenth of a mile. Someone needs to tighten ship.

John E. Runninger
Rome, NY

Forgive us, we knew knot what we did.
—The editors ⊠

Soviet philologist Tatiana Dobrosklonskaya, with children Alexis and Kathy, reading Omni.

JEAN-MARC BOUJU – AP



WHEN FEAR STRIKES. *Children wait outside the Kikwit hospital where the Ebola virus (below) struck and the infected were isolated.*

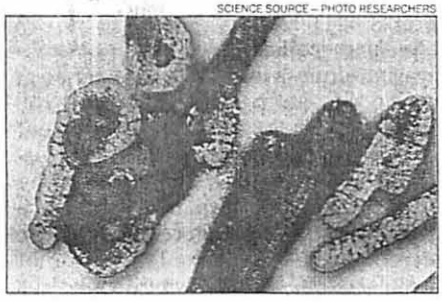
Horror in the hot zone

A killer virus in Zaire reminds the human species of its vulnerability

The usually bustling, noisy hospital in the town of Kikwit, Zaire, was eerily quiet last week. The breezeway down the center of the one-story cinder-block buildings was banked by nearly empty wards, its 350 beds vacated by the news that the deadly Ebola virus had broken out in the hospital. Only 20 patients remained, cared for by one doctor, three nurses and a few laboratory workers. At times outside the hospital, armed Zairian soldiers stood guard, letting neither patients out nor their families in. At other times, no enforcement was in evidence. The families were anxious to bring their sick relatives food, because it is not provided there. "Patients are afraid they may starve to death," explains Gerda Bossier, a spokeswoman for Médecins Sans Frontières, a medical relief agency.

In fact, though, an even more gruesome kind of death stalks them. All those left in the hospital are infected with Ebola, the terrifying virus first discovered in 1976. Their chances of survival, even if they were at the best of hospitals, are slim. About 90 percent of those who contract the virus die horribly. First fever and malaise set in; then comes the vomiting and rash about three to 16 days after infection. The patient begins to hemorrhage internally, leaking blood from the mouth, eyes, ears and other orifices. Within a week or so, internal organs, now turned to bloody mush, shut down and the patient is dead.

SCIENCE SOURCE – PHOTO RESEARCHERS



At the weekend, at least 48 people were dead and an additional 55 were known to be infected with the virus. Relief workers have estimated there may be as many as 100 cases by now, but they won't know for sure until blood samples are sent to France and America for diagnosis. Spread by fleeing pa-

tients, the virus also might have reached the towns of Vanga, Mosango, Yassa Bonga, Bokangalonzo and Kenge. Officials from the World Health Organization were on a desperate hunt in the slums of the capital, Kinshasa, for a nurse named Marceline Mbuka Pindo, who left Kikwit with Ebola-type symptoms, was treated briefly in a Kinshasa clinic and is thought to be seeking her family in the Ngaba ghetto. If she gets sicker in that dense population, it could be a disaster.

ROBERT KEMP – USN&WR

Zaire: Portrait of dystopia

The nation's economy has collapsed since 1991, when unpaid Army members looted much of the country.

Map key: Recent Ebola outbreak; 1976 Ebola outbreak; Refugee camps

Total land area:	905,564 sq. miles
Population:	44.1 million
Average life expectancy:	48 years
Share of population with access to: Health services: 40% Safe water:	33%
Infant mortality (per 1,000 live births):	108
GDP per capita:	$469
Adult illiteracy rate (15 and older):	26%

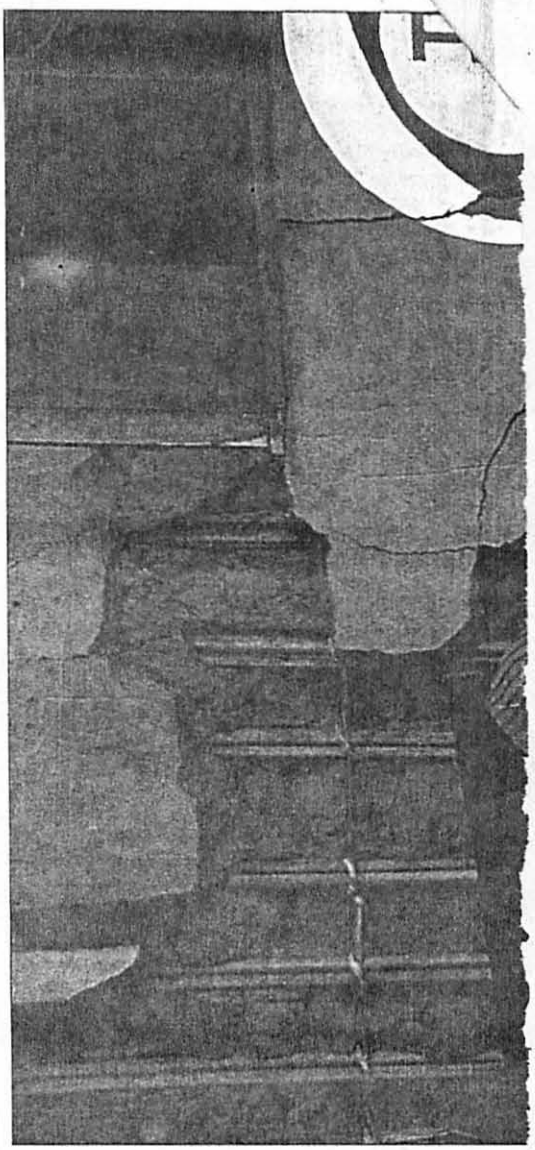
Note: All figures are latest available.
USN&WR–Basic data: Population Reference Bureau, United Nations, Magellan Geographix/Distributed by L.A. Times Syndicate

There were reports of panic in and around Kikwit, where some huts were reportedly burned and many residents fled to outlying villages and perhaps into more remote forests and jungle. The Zairian government tried to impose a quarantine, but Western diplomats said that despite that well-meaning gesture, it was impossible to enforce. Meantime, the Centers for Disease Control and Prevention in Atlanta sent three men from its special pathogens branch to join others from the Geneva-based WHO, Médecins Sans Frontières, Institut Pasteur in Paris and the Institute of Virology in South Africa to help track down the virus and stop its progress.

The relatively quick response prompted experts to say that the chances of the virus's spreading beyond Zaire's borders were remote. Air passengers were being screened with some rigor. The other encouraging factors are that the virus is relatively hard to catch and it kills quickly, lessening the chance victims will infect others. It is transmitted by contact with body fluids like blood, vomit and semen or contaminated syringes and is not known to be passed along through casual contact. That prompted U.S. officials to assure that even in the rare likelihood the virus were transported to America, it would be noticed by well-trained medical personnel and "burn" itself out quickly, with only a small number of persons infected.

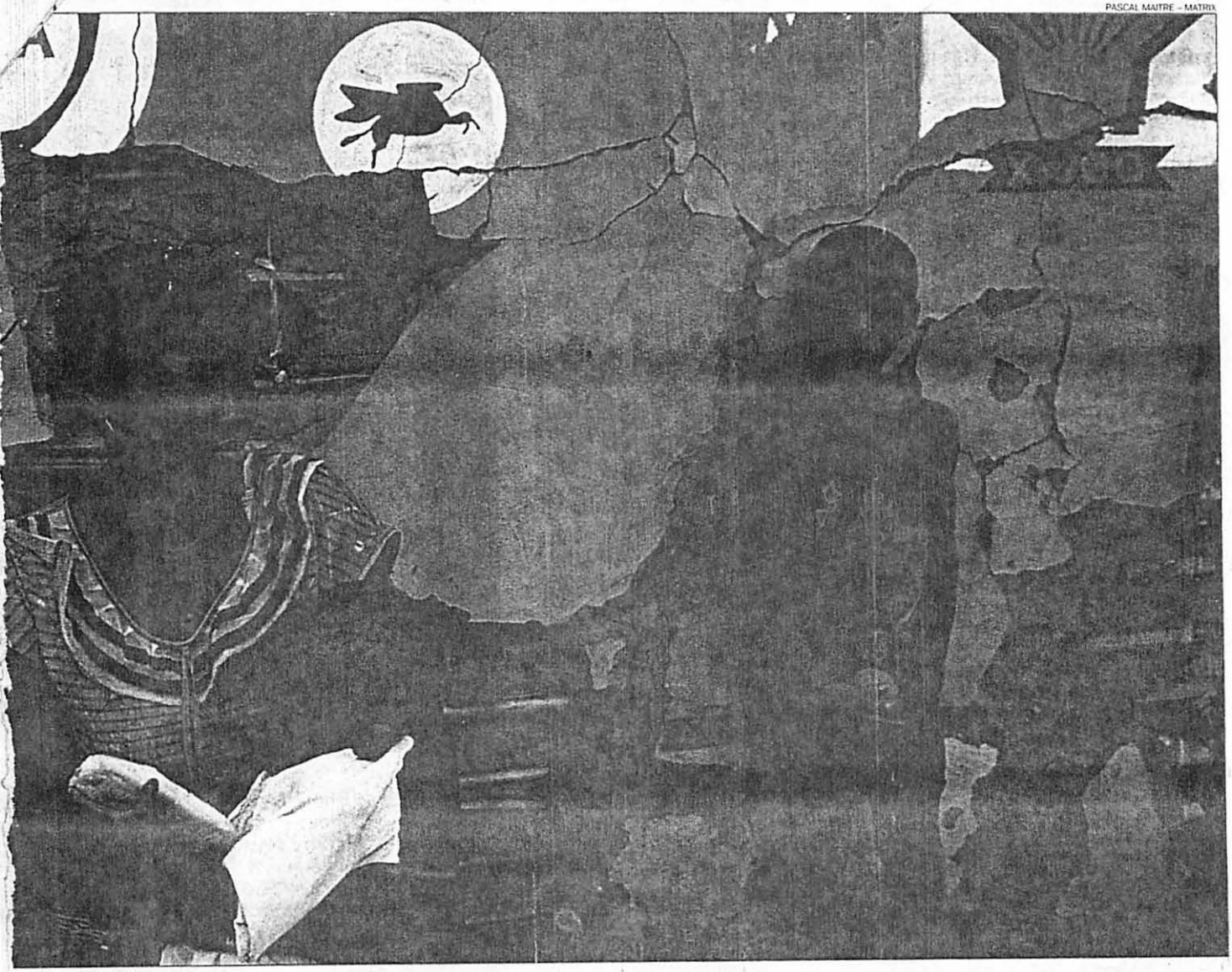
Beyond containing the virus, the top priority of medical teams will be tracing the virus's origin. Their efforts will be watched closely by a growing community of researchers—and anxious citizens—who know that this is but the latest example of how mysterious microbes emerge suddenly to attack people and defy treatment. This new awareness of the threat posed by runaway pathogens has become acute in recent months because of the worldwide popularity of several books and movies. In fact, a growing number of scientists are arguing that the outbreak of new virulent pathogens is traceable to the increasing contact people have with rain forests and the exotic animals they contain. "Development is putting people more and more in contact with these rarer diseases," says James Meegan, a virologist at the National Institutes of Health.

Seeking "patient zero." The painstaking what-done-it investigation is aimed at finding the first patient to contract the virus—the "index patient" or "patient zero, in epidemiological terms." If a disease's human starting point can be found, researchers can trace that person's movements to figure out how the microbe invades humans. In the case of Ebola, no one is sure where the virus



VINCENZO LOMBARDI – AP

PASCAL MAITRE – MATRIX



VINCENZO LOMBARDI – AP

TOUGHER BURDENS. *Life worsened in Kikwit since the nation's civic order collapsed in 1991. International charities filled the gap. At least three of those who died from Ebola were Italian nuns who worked in the hospital, including Floralba Rondi (far left) and Clarangela Ghilardi.*

originates, though monkeys are known to be infected by it. No confirmed patient zero has been found in the three other human Ebola epidemics—in 1976 in Zaire and Sudan and in 1979 in Sudan again. Thus, finding a patient zero could be a breakthrough in helping officials alert civilians to what animals they should avoid and diagnose the illness more quickly.

International health workers in Kikwit say the epidemic began in early April, when a lab technician named Kimfumu appeared at the hospital suffering from acute intestinal disorder. Unsuspecting medical staff operated on him twice on April 9 and 10. He died four days later of what appeared to be Ebola. At least one report from Kikwit said that the entire surgical team also came down with the virus. The WHO reports that at least 10 medical workers involved with Kimfumu have died, including three Italian nuns who worked in or around the hospital.

Ironically, the outbreak appears to have been spread by infected medical workers. At first, doctors did not know what they were battling. Since early January, Kikwit has suffered its yearly epidemic of bloody diarrhea, which has killed at least 100 people and afflicted nearly 100 more in the past four months. Some early symptoms of Ebola look like dysentery, says Innocente Bakanseka, an

administrator based in Kinshasa who works for the British charity Oxfam. "They did not know in the beginning. They did not recognize the virus."

This mistake cost precious time as the infection spread without adequate precautions being taken at medical facilities. The first blood samples were flown in late April to the Tropical Disease Institute in Antwerp, Belgium. American CDC officials did not get samples until May 9. Within 36 hours, the CDC scientists had their answer: The blood from 14 of the 16 patients contained Ebola. The epidemiological team was dispatched and the race against the disease began.

Researchers will face obstacles similar to those that hampered earlier efforts to find the first infected patient. The first diagnosed patient in Kikwit is almost surely *not* patient zero. He was a Kikwit man, and experts believe it is not likely he contracted the disease inside city limits. It is far more probable that the index patient came from a surrounding village and brought the infection to Kikwit.

Even if they track down patient zero, other obstacles might arise. In the first Ebola outbreak in Zaire in 1976, for example, doctors traced the virus to a single man in a small village. They determined that he probably had become infected from a syringe, but they lost the trail after that. They speculate that a stranger passing through the village visited the local clinic, contaminated a syringe or some other piece of equipment and then moved on, dying an unnoticed death on the road.

When scientists do find patient zero, they will still struggle to figure out the original source of the infection. Since the 1976 Ebola outbreak, CDC scientists have combed Zaire for the animal host of the disease, trapping everything from spiders to monkeys, but have had no luck tracking the pathogen. "It takes a massive, massive effort" to search species thoroughly, says Michael Kiley, who studied Ebola for 12 years.

A nice host. The virus could not have picked a more opportune place to strike than Kikwit, a city of several hundred thousand. A decade ago, resource-rich Zaire had one of the best health care infrastructures in Africa. The Belgians, who ruled Zaire until 1960, built a string of hospitals in cities and villages and most provided decent care through the 1980s.

But then all hell broke loose in 1991 when soldiers unpaid by the tyrannical President Mobutu Sésé Séko looted Zaire and quickly sent it hurtling back to another century. In Kikwit, the main road link to Kinshasa fell apart, and the trip these days—if anyone dares it—can take 16 hours through potholes, ruts and roadside threats. Phone lines were hitched to the backs of cars and dragged from poles to the point where U.S. Embassy officials say there is hardly a phone in the city. The once vigorous health care system now lies in tatters. Doctors and nurses do not even have the luxury of disposable gloves or masks to protect themselves and prevent the spread of disease, much less medicine for their patients. "We are still functioning in the Middle Ages," worries Pakisa Tshimika, a public-health expert from Kinshasa with the Mennonite Brethren.

Rain forests' revenge. The biggest mysteries about new killer viruses are where they reside and how they invade humans. Scientists know Ebola infects monkeys, but the virus kills them so quickly that they can't be the pathogen's natural "reservoir." The reservoir species of any microbe must tolerate the germ fairly well. Otherwise, the germ will kill off all its own hosts, a "strategy" that doesn't allow the pathogen to survive indefinitely.

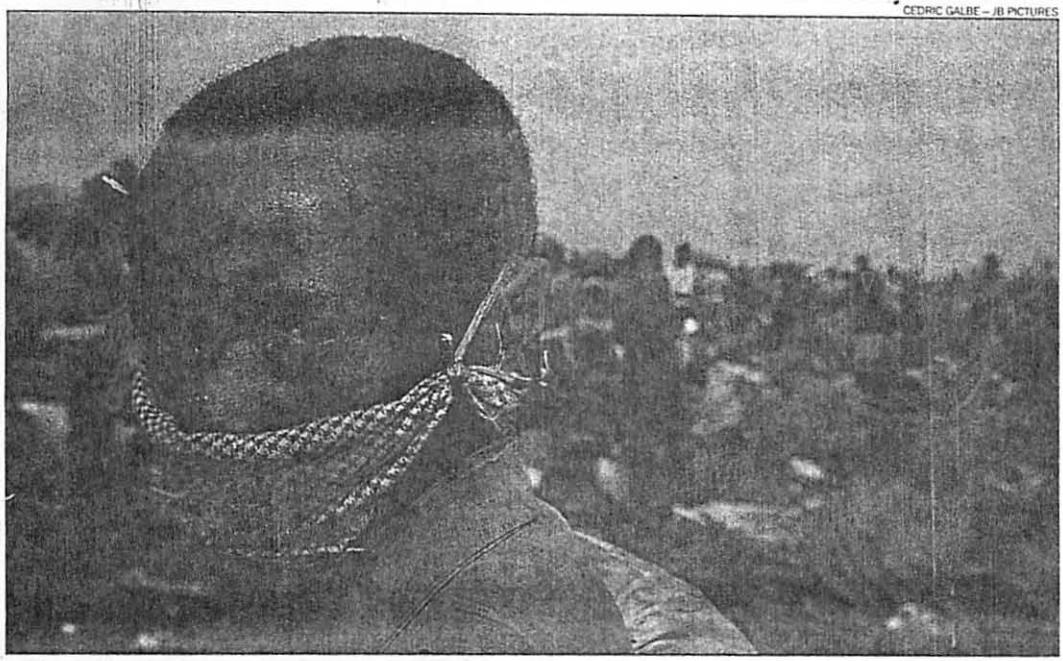
PATRICK DE NOIRMONT—REUTER

MOBUTU'S WAY. *The Zairian dictator has kept power by making sure that Army members are the first, and sometimes the only, government workers who get paid regularly. He paid dearly in 1991 when he stopped their checks and saw the country looted beyond imagination. Outsiders claim he has also plundered the economy himself and amassed a fortune valued at over $10 billion.*

Ebola is not the only mystery pathogen. A close relative, the Marburg virus, also kills monkeys and people; it, too, has no known reservoir. The first Marburg outbreak occurred in 1967 in European lab monkeys imported from Africa. Seven people died. Scientists suspect that somewhere in Africa, a creature lurks that harbors Marburg or Ebola or both. But no one knows what it is—and it's impossible to tell people how to avoid outbreaks until the reservoir is found. "Until we can do that, we're at the mercy of nature," says Karl Johnson, a veteran pathogen investigator.

Not much is known about how germs jump from animals to humans. Organisms that take up residence in a new, human host are called zoonoses. Many are benign and can even come from house pets. But some zoonoses kill. Many scientists believe AIDS, for one, is a zoonosis. The AIDS virus, called HIV, is similar to a monkey virus called simian immunodeficiency virus, or SIV. Monkeys infected with SIV don't get sick. That means SIV may have infected a human, then mutated into HIV, says Beatrice Han, a virologist at the University

CEDRIC GALBE – JB PICTURES



THE OTHER CRISIS. *Fleeing ethnic strife, 1.2 million Rwandans live in camps in the Zairian towns of Goma and Bukavu. Thousands of Hutus tried to return home earlier this year, but the repatriation drive fizzled amid fears of new Tutsi violence. Disease and sanitation problems plague the camps and violence breaks out at times. But Zaire's hope of sending the refugees home probably won't be fulfilled soon.*

of Alabama at Birmingham. But it's not known whether the virus jumped from monkeys to humans recently or jumped a long time ago and didn't spread.

This is an all-too-common problem. Scientists don't know why some viruses invade humans and others don't. They don't know what triggers such a jump or whether some humans are more susceptible to zoonoses than others.

Nor do they know why so many of the nastiest pathogens—from Ebola to Marburg to the lethal Lassa fever—seem to come out of Central Africa. Some speculate that Africa's high primate population may nourish microbes that hop easily to humans. Others point to the rapid spread of humans into the African wilds, which puts them in contact with increasing numbers of exotic disease agents.

New diseases. All this is speculation. But it is clear one of the biggest allies of microbes is environmental disturbance. Anything that upsets the normal state of things—deforestation, climatic changes, road building—can expose humans to unusual pathogens, resulting in a rare- or unknown-disease epidemic. That means as greater incursions are made into the world's tropical rain forests, it is almost inevitable that new diseases will emerge. "We're going to see more of these kinds of surprises," warns CDC infectious disease specialist James Hughes.

Evidence for this comes from all over the globe. When the dry forests of South and Central America were chopped down, a microbe called triatomine moved out of rabbit burrows in the jungle and into thatched huts, bringing along the parasite that carries Chagas' disease, which damages the heart, intestines and nervous system. When the Trans-America Highway was built, the workers, pushing through undeveloped forest, were struck by epidemic after epidemic.

In the west African country of Mauritania, an epidemic of Rift Valley fever killed 244 people soon after a dam was completed on the nearby Senegal River. Rift Valley fever, which causes headaches and joint pain and can progress to hemorrhaging, is carried by mosquitoes. A similar Rift Valley outbreak occurred in Egypt in 1977, several years after the Aswan Dam was built on the Nile.

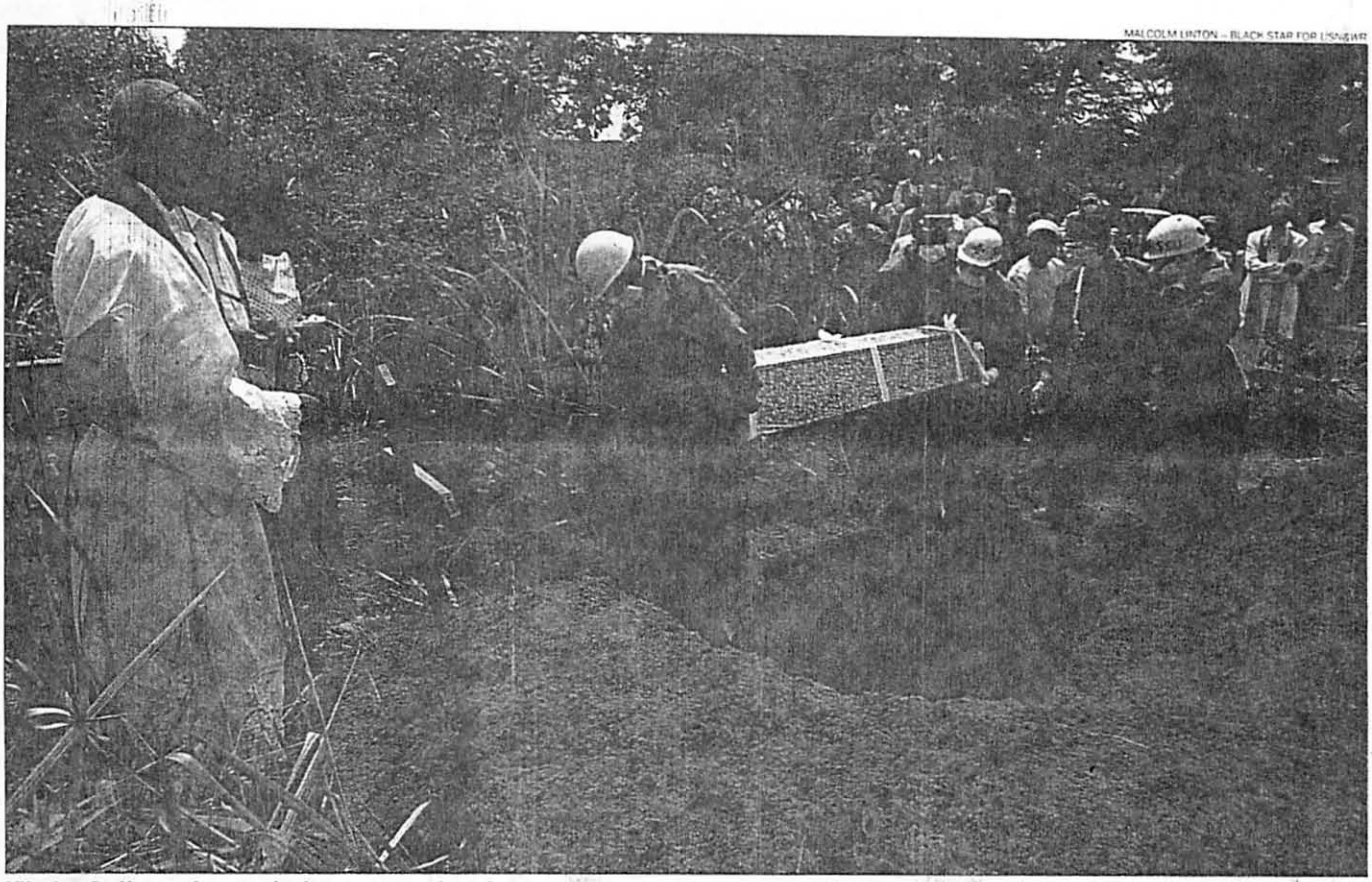
GERRY ELLIS NATURE PHOTOGRAPHY

INFECTIOUS AGENT? *Monkeys are suspected of passing along some of the worst viruses, like Ebola, to humans.*

Even the highly developed United States isn't immune to such upheavals. After the 1994 earthquake in Southern California, an epidemic of "Valley fever" hit about 200 inhabitants, killing three. The fungus is always present in the ground, but only the 6.8-magnitude tremors shook it out of the soil and into people's lungs. In the Southwest, an especially heavy rainfall is blamed for a 1993 epidemic of hantavirus, which can kill healthy young people in a matter of hours by causing their lungs to fill with fluid. The connection: The abundant rains produced a bumper crop of seeds, which produced a bumper crop of mice, which carry the virus. Some scientists also think that global warming may increase the range of disease-carrying mosquitoes in the United States.

Still, it is hard to imagine that any future outbreak will be more terrifying than the one that has struck Kikwit. For hundreds of people there, the next few weeks will be filled with fear. Oxfam's Bakanseka says that one of the charity's administrators in Kikwit was admitted to the hospital May 9 with clear signs of Ebola. He had visited a friend in the final, devastating stages of the illness. Now, another Oxfam colleague, Freddie Malomba, has shut himself in his home in self-imposed quarantine. Alone, he waits for the onset of the deadly fever. ■

BY SHANNON BROWNLEE, ERIC RANSDELL IN KINSHASA AND TRACI WATSON WITH FRED COLEMAN IN PARIS AND VIVA HARDIGG



Kikwit. *Coffin makers jacked up prices when foreign donors and the government offered to pay Ebola victims' burial costs.*

The most persistent virus

Ebola is a menace, but corruption is what is killing Zaire

If the Kikwit General Hospital had proper equipment, the 36-year-old laboratory technician named Kimfumu might not have been forced to use his mouth and a glass straw to transfer blood samples from one test tube to another—and thereby become the first known victim of the current epidemic of the Ebola virus. But such is Zaire, a sprawling country where the state and its riches have been plundered for personal gain. At the top of this pyramid of corruption is Mobutu Sésé Séko, Africa's longest serving dictator. At the bottom are the coffin makers of Kikwit who, upon hearing the government and World Health Organization were willing to pay for coffins to bury Ebola victims, immediately quadrupled their prices.

Five years and 14 governments after Mobutu promised to put his massive country on the road to democracy, Zaire remains a diseased body politic where all-pervasive corruption cuts through any barrier, including the Ebola medical quarantine. "The worst virus is bad politics," says Leonard Mashako Mamba, director of the Yolo Medical Hospital in Kinshasa, "those people who are not fighting for the welfare of the nation but for their own piece of cake."

In control. The leader of those people is Mobutu, the country's 64-year-old president. Despite massive opposition, the looting of Zaire's major cities and inflation of 8,000 percent, Mobutu has remained in control. A consummate politician, he has turned the democratic opposition against itself by buying off opponents. Today a majority of the more than 200 opposition parties are thought to be controlled by Mobutu allies, or what Zairians call "taupes" (moles).

But surviving the upheavals and challenges has not been cheap for the dictator, who was once considered one of the world's richest men. Gecamines, the massive state mining corporation whose coffers Mobutu often treated as his own personal bank account, has collapsed from plunder, pillage and ethnic cleansing by pro-government factions in the mining regions of Shaba province.

A little fraying around the edges was in evidence last week when Mobutu, nicknamed "the cock who leaves no hen alone," made a rare public appearance in Kinshasa. His coterie of ministers, glad-handers and hangers-on were showing the subtle signs of tightened purse strings. The Mercedeses in Mobutu's convoy were not the latest models. The French fashions of his female associates were slightly dated. And some male sycophants had even reverted to wearing frayed *chemises Mobutu*—a

strange blending of the Mao jacket and the ill-fated 1970s leisure suit said to be invented by the president himself.

Travel bans against Mobutu by the French, Belgian and U.S. governments to protest his thwarting of democracy are part of the problem. The ban has ended the days when Mobutu and his extended family would take the presidential jet to Paris for a shopping spree or to the United States for a visit to Disneyland.

Robertson to the rescue. Yet Mobutu's diplomatic quarantine may prove as easy to breach as the corruptible *cordon sanitaire* around Kikwit. At his Kinshasa press conference, Mobutu was flanked by American televangelist Pat Robertson. According to diplomatic sources in the capital, Robertson's organization has been involved in Zaire's lucrative and wildly unregulated diamond business since 1992.

Many analysts feel such scenes are part of a larger plan by Mobutu to reinvent himself. And Africa's tragedies have been good to Mobutu. Last year's genocide in Rwanda and the influx of more than 1.5 million refugees forced reluctant Western powers to deal with the only man who could control the explosive situation. And the Ebola outbreak forced them to deal with him again.

Mobutu appears to be positioning himself as a bulwark against the sort of Somalia-like anarchy that Western conservatives abhor because it could lead to costly humanitarian intervention. "What the international community cannot afford is to have such a mess around here," says a source with close ties to the Zairian military, "so Zaire is once again put in the center of interest in Africa."

Many believe that Mobutu has every chance of winning an eventual election. Yet more Mobutu means more corruption, and possibly more Kikwits. By week's end, Ebola had killed 89 out of 124 infected, and the World Health Organization was expecting the death toll to continue rising for some time. Recently in Kikwit, Moungala Kepasa, a doctor from General Hospital, surveyed a makeshift graveyard near the city morgue. "See all these graves? These are my colleagues," he said. "They all died one by one, but we had to keep working even though we knew we might be next because when you are a doctor, you have to keep the faith." That kind of faith is what keeps Zaire functioning, but it remains to be seen how long it will continue in the face of corruption, the most persistent virus of all. ■

BY ERIC RANSDELL IN KINSHASA

IVORY COAST OUTBREAK

In the lair of the Ebola virus

A Swiss graduate student is proof that even hardened killers sometimes spare a victim. Six months ago, the student took ill with the murderous Ebola virus while doing research in an African rain forest. Today, she is back at work in the jungle. Her story, which appeared in last week's issues of the British medical journal *Lancet* and *Science*, gives hope that Ebola may be tracked to its secret lair.

The infected researcher was in prime virus territory: She was part of a team studying a troop of chimpanzees in the Ivory Coast's Taï National Park. The remote park is the largest intact lowland rain forest in western Africa.

Fourth variety. Last November, 12 of the Taï's chimps died. The Swiss scientist, whose name has not been revealed, helped perform an autopsy on one of the chimps. Soon she was so sick with fever, diarrhea and liver problems that she was flown to Switzerland. There she recovered, and not until weeks later did the source of her illness become clear: a new, apparently milder Ebola virus, the fourth known variety.

The new strain is as baffling as its kin. The infected researcher was wearing gloves and mask and cannot recall coming into contact with the chimp's blood. How, then, did she catch Ebola? And how did 23 other people who came into contact with either the infected researcher or the chimp's tissues escape the virus?

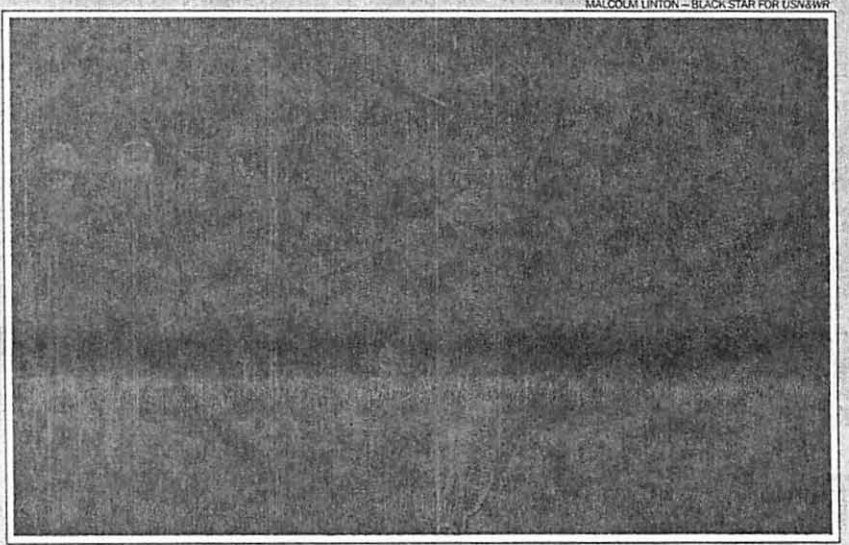
Despite the uncertainties, the Taï outbreak could prove to be the clue that unmasks the Ebola virus. One of the biggest mysteries is the bug's host. Neither chimps nor humans are the natural host, which could be an insect or another mammal. But the Taï chimps must have come into contact with the natural host or an intermediary somewhere in the park.

Virologists from the Institut Pasteur in Paris and the Centers for Disease Control and Prevention in Atlanta plan to test many of the park's species for Ebola. The 15 years of extensive records on the chimps' behavior may hold clues. For example, male chimps hunt animals such as red colobus monkeys at the end of the rainy season—which coincided with waves of chimp deaths in 1992 and 1994.

Those very deaths, however, threaten the search for the virus. Researchers spent three years accustoming the Taï troop to humans. But so many of the male chimps have died that the troop will probably break up. It will be at least a year before a new troop allows close human observation.

BY TRACI WATSON
WITH SHANNON BROWNLEE

MALCOLM LINTON—BLACK STAR FOR USN&WR



Will it happen elsewhere? *The body of an Ebola virus victim in Zaire*

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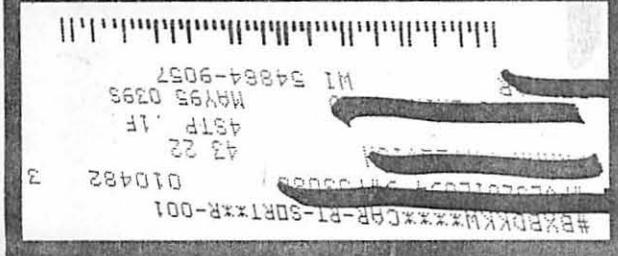
Tales from THE HOT ZONE

How shock troops from the Centers for Disease Control hunt down killer microbes



Researcher at a CDC biohazard lab

THE DISEASE BUSTERS

When an epidemic threatens, shock troops from the Centers for Disease Control are the first into battle

Paul Mead heard about the call late on a chilly Wednesday in January. A cruise ship on its way from Acapulco to Fort Lauderdale needed help from the Centers for Disease Control and Prevention in Atlanta. At least 12 passengers had come down with severe diarrhea. Word passed quickly from office to office at the CDC.

Could it be a real epidemic? It might be nothing more than a few cases of stomach flu among the ship's 861 passengers. But the epidemiologists knew that the 12 reported cases were just those who showed up at the ship's infirmary; the true number was probably much higher. Maybe it's shigella, somebody said, the debilitating organism that had ripped through the Rwandan refugee camps. It might be cholera. That would be very serious.

None of the epidemiologists wished a terrible illness on the passengers, but they all secretly hoped the situation was just serious enough that one of them would be sent to the ship. That's what the CDC's epidemiologists do; they are the Sherlock Holmeses of epidemics. That is the word they use: *epidemic*. *Outbreak* seems less menacing; at least it did until the new hit movie came along. *Epidemic* shouts, "Pestilence!" "Panic!" But, in fact, when an illness arrives suddenly and sweeps through a town or a cruise ship or part of an entire nation, that's an epidemic. For the CDC's epidemiologists, each epidemic is a chance to vanquish a microbe.

When Mead got the word two days later he was being sent out, two emotions rose in him. He was excited, and within two days his enthusiasm would be justified. By the time he boarded the ship in Cozumel, Mexico, 434 passengers had been stricken. But there was a nagging little voice in his head that said, "Don't screw up."

Every one of the 20 young epidemiologists assigned to the CDC's Center for Infectious Diseases has heard that little voice. There are two ways to screw up on an epi-aid, which is short for epidemic aid, CDC parlance for being summoned to track down an epidemic. One, you can fail to find the organism that is making people sick. That happened to Mead on his last epi-aid. He sent samples of blood and stool back to the lab at CDC, and not one of them contained a disease-causing bug. Big humiliation. He was still getting teased for that one. You can also screw up by failing to locate the source of the bug. If it's in the water on a cruise ship, say, and you don't figure that

CDC AT A GLANCE

- **Beginning:** Formed in 1946
- **Budget:** $2 billion; $130 million of it for infectious diseases
- **Work force:** 7,000, of whom 1,000 battle infectious diseases
- **Top three concerns, 1946:** Malaria, typhus, dengue fever
- **Top three concerns, 1995:** AIDS, immunization, chronic illnesses like heart disease and cancer
- **Greatest hits:** Globally, helped wipe out smallpox; in U.S., polio
- **Greatest error:** In 1976, spent $135 million in swine flu panic

KEVIN HORAN FOR USN&WR

FAA guidelines by Nov. 1, 1992—a tight deadline for all of the airlines. In an Aug. 27, 1992, letter to Simmons, inspector Robert Wieckowski laid out the requirements for the new de-icing regimen. They included providing documentation that the type of de-icing fluid used was proper for the ATR-42. That letter prompted a flurry of letters back and forth. McDonald of Simmons says that Wieckowski was demanding that the airline apply a 1 percent weight penalty to ATR planes because the de-icing fluid would cling to the plane's skin, adding weight. McDonald called the penalty unnecessary and said it would mean displacing passengers and luggage. But Wieckowski did not back down. On Sept. 28, 1992, he even wrote AMR Eagle President Robert Martens, requesting his help.

Impasse. In the month that followed, no solution was found. On Oct. 28, 1992, the chief FAA inspector overseeing the Simmons fleet notified the agency's focal-point office in Dallas that he was rejecting the Simmons plan. The letter noted that Simmons lacked even a description of conditions under which de-icing would be necessary. The plan, the letter stated, also failed to explain how de-icing fluid would affect the ATR's flight characteristics, and it included confusing language in a formula for mixing the de-icing fluid, a problem that had led to "a prior icing accident." The letter, sent by inspector Walter Moor, closed by noting that "the history of the ATR aircraft has been extremely precarious while operating in icing conditions." (A Simmons spokesman says today that the problems in the de-icing program raised in Moor's letter have since been corrected.)

The impasse was finally broken—but not in the way FAA inspectors had expected. A day before Moor sent his letter, David Hanley, manager of the FAA's flight standards division in the Great Lakes office, wrote to Simmons President P. A. Piper to inform him that he was approving Piper's October 13 request that the Simmons inspection certificate be moved to the FAA's Dallas office. Hanley says the change was due to Simmons's anticipated move to Dallas and its acquisition of Metro Airlines. He says the de-icing program was not raised in those discussions and that it "was not an issue."

Meanwhile, earlier this month, another ATR rolled slightly, but unexpectedly, during icing conditions between Los Angeles and Las Vegas. The FAA is investigating. ■

BY STEPHEN J. HEDGES

airline made its request on Oct. 13, 1992. It won approval 14 days later. Although Simmons's inspection certificate moved, the company's primary maintenance facility stayed in Marquette, Mich., until just a few months ago.

The Simmons plan for de-icing airplanes on the ground had nothing to do with the crash of American Eagle Flight 4184 last October. That plane had been flying in icing conditions for more than half an hour before it rolled and plunged from the sky. Simmons says the certificate transfer had been in the works for months, ever since it began negotiations to buy Metro Airlines in Dallas. Simmons closed that deal in December 1992, says Lance McDonald, Simmons's vice president for operations, and it moved its corporate offices to Metro's Dallas hangar. McDonald says the notion that Simmons moved its certificate because of the de-icing program "is absolutely incorrect. The reason we moved the certificate is because we were already going to purchase the assets of Metro Airlines." McDonald notes that the same de-icing program that FAA inspectors in the Midwest rejected was approved by other FAA inspectors for three other American Eagle lines.

The FAA defends its action. "The movement of that certificate had to do with the airline's request," says Bill White, the FAA's deputy director for flight standards services. "It had nothing to do with the de-icing program." Though White agrees that the transfer was quick, he says it was part of AMR Eagle's long-range plan to transfer the certificates for all four regional carriers to Dallas. AMR Eagle has not moved the certificates of its three other carriers.

The certificate change incensed FAA inspectors in the Great Lakes office, who had extensive experience with Simmons's growing ATR fleet and with winter weather. Their objections remained an internal FAA affair until the Roselawn accident last October. In the course of investigating that crash, National Transportation Safety Board examiners also delved into the practices of the FAA, Simmons and Avions de Transport Régional, the French-Italian consortium that makes the ATR planes. A review of NTSB documents by *U.S. News* yielded several unusual findings:

■ A report that notes an FAA inspector's concerns about an ATR's flight in icing conditions a full nine months before the Roselawn crash. The report concerns a November 1993 incident in which a Simmons ATR-42 experienced unusual pitching as it descended into Marquette, Mich. Ice on the plane's wings and tail was suspected. The report states that Jerry Barron, the Dallas-based FAA inspector assigned to Simmons, convened a meeting with pilots, flight attendants and Simmons officials in Chicago in January 1994 to investigate the ATR and icing conditions. A second meeting was held a month later in Dallas. Officials from ATR attended that session, as did Simmons officials. Barron, the FAA report notes, "continues to be concerned with potential icing problems on the ATR aircraft." Apart from him, however, "the seriousness of the incidents was downplayed by the majority of the attendees," the report says. Barron later told investigators that ATR was "not receptive to the idea that something was wrong with their aircraft." Instead, he said, ATR attributed the November 1993 mishap to "a jittery crew and suggested that perhaps drug tests for the pilots were in order."

■ The FAA has informed the NTSB that it cannot find an agency report entitled "ATR-42 icing problems." (The ATR-42 is a smaller but similar version of the ATR-72.) The report, known as a continuing airworthiness statement, examines at least five icing incidents involving ATR airplanes in the 1980s.

■ The Midwest inspectors assigned to Simmons experienced conflicts with the FAA's Southwest Region office over AMR Eagle's management of Simmons. AMR wanted to standardize operations for its four commuter airlines to create "seamless service" between the regional carriers and American Airlines. To do that, the FAA established a "focal point" office in Dallas through which all of the AMR Eagle safety changes would flow. Inspectors in the field felt the arrangement undercut their efforts to monitor the planes, maintenance procedures and pilots. In June 1990, five Great Lakes inspectors wrote to Washington to "express our disgust at the method employed by the Southwest Region in concert with AMR Eagle Inc., under the ploy of the standardization of the American Eagle carriers." The inspectors said the focal-point program was "strictly an economic program with the buzz words safety and standardization used to enhance the plan's acceptability."

Of the FAA correspondence unearthed by the NTSB, some of the most intriguing involves the conflict over Simmons's de-icing plan. In March 1992, after the crash of a Fokker F-28 jet at New York's La Guardia Airport killed 27 people, the FAA announced a new de-icing regimen. The method each airline employed to clean snow and ice off its aircraft before takeoff would have to conform to new

TOM STRATTMAN - AP

Investigating. *In Roselawn, looking for clues to the crash of Flight 4184*

never practiced before the bar, took a lead in the recent House debate over tort reform. "We have to keep small companies from being engulfed by frivolous lawsuits," asserts Christensen. "We've all heard of the family business forced into bankruptcy by a single judgment." The first-termer from the Omaha area lobbied to expand the scope of the legal reform bill by successfully coauthoring two amendments. The first would limit "emotional distress" awards in medical-malpractice cases to $250,000; the second would end the practice of forcing defendants found only slightly at fault to pay an entire damage award.

Debt. John Baldacci, 40, one of the few Democrats in the House's freshman class, knows what it feels like to almost lose a business. But it was too much bank debt—not a lawsuit—that got his family's Bangor, Maine, restaurant in trouble. Baldacci was forced to drop out of college temporarily in 1974 after Mama Baldacci's, which he runs with four of his seven siblings, went bankrupt. The restaurant was eventually revived. But two years ago, after his father died, Baldacci had to ante up again. This time, he had to invest most of his life savings to help pay down more debt that had accumulated. Today, when Baldacci returns to his district on weekends, he opens his family's working-class eatery in the morning, makes the coffee, turns on the ice machine, performs minor repairs and then waits on tables Saturday nights. "Small-business people unlock the door in the morning and lock it again at night," declares Baldacci. "That is our life, and no one from Washington can help us. We just don't want them to hurt us."

The class of 1994 in the House of Representatives has embraced the credo of small business in an unprecedented way. But it's too early to tell whether the entrepreneurs who have migrated from the private sector to public service will have a lasting impact on Congress—and on commerce. Some analysts believe that despite their dynamic drive and determination, these new House members will ultimately fall short. Argues Barbara Sinclair, a political scientist at the University of California at Riverside: "Small-business people tend to think that their reality is the only reality." Perhaps the most that can be said thus far, however, as the 104th Congress re-examines the size and role of government, is that small-business legislators have brought a real hardheadedness to the debate. ■

BY LINDA GRANT WITH DAVID HAGE

TOM STRATTMAN, AP

Grieving. *Relatives of those killed on American Eagle Flight 4184*

The feds, a troubled plane and tragedy

Behind the controversial ATR aircraft

U.S. NEWS INVESTIGATIVE REPORT

In October 1992, two years before one of its sleek ATR-72 commuter airplanes crashed in an Indiana farmer's field, the executives of Simmons Airlines faced a dilemma. Federal Aviation Administration inspectors assigned to watch over the Simmons fleet were refusing to approve the company's new de-icing program, which was designed to remove dangerous levels of ice from the planes before takeoff. The inspectors said there were deficiencies in the plan. The time it would take to fix them was running out. If Simmons didn't get the de-icing plan approved by Nov. 1, 1992—the FAA's mandated deadline—its planes could have been prevented from flying into winter weather.

What happened next was unusual. Just five days before the deadline, the FAA approved a Simmons request to move the inspection authority over its fleet from the agency's Great Lakes Region office to an FAA office in Dallas, where Simmons's parent company, AMR Eagle Inc., is located. (That company is in turn a subsidiary of the AMR Corp., which operates American Airlines.) Three days after that—and just two days before the FAA's de-icing deadline—the FAA's Dallas office approved the de-icing plan. Simmons was not required to make any of the changes demanded by the FAA's Great Lakes office.

Moving an airline's operating certificate usually takes months. The Federal Aviation Regulations require notice "at least 30 days in advance" for such a move. In the Simmons case, the

TOM STRATTMAN, AP

Explaining. *ATR flight-test director Gilbert Defer*

■ SCIENCE

Ants in Our Pants

Forget killer bees. Here's a bug from south of the border that's even more frightening

By MICHAEL D. LEMONICK

ONE PAINFULLY MEMORABLE DAY THIS spring Jack Reese did a crazy dance in the middle of a persimmon grove on his farm in Oktibbeha County, Mississippi. Flailing wildly, he tried to yank off his pants and swat his ankles at the same time. He had made one of the worst mistakes a Southern farmer can make: he forgot to watch the ground for a moment and thus tromped on a foot-high mound full of fire ants. Incensed by the intrusion, the insects promptly swarmed up Reese's legs, stinging him mercilessly over and over again. It felt like dozens of hot needles being plunged into his skin.

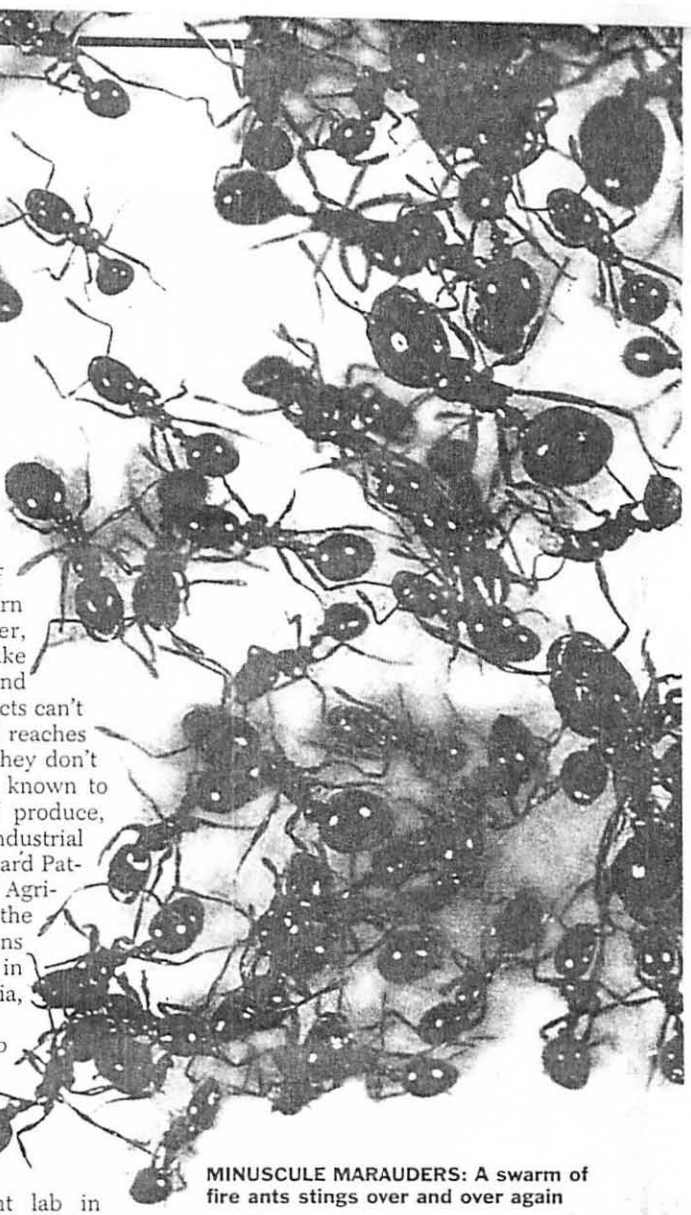
Killer bees, it turns out, are not the most menacing marauders to hail from South America. Their less publicized cousins the fire ants are more widespread in the U.S., more destructive and, so far, deadlier. The antagonistic ants have been harassing people, mostly in the South, for decades—ruining picnics, forcing the cancellation of high school football games, making small children afraid to venture into their backyards—and the threat is getting worse than ever. In some areas the rapidly spreading ants are crowding out (or killing) other insects, lizards, birds and small mammals, knocking natural ecosystems completely out of whack. Their mounds—up to hundreds of them per acre—have made many a farm field all but unplowable. And because the ants are strangely attracted to electric current, they have been known to chew through underground cables, disrupting everything from telephone service to airport runway lights and even starting fires.

While the minuscule monsters have traditionally attacked only people who stepped on their turf, they've recently brought their mayhem indoors as well. Says Marion Bernhardt, 78, of West Palm Beach, Florida, who last year survived an ant assault in a hospital bed: "I was stung all up and down my legs, and I had welts all over them and on my side. They burned for days. I never had such an experience in all my life." She was lucky. At least 50 people have died in recent years from allergic reactions to fire-ant stings.

Worst of all, fire ants are on the move. They are already established throughout the South, from Texas east to Florida and north to Tennessee, with isolated pockets even farther north—there's a colony, for example, in Virginia Beach, Virginia. Most of the West Coast, from Southern California up to Vancouver, British Columbia, would make fine fire-ant habitat as well. And while the moisture-loving insects can't spread westward through arid reaches of West Texas on their own, they don't have to. Fire ants have been known to hitch rides on truckloads of produce, nursery stock and even industrial chemicals. According to Richard Patterson, a U.S. Department of Agriculture entomologist based at the University of Florida, infestations of fire ants have been found in Arizona, New Mexico, California, Oregon and Washington.

So far, the bugs' aversion to frost has kept them out of the Midwest and Northeast, but even that may change. Tim Lockley, an entomologist at the Agriculture Department's fire-ant lab in Gulfport, Mississippi, says the ants have now settled in the mountains of east Tennessee, where as much as 7% of the population survived the especially frigid winter of 1993-94. Says Lockley: "It's just amazing how adaptive they are."

The threat began in the 1930s, when the aggressive red fire ants came to Mobile, Alabama, perhaps on shiploads of lumber imported from the insects' home territory in South America (the milder-mannered black fire ant had arrived, also from the Southern Hemisphere, in 1918). In the 1950s and early '60s concerned government officials tried to eradicate the insects with such powerful chemicals as heptachlor and mirex. The program was later dubbed "the Vietnam of entomology" for both its destructiveness and its futility. The poisons killed not only their targets but also most other wildlife in the treated areas. By the late '70s the pesticides were banned.



MINUSCULE MARAUDERS: A swarm of fire ants stings over and over again

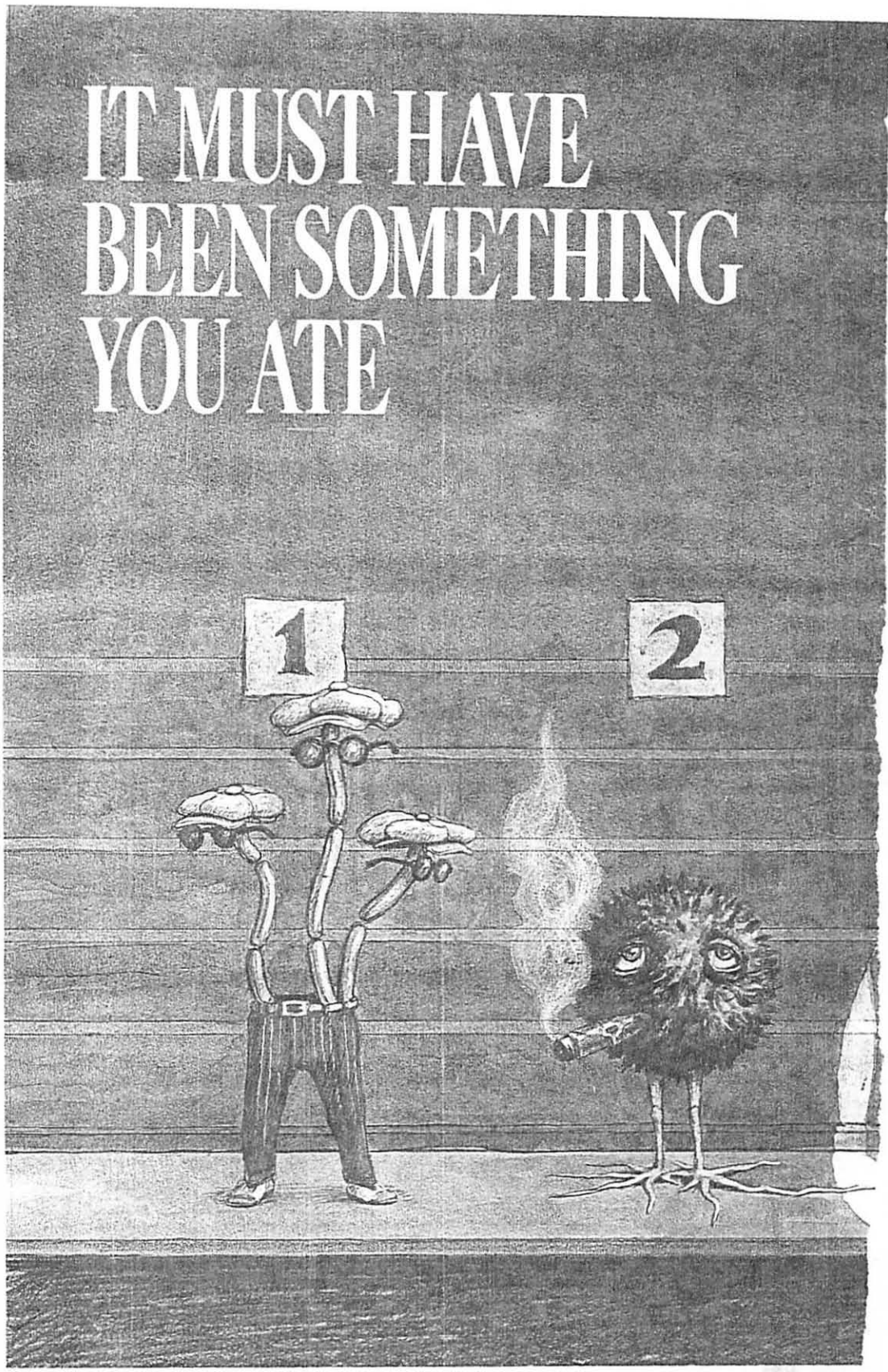
PAUL A. ZAHL, PHD PHOTO RESEARCHERS

Now the ants have grown so nasty that some folks argue for a return to chemical warfare. Says Republican Congressman Tom DeLay of Texas, the House majority whip and a former exterminator: "The scientific evidence doesn't justify the mirex ban."

Still worried about the pesticides' impact on the environment, government scientists think they may have a better answer to the fire-ant menace. Patterson's lab at the Agriculture Department is studying a tiny parasitic fly that lays its eggs right on the fire ant's body. The fly maggots then eat their way into the ant's head and eventually sever the head from the body. Best of all, the fly seems to attack only fire ants. If laboratory and field tests show that the fly is indeed safe to use, says Patterson, the natural ant killer could be available within a year—and Jack Reese will no longer have to be so careful about where he steps. *—Reported by David Bjerklie/New York and Scott Norvell/Atlanta*

BY PENNY WARD MOSER

IT MUST HAVE BEEN SOMETHING YOU ATE



The force was with me. Or, rather, inside me. And it was trying to get out in a hurry. My intestines felt as if they were playing host to a Bears-Raiders game. I was sick.

It has happened to all of us. In a few hours we go from well to wretched—and retching. Most of the time we blame the flu, and we even make up strains to suit the occasion, like "the twenty-four-hour bug that's going around."

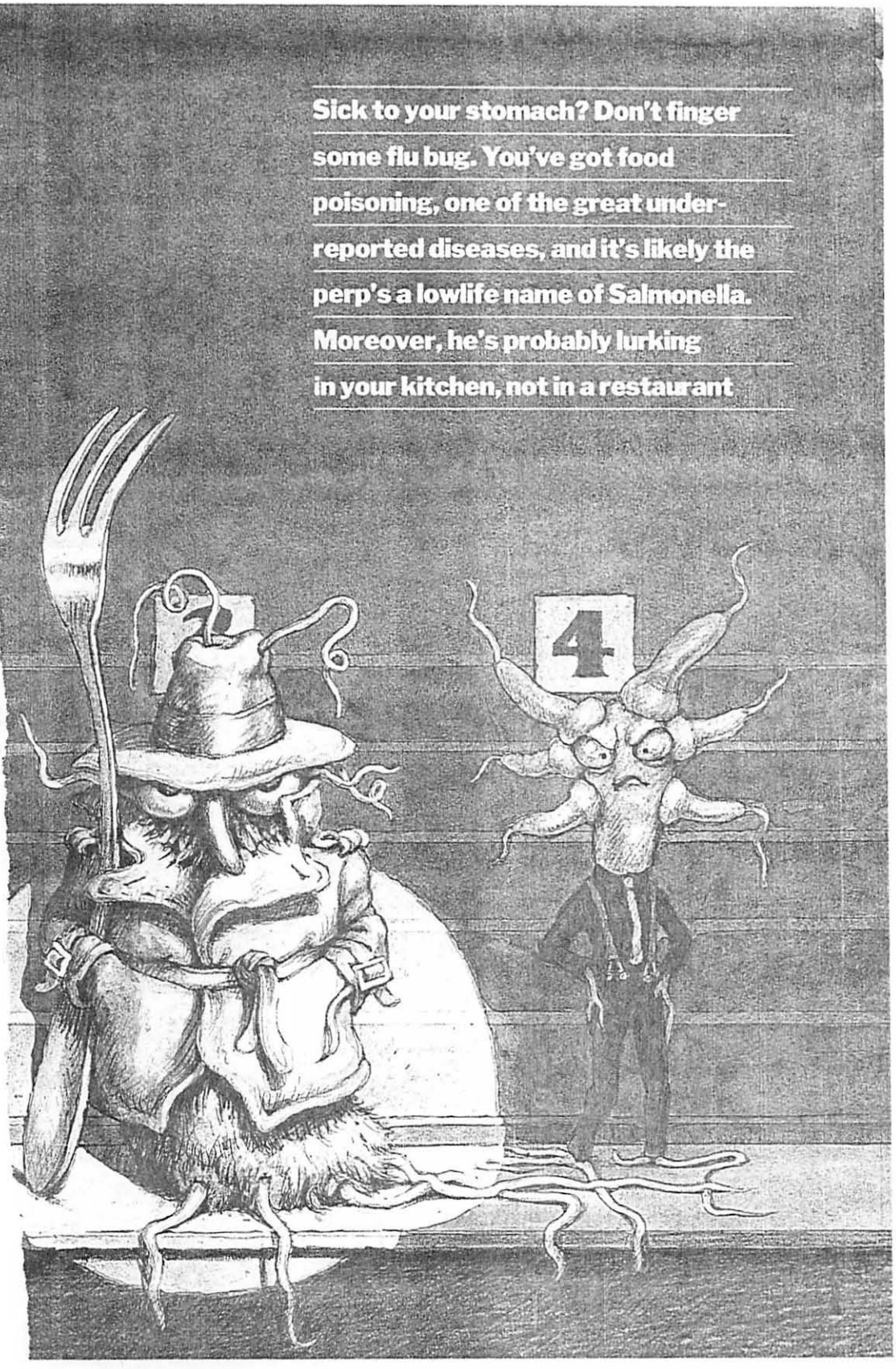
But influenza is getting a bad rap. Last year in the U.S., perhaps as many as 81 million times, what made us sick was something we ate. Roughly nine thousand people died of food poisoning, or food-borne illness as government health officials call it.

My affliction was salmonellosis, so named because it was the result of my ingesting salmonella bacteria. The strain that got me was *Salmonella enteritidis*, from an antigenic group that includes the rare—at least in the U.S.—and deadly *Salmonella typhi*, the cause of typhoid fever. The primary symptoms of *Salmonella enteritidis* are a headache, followed by days of what doctors term "unrelenting diarrhea." The usual treatment is to tough it out. My week of toughing it out included a diet of soy milk, cornflakes, and rice selected from a list of foods my doctor recommended.

He also told me that each of us probably gets salmonella poisoning a hundred times during our lives. "Sometimes it makes us very sick and sometimes it doesn't," he said. "We usually pass it off as the flu, but flu is customarily respiratory—more coldlike and achy. If diarrhea is the primary symptom, it was probably something you ate."

While I was recovering, I punched a request into my computer: search the Nexis data base for news references

Sick to your stomach? Don't finger some flu bug. You've got food poisoning, one of the great under-reported diseases, and it's likely the perp's a lowlife name of Salmonella. Moreover, he's probably lurking in your kitchen, not in a restaurant



ILLUSTRATIONS BY ROGER ROTH

to food poisoning during the past two years. It hummed and hummed. Finally, it produced a list of more than 1,000 news articles. From these and other sources, I learned that food poisonings in America are increasing markedly.

The U.S. Centers for Disease Control (CDC) recognizes some 300 food-borne illnesses, from common salmonellosis to the rare tetraodon puffer fish poisoning. It's impossible to determine exactly how often people are laid low by these pathogens—the 81 million cited above is the roughest of estimates—because most victims never know what hit them. Even the infections of sufferers who seek medical attention are usually undiagnosed, because doctors allow the infections to run their course and seldom order tests to pinpoint the bacteria involved. A victim of food poisoning is likely to receive a specific diagnosis only if he becomes very, very sick, or gets sick at the same time and in the same way as a whole bunch of other people.

And the articles provided me with startling instances of food-borne illnesses:

- In the spring of 1985 as many as 200,000 people in the Midwest got salmonellosis. Investigators believe the most likely source was a valve that allowed a small amount of raw milk to leak into pasteurized milk at a dairy outside Chicago.
- In the summer of 1985 *Listeria monocytogenes* bacteria in soft cheese killed at least 80 Californians.
- In August 1985 botulism in salt-preserved fish killed two elderly New Yorkers.
- In April 1986 salmonella in gefilte fish made 56 people in suburban Washington, D.C. severely ill.
- In May 1986, 20 people who had attended a buffet in New Jersey got salmonellosis from

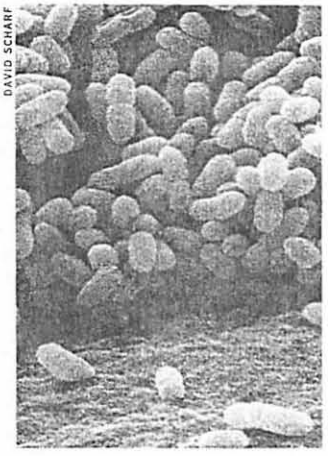
eating stuffed pasta shells and lasagna. This led to a recall of 120 frozen pasta products in 47 states.

- In July 1986, more than a hundred Chicago-area diners got sick from salmonella that health officials believe came from low-acid tomatoes that had been sliced by a carrier who had traces of his own feces on his hands.
- In August 1986 typhoid fever made nine people desperately ill after they'd eaten shrimp salad prepared by a typhoid carrier at a Maryland restaurant.
- In November 1986 five people died in a Connecticut nursing home from salmonella traced to puréed food.
- In December 1986 Nabisco ordered a nationwide recall of millions of Baby Ruth candy bars after traces of *Salmonella meleagridis* were discovered in some lots.

My friends, like my computer screen, were also full of food-poisoning anecdotes. It was my own affliction, salmonellosis, that came up most frequently. This wasn't a coincidence. "Of all the food-borne pathogens we have to contend with in this country, salmonella is the one that's gaining on us fastest," says Robert Tauxe, an enteric disease epidemiologist and salmonella specialist at the CDC.

The Food and Drug Administration believes that in 1985 four million Americans had a member of the *Salmonella* family for dinner. Thirty-five thousand were hospitalized with salmonellosis, which killed more than 1,000 people and left 120,000 others with chronic crippling diseases like arthritis. Says Tauxe, "Salmonellosis was very rarely reported in the nineteen-forties. It picked up steam in the sixties. Today the numbers just keep going up." The reasons, he says, "are many and subtle. You could point to the way we raise our livestock, and the way we process and handle our food." As recently as 1975, only 23,448 cases were reported to the CDC. By last year that number had more than doubled. Those cases were just the tip of that possible four-million-case iceberg. "Whatever numbers you want to use," Tauxe says, "I can tell you that the salmonella problem is one of the great underreported diseases in the country today."

Because it's hard to cook, turkey is the main source of salmonellosis among meats. If it's pink, you may get sick



DAVID SCHARF

The villain, *S. enteritidis*, magnified 21,000 times

A salmonella infection is usually just an unpleasant bout of gastrointestinal distress. The exception is the strain that causes typhoid fever, which is in a class by itself. It becomes systemic, invading the blood stream and infecting many organs, where it triggers severe and sometimes fatal inflammations. Only about 500 cases are reported in the U.S. annually, and two-thirds of those are contracted abroad. Typhoid fever used to kill about one-third of its victims, and even with today's antibiotics, it still does in about one in ten. Non-typhoid salmonella is usually only a killer when it invades the blood stream and colonizes in the brain or other organs. The intestines try to keep this from happening: diarrhea, in fact, is an immune response to ingested pathogenic bacteria. It flushes out the salmonellae that permeate the protective mucous coating and attach themselves to the intestinal walls.

But flushing the system of salmonellae may not end the illness. Says Doug Archer, director of the division of microbiology at the FDA, "Between two and three per cent of salmonellosis sufferers are going to find out weeks later that they have reactive arthritis, which may be contracted because a component of the bacterium somehow fools the immune systems of people who have a certain gene—HLA-B27. Ten per cent of the U.S. population have this gene." That means that if four million people had salmonellosis last year, 120,000 of them will develop chronic arthritis as a result.

A smaller number of salmonella victims—perhaps only one in ten thousand—experience septic arthritis, a painful condition in which the bacteria invade the joints. Still others may suffer post-infection Reiter's syndrome, characterized by inflammation of the urethra, eyes, and joints.

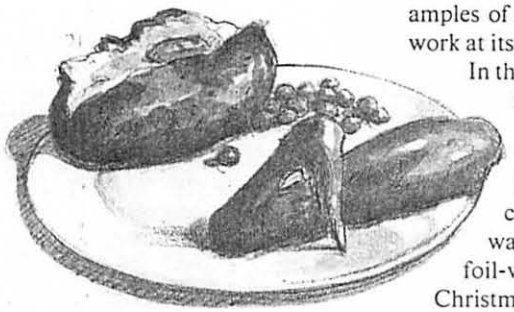
Although farmers and people who process food are well aware of the dangers of salmonella, they're sometimes helpless to prevent its spread. Tauxe and Mitchell Cohen, a CDC colleague, have reported that a growing number of antibiotic-resistant salmonellae are being passed from animals—which are fed antibiotics that eliminate sensitive strains and lead to the development of resistant ones—to man via food. As if all this weren't enough, sloppy housekeeping and food handling is abetting the increase in salmonella infections—especially in the home. Says Tauxe, "A lot of folks don't know how to cook any more."

"It's easy to blame restaurants," says U.S. Department of Agriculture food safety expert Georgia Stevens Neruda, a home economist. "But when it comes to food poisoning, we usually do ourselves in at home."

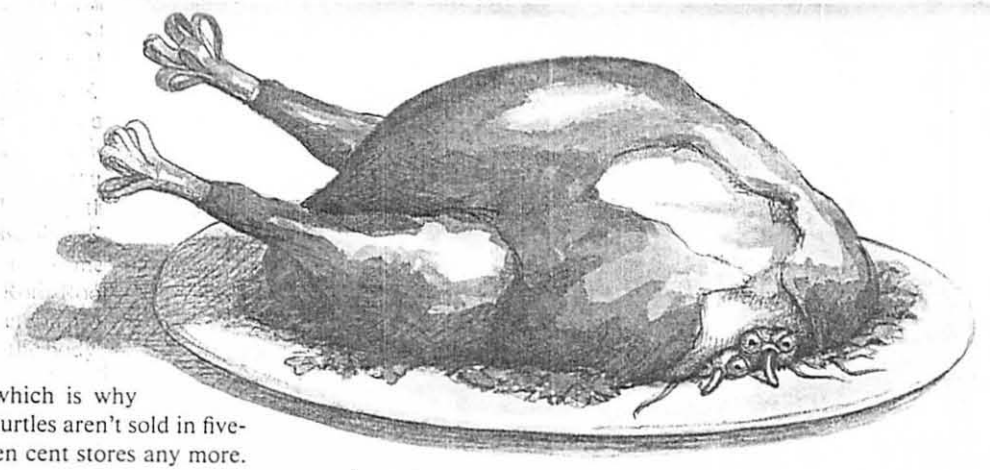
Despite its name, this thing that's waiting to ambush us in our kitchens has nothing to do with salmon. It was named for USDA microbiologist Daniel Salmon, who isolated it in 1885. Salmonella is a general term applied to a group of about 2,000 closely related bacteria. Each salmonella serotype—a subgroup sharing antigens—has its own name. The serotypes that most often

made people sick in 1985 were *S. typhimurium*, which accounted for 49.7 per cent of reported cases, followed by *S. enteritidis* (ten per cent), *S. heidelberg* (nine per cent), *S. newport* (4.3 per cent), and *S. hadar* and *S. agona* (two per cent each). In the 25 years that the CDC has been keeping track of serotypes, it has isolated close to 500 kinds of salmonella in humans.

Some serotypes prefer different species of animals as hosts, but they're all spread in essentially the same manner.



Since salmonella bacteria are shed alive and well in the feces of most animals, and since they're so tough they can withstand very hot and freezing weather, rain and drought, they're found wherever food animals live. The animals pick up the salmonellae from the soil or even from contaminated processed feed. Once consumed, the bacteria live in the animals' intestines, where, depending on their serotype and number, they may or may not make the host sick. Then, during slaughtering and processing, minute numbers of the salmonellae can lead to contamination of food products. That's why the bacteria are found on or in raw meats, poultry, eggs, milk, fish, and shellfish. They're also carried by pets, especially birds, fish, dogs, cats, and turtles. In fact, the CDC determined that 14 per cent of all salmonellosis cases in 1971 had been caused by pet turtles, which is why little turtles aren't sold in five-and-ten cent stores any more.



Some serotypes are geographically specific—or were, until we started shipping them all over the global village. Because of this specificity, salmonella makes for "fascinating epidemiological studies," says Tauxe, who then cites two examples of microbial detective work at its best:

In the winter of 1973-74, a strain of *Salmonella* struck 80 people living in different areas of the country. The source was discovered to be foil-wrapped chocolate Christmas balls. The chocolates were tracked to a Canadian processor, and the bacteria, *S. eastbourne*, were traced to the cocoa beans used in manufacturing the candy. The beans were believed to have come from Ghana, where *S. eastbourne* has been isolated, and the bacteria weren't in the beans, but *on* them. There's speculation that the beans had been contaminated by gull feces while sitting on a dock in Africa.

S. agona was almost unheard of in the U.S. until 1970, when it found its way to North America in Peruvian fishmeal. The fishmeal was fed to chickens, which were then eaten by humans. Today *S. agona* is the sixth most common pathogenic serotype in America.

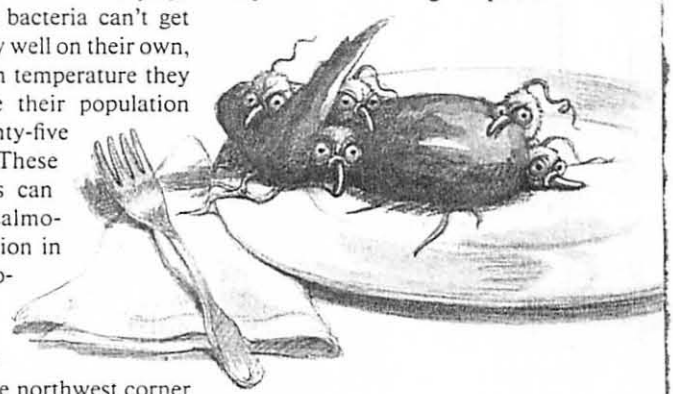
Salmonella shouldn't be confused with the common food poisoning *Staphylococcus aureus*, sometimes nicknamed "Roto-Rooter disease." These bacteria produce a toxin to which the body responds almost immediately by ordering a total evacuation. This provokes violent spasms throughout the digestive tract, frequently causing vomiting and diarrhea simultaneously. However, it's rarely serious and the symptoms last only a few hours.

By contrast, salmonellosis symptoms, which include headache, rumblings in the bowels, diarrhea, and sometimes fever and vomiting, appear between six and 72 hours after the bacteria are ingested. That's why it's often tough to figure out what food did you in, unless you're one of a group of sufferers who all ate the same thing. Even then, salmonella plays tricks. The bacteria can't get around very well on their own, but at room temperature they can double their population every twenty-five minutes. These two factors can make a salmonella infection in food very localized: all the party-goers who eat from the northwest corner of the scalloped-potato pan might be fine, while those who serve themselves from the southeast quadrant become sick. This also explains how it is that two people can eat the same thing at a restaurant, but only one gets sick.

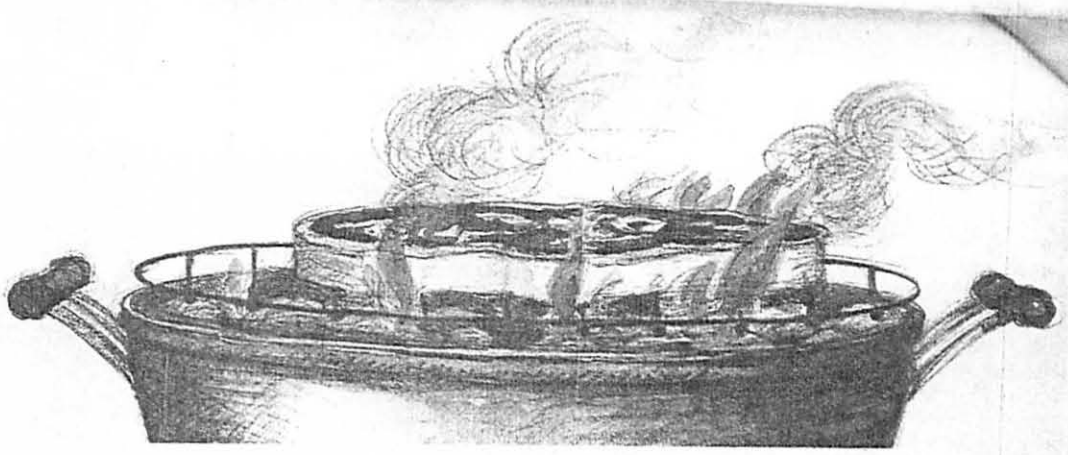
The severity of salmonellosis is also directly related to dosage—i.e., a small amount of the bacteria will make you a little bit sick, while a whole lot will make you a whole lot sick. What constitutes a whole lot can vary from serotype to serotype. It can take a million of some kinds, but the potent *S. ealing* can make you sick if only a single bacterium makes its way down your throat.

Salmonellae find body temperature very comfortable, and if you've eaten food they like—say, a big turkey dinner and eggnog, which neutralize stomach acids—they survive more easily and travel to the intestines, multiplying as they go.

When a sufficient number of pathogenic bacteria reach the intestinal wall, the bowel's defense mechanism responds by manufacturing copious amounts of mucus, drawing water from the blood stream, and pushing everything out of the intestine quickly. By repeating this process frequently over as long a period as necessary, the body will excrete most of the bacteria. However, while the bacteria don't usually colonize in the body, about three per cent of salmonellosis sufferers become carriers, like the infamous Typhoid Mary, who was the direct cause of more than fifty cases of ty-



Make sure the plate that carries the meat in from the grill isn't the same one that carried it out



phoid fever and three deaths while working as a cook in New York in the early 1900s.

Salmonella is transmitted from person to person only about ten per cent of the time—usually when a carrier's feces, not washed from his hands, contaminate food during preparation. Most often, the illness comes from food contaminated by animal feces—meat, eggs, and dairy products, for the most part. The bacteria have been cultivated from protein powders, peanut butter, chocolate, green beans, peppercorns, ground beetles (which were once used to make red food coloring), and even powdered snake sold in health food stores. Salmonella occurs in dairy products when raw milk is used, or when it's contaminated after pasteurization. This is probably what happened in the aforementioned Chicago outbreak: a leaky valve in the dairy's five miles of pipe let raw milk trickle into the bacteria-free pasteurized product.

My salmonella came from eggs. My doctor and I think I got it from an egg-salad sandwich I bought in a New York deli. The eggs, not the mayonnaise in the salad, would have carried the bacteria. When people get sick from eating food with mayonnaise in it, they tend to blame the mayo, but they shouldn't. This misconception is left over from the days of home-made mayonnaise and unpasteurized eggs. Today mayonnaise processors use liquid pasteurized eggs. According to Best Foods, the maker of Hellmann's mayonnaise, there has never been a case of food poisoning linked to modern commercial mayonnaise. "Not only do we use pasteurized eggs, but our mayonnaise has sufficient quantities of acid and salt to stop harmful bacteria right in its tracks," says Phil Wells, Best Foods' expert on microbes and mayo. "Mayonnaise is self-pasteurizing. In salads it retards rather than encourages bacterial growth." By and large, an egg becomes contaminated when a microbe from a dirty shell gets into it when it's cracked open.

In the U.S., turkey is the most common source of salmonella outbreaks from meat, followed by beef and chicken. (Samplings of turkey carcasses by the FDA also showed that half of them were infected with the diarrhea-inducing bacteria *Campylobacter*.) While the USDA says that as many as 37 per cent of chicken carcasses may contain salmonella bacteria (and that chicken is therefore a hazard in the kitchen if mishandled), cooked chicken is infrequently a source of salmonellosis because chicken isn't eaten rare. Turkey isn't usually served rare either, but a turkey is more difficult to cook than a chicken. If the middle isn't done, and the bird sits at room temperature so guests can pick at it all afternoon, they may get sick.

The shocker in all this is that so much of the meat we eat is contaminated with feces. How can this be? In the case of chicken, the demand for huge numbers of low-priced birds—Americans ate 4.7 billion chickens in 1985—is answered by high-speed plants that can process 80 chickens a minute by machine. Contamination of the meat can occur during defeathering, when the machinery sometimes presses the bird so hard that feces spurt from the cloaca and drip onto the feathers. The "rubber fingers" that remove the feathers can then inadvertently press the bacteria onto the skin, where it can hide in the empty feather follicles. Contamination can also occur during disemboweling, when high-speed gutters may rip the intestine, spattering small amounts of fecal matter onto the carcass.

Meat from other poultry, as well as that from four-footed animals, can be contaminated during slaughtering or processing. Ground meat, which is handled a lot, is more vulnerable than other cuts to salmonella, or to cross-contamination, if, say, some liquid from a chicken got onto the beef chunks heading for the burger grinder.

The danger of getting salmonella from beef is compounded by the fact that it's often eaten rare. Salmonella researchers eschew steak tartare, even rare hamburger. Most food safety experts at the USDA recommend that all ground beef be cooked to at least 170°. "It won't be pink any more," says Neruda, "but it'll be safe to eat."

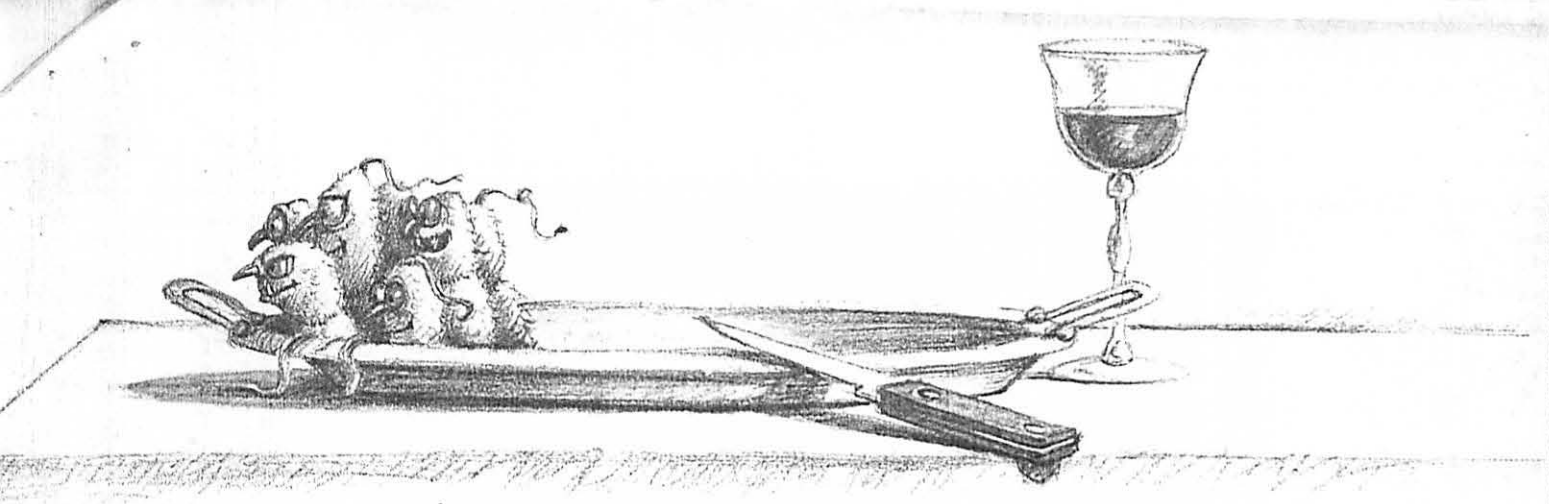
All these forms of contamination occur before the food gets to a restaurant or a home kitchen, where the problems really begin. Contrary to what most people think, Americans are far more likely to get salmonellosis at home than in a restaurant. It's just that restaurant outbreaks attract more attention because large numbers of people getting sick simultaneously make the evening news.

"One problem is that we don't cook like grandma used to," says Tauxe. Since the USDA set up a toll-free meat and poultry hotline* in 1985—in part to deal with salmonella—it has received 50,000 calls. Hotline questions have included "Is it O.K. to eat groceries that my husband left in the trunk of the car for a week?" and "Can spaghetti sauce left open on the counter for three days hurt me?"

"We hear from very young parents, and even children who are trying to cook," Neruda says. "I'd have to say that the breakdown of the family unit has added to the food safety problem."

Then there are the baby boomers who have rebelled against the overcooked food of their youth. "We live in a kind of happy, yuppie, raw-is-better-and-healthier society," says Tauxe. "We want things fast. We want to heat and

*800-535-4555; in D.C. 447-3333

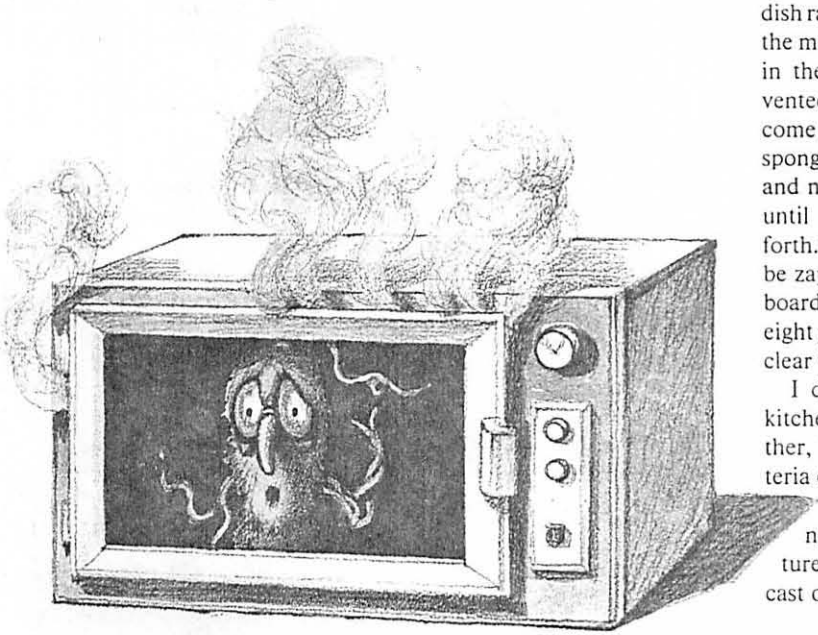


serve. Sometimes we don't wait for things to get thoroughly hot."

The microwave oven should be part of the solution, but sometimes it's part of the problem. Certainly, it's an effective sterilizer: "It'll blow up a bacterium the same way it would blow up a canary," says an FDA official. But some older models don't heat evenly, and different models generate different levels of energy, or use more or fewer microwaves a minute. "It's tough to explain on a label," says microbiologist Dane Bernard of the National Food Processors Association, "but six minutes in my microwave might equal ten minutes in yours."

As I recovered from salmonellosis, I resolved to change my ways. I now never leave any food out at room temperature for more than two hours. I have a thermometer in my refrigerator to make sure it keeps foods colder than 40°. I know that hot foods at a buffet—something in a chafing dish, for instance—should be kept at more than 140°. This means the corners too, not just the part over the Sterno.

Dish rags and sponges, often the most bacteria-laden objects in the kitchen, can be sterilized by nuking them in a microwave oven



I also know all the places salmonellae can hide. I bought an acrylic cutting board for meat, because bacteria love to hunker down in little crevices in a wooden board. Tens of thousands can live in a single knife mark, but an acrylic board can be put in a dishwasher. Salmonellae can also dry up—sort of hibernate for a year or more—and then wake up when something inviting (i.e., your food) presents itself. Wiping a cutting board with hot soapy water won't remove it. In fact, a dish rag or sponge is frequently the most bacteria-laden object in the kitchen. (I've also invented a nifty trick to overcome this. I now rinse my sponge with hot sudsy water and nuke it in the microwave until clouds of steam spew forth.) Salmonella bacteria can be zapped by rubbing down a board with one part bleach to eight parts water, followed by a clear water rinse.

I don't thaw meat on the kitchen counter any more either, because salmonella bacteria can double in number so quickly that a tiny colony left at room temperature for the day can become a cast of millions. I now defrost frozen food in the refrigerator or the microwave oven. I wash poultry in cold water before I cook it, and I'm careful not to let raw meat juices drip on other foods. When I'm finished washing my meat, I rinse my sink with hot soapy water. I also make sure the plate that carries meat back in from the grill isn't the same one that carried it out. Says Stephen Pretanik, the director of science and technology for the National Broiler Council, "The biggest problem with salmonella infection in meat isn't that the meat is undercooked, but that it's re-contaminated in the kitchen after it's cooked. That's why leftovers get you." And the knife, the fork, anything you use on meat—and God forbid it should be a wooden utensil—should be thoroughly washed with hot, soapy water.

I've also learned that when it comes to salmonella, you can't trust your nose. You can't see salmonellae, you can't smell them, and you can't taste them. "A lot of people think that if something left over doesn't make you go 'yuck,' it can't hurt," says Neruda. "They're wrong."

Now that I've got my kitchen under control, the big question is whether I dare dine out again. I mean, whom can I trust? And whom should I blame for all this salmonella that's going around? "What we have to wonder is to what degree is the individual responsible?" says Tauxe. "You can't

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protect yourself against salmonella in every situation. It can't always be dealt with at a personal level. We're vulnerable."

And we may be getting more vulnerable. In their paper on the public health dangers of the increasing numbers of antibiotic-resistant salmonellae, Cohen and Tauxe wrote, "Combined epidemiologic and laboratory studies with the use of new methods in molecular biology make it possible to trace [antibiotic]-resistant salmonellae to their primary source—foods of animal origin." Half of all the antibiotics used in the U.S. in 1985 were fed to food animals—as growth enhancers, prophylaxes, and cures. Tauxe and Cohen think this has fostered resistant strains of bacteria.

Although salmonella poisoning in people isn't usually treated with antibiotics—because such treatment won't make the victim feel any better and may prolong the period during which he can infect others—antibiotic-resistant bacteria are risky for a number of reasons. Doctors sometimes prescribe antibiotics for infants, the feeble, and the elderly to prevent salmonella-triggered local infections or bacteremia. Tauxe, Cohen, and other researchers fear that as the number of antibiotic-resistant salmonellae goes up, the physician's choice of therapeutic antibiotics will go down.

Another dangerous aspect of antibiotic-resistant salmonellae is that they can hit a victim when he's already down. "Say you're taking antibiotics for a sore throat or dental work," Tauxe says. "You've now suppressed the good bacteria in your body, including the natural flora of the digestive tract that helps protect you from the invading salmonella. Along comes some salmonella that happens to be resistant to that antibiotic and it just thinks, 'Oh, boy,' and takes off. Now you have salmonellosis.

"Philosophically I suppose you could say, 'So you get sick. So what? Life's full of hazards.' Myself, I think people ought to be writing to their state legislators about salmonella."

At the FDA, which is responsible for ensuring the purity of milk and dairy products, microbiologist John Kvenberg says, "The load of pathogens coming at us in our food supply today is out of control. At the FDA we've never worked harder, never been busier. We're taking a hard look at critical safety control points during manufacturing."

Over at the USDA, which has jurisdiction over meat and poultry, officials are beset by accusations that they've been asleep at the salmonella switch. "That's not true." says public affairs specialist Danielle Schor. "We realize there's a problem, and we're going after it."

To that end, the USDA is researching ways to help industry clean up its act. "We're looking at adding chlorine or acetic acid to the chiller sprays in meat processing plants," Schor says. "We've petitioned the FDA to allow meat packers to irradiate poultry at levels sufficient to kill salmonellae. We're urging producers to look for safe feeds that aren't contaminated with salmonellae. And, most important, we're looking into voluntary microbial standards for meats. What we'll say to the meat industry is 'You meet these standards, and we're going to let you say it on the label.' We think that if just one major producer has this labeling, the consumer will notice, and the others will fall in line."

The reduction of antibiotics in the food chain, a step that's stalled in the Washington bureaucracy, may soon be forced on meat producers by consumers. In my neighborhood gourmet market, a sign over the pricy birds promises that they've been raised without antibiotics. The same claim is made for beef in a local supermarket. Indeed, in the face of generally slumping beef sales, the demand for low-fat, drug-free beef is rising. But while the FDA has proposed banning the use of tetracycline and penicillin in animal feeds since 1977, Congress, influenced by powerful industry lobbies, has repeatedly asked the FDA to conduct further tests. It's unlikely that action on antibiotics will come during the anti-regulatory Reagan administration.

Some hope is offered by newly patented rapid salmonella-screening tests that allow those who process food to detect contamination within 24 hours—about a quarter of the time older lab methods take. "Still, what we basically have to do is assume salmonella is everywhere," says Bernard, "and process foods to keep bacteria from the consumer."

But just in case some of those bad bacteria find their way into my kitchen, I'll be there nuking my sponges, rinsing my poultry, and junking my yuppie menu. I'd forgotten how good and comforting a pot roast, braised until the beef is tender—and safe—tastes. Now I'm thinking of bringing back chicken fricassee. □

■ MEDICINE

IN SEARCH OF THE DYING

At the center of the Ebola outbreak in Zaïre, relief workers discover the danger is far from over

By NANCY GIBBS

IN THE DARKENED DOORWAY OF AN abandoned building, the medical team finds an empty coffin, waiting like carrion. One by one, neighbors explain, the family that lived there died. First the daughter, 18, went to the Kikwit 2 maternity hospital in late March for a caesarean section. When she got home her incision began to bleed. Then her organs began to melt. The red-black sludge wiggled out of her eyes, her nose, her mouth. Soon her parents got sick. Her father, some villagers believe, died of horror: he told his wife that if she died, he would die too on the next Friday. And he did, followed by another daughter, then two sons, and a nurse who had helped tend them.

Two houses away, a new widow sits and watches the visitors making their way through town. Her husband, she quietly admits, also helped take care of the sick family. Then he died. She buried his body, but the mattress where he lay sick is still in the house. Dr. David Heyman of the World Health Organization listens to her story, and his heart sinks. He knows as much about the lethal Ebola virus as anyone alive; he was part of the team that investigated the first recorded outbreak, also in Zaïre, two decades ago. Now he is leading the international brigade that has come to the city of Kikwit to battle the new emergency. "The virus is still loose, and it's spreading," he says. "If the mattress is warm and damp, and people go in and sleep on it, we're going to be in trouble." The villagers are terrified, and resigned. "It's useless for us to do anything," says a neighbor, Mbangu Fioti. "What can we do against this disease?"

For a while last week it looked as though the outbreak might soon be brought under control. The plague police—medical teams dispatched by WHO in Geneva, the Centers for Disease Control and Prevention (CDC) in Atlanta and other public health groups—had set up an effective isolation ward at the main hospital in Kikwit, where the first case had been identified. Belgium's Doctors Without Borders (Médecins Sans Frontières, or MSF) rushed in loads of gloves, gowns, masks and other essential equipment to restore hygiene to filthy clinics. But when the strike forces, aided by local medical students, fanned out through the countryside around Kikwit, trying to follow the path of the fever, it became clear that the danger was far from past.

The teams' job was to figure out who might have been infected already and to warn people at risk. At first doctors thought the victims could all be traced back to a 36-year-old lab technician named Kimfumu, who died at Kikwit's main hos-

Photographs for TIME by Patrick Robert—SYGMA

SORROWFUL SCENES In Ward 3 at Kikwit's main hospital, workers trying to save the sick sometimes leave the dead lying on the floor; a funeral procession draws tears; the people who must handle the bodies are now shunned by neighbors

pital last month. But once they discovered the case of the woman infected even earlier at the Kikwit 2 maternity hospital, they realized the crisis was worse than they had imagined. "It's a huge epidemic," Heyman says of the previously unrecorded cases, "and it's got nothing to do with the main hospital." By week's end WHO doctors had counted 97 Ebola deaths, and the toll seemed certain to rise much higher. The only good news was that the disease had not yet spread—as far as anyone could tell—to the 4 million people of Kinshasa, 250 miles to the west.

When the doctors descended on Kikwit 2, the only hint of hygiene was a torn

GRAVES AND GRIEF A crowd gathers for a burial, and another makeshift wooden cross rises; the virus has killed at least 79

garbage bag on the rusting operating table that clearly had not been changed for months. There were no lights, no running water; health workers collected rainwater from a cistern or went down to the river with buckets. Conditions were perfect for breeding a plague.

And there is more bad news. Since Kimfumu perished a month ago, no one has dared enter the thatched-roof hut where he lived. Mute children and frightened neighbors stare at the stick fence and whisper, as medical students arrive to search for the dead man's family and friends. Where is the cure, a man named Mola asks. A student explains that there is no cure; the only hope is prevention, staying away from the sick, not touching the body. Mola frowns. "I don't know what to say," he says. His father has just died from the virus. "I am the one who helped him. I have already touched the body. And now you tell me I must avoid contact?"

Mola confirms a grim fact about how the disease has spread. Though the Ebola virus is not easily transmitted—it is passed by contact with blood and body fluids—Zaïrian custom requires that preparation of a body for burial must include the handling of various organs. Health officials had hoped only family members were involved in the burial; from Mola and others they learn that friends help as well, which means even more people are in peril than the doctors had realized. "We are telling people of the enormous risks involved in doing this, and offering a safe and respectful form of burial with the aid of the Red Cross," says WHO spokesman Thomson Prentice. When the family insists on a traditional burial, he adds, "we are trying to tell families how to do so at the lowest possible risk. But it's really a tough fight."

AS DEDICATED AS THE RELIEF EFFORT has been, Heyman realizes that it is not enough. He consults with local officials and orders that the teams of students tracking down possible victims be doubled. He wants bicycles, so the teams can travel more quickly, and more gowns, more rubber gloves, more masks to help protect families of the sick and workers in local clinics. He continually quizzes the students, to make sure they are asking the right questions and searching for the right clues.

He knows how hard their job is; their own friends and families are shunning them. "Even the taxis will not take us," says a pretty third-year student named Isabelle Lumbwe, 23. "Our friends say we should be quarantined." But the students are undaunted. "This is going to be our work," she says. "What kind of soldier are you if you flee the battle?"

The problem, the medical teams realize, is that since all the early cases were centered in hospitals, people are afraid to go to them. Officials try to spread the word that the main hospital, at least, is cleaner now, with better staff, supplies and hygiene. But whether out of fear or custom, the sick prefer to go home to die.

Relief workers are finding eight, nine people living under the same roof with a potential Ebola patient. So teams of local workers fan out through the towns with bullhorns, describing symptoms, advising people of the risks and preparing pamphlets with pictures—designed for those who can't read—about how to care for the sick without catching the virus. The personnel are quickly engulfed by huge crowds of people desperate for information and reassurance.

Meanwhile, at the main hospital, a group of low, tin-roofed buildings painted sky blue in the center of town, Dr. Pierre Rollin, chief of the CDC's pathogenesis section, has restored some semblance of order since patients and workers fled the catastrophe. "When we arrived," he says, "it was very bad. People were vomiting; there was diarrhea and blood all over the floors and walls. The dead were lying among the

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WHO MURDERED AFRICA

*by William Campbell Douglass, M.D.**

There is no question mark after the title of this article because the title is not a question. It's a declarative statement. WHO, the World Health Organization, murdered Africa with the AIDS virus. That's a provocative statement, isn't it?

The answers to this little mystery, *Murder on the WHO Express,* will be quite clear to you by the end of this report. You will also understand why the other suspects, the homosexuals, the green monkey and the Haitians, were only pawns in this virocidal attack on the non-Communist world.

If you believe the government propaganda that AIDS is hard to catch *then you are going to die even sooner than the rest of us.* The common cold is a virus. Have you ever had a cold? How did you catch it? You don't *really* know, do you! If the cold virus were fatal how many people would there be left in the world?

Yellow fever is a virus. You catch it from mosquito bites. Malaria is a parasite also carried by mosquitoes. It is many times larger than the AIDS virus (like comparing a pinhead to a moose head) yet the mosquito easily carries this large organism to man.

The tuberculosis germ, also much larger than the AIDS virus, can be transmitted by fomites (inanimate objects such as towels). *The AIDS virus can live for as long as 10 days on a dry plate.*

You can't understand this murder mystery unless you learn a little virology.

Many viruses grow in animals and many grow in humans, but most of the viruses that affect animals don't affect humans. There are exceptions, of course, such as yellow fever and small pox.

There are some viruses in animals that cause very lethal cancer in those animals, but do not affect man or other animals. The bovine leukemia virus (BLV), for example, is lethal to cows but not humans. There is another virus that occurs in sheep called sheep visna virus which is also non-reactive in man. These deadly viruses are "retro viruses" meaning that they can change the genetic composition of cells that they enter.

The World Health Organization, in published articles, called for scientists to work with these deadly agents *and attempt to make a hybrid virus that would be deadly to humans*[1]: "An attempt should be made to see if viruses can in fact exert selective effects on immune function. The possibility should be looked into that the immune response to the virus itself may be impaired if the infecting virus damages, more or less selectively, the cell responding to the virus."

That's AIDS. What the WHO is saying in plain English is "Let's cook up a virus that selectively destroys the T-cell system of man, an acquired immune deficiency."

Why would anyone want to do this? If you destroy the T-cell system of man you destroy man. Is it even remotely possible that the World Health Organization would want to develop a virus that *would wipe out the human race?*

Dr. William Campbell Douglass, M.D., Director, Douglass Center for Nutrition & Preventive Medicine (see pg 2).

If their new virus creation worked, the WHO stated, then many terrible and fatal infectious viruses could be made even *more* terrible and *more* malignant. Does this strike you as being a peculiar goal for a health organization?

Sometimes Americans believe in conspiracies and sometimes they don't. Was there a conspiracy to kill President Kennedy? Twenty-five years later the debate still continues, and people keep changing their minds. One day it's yes, the next day it's no—depending on what was served for breakfast or how the stock market did the day before.

But it doesn't take a bad breakfast to see an amazing concatenation of events involving Russian and Chinese communist nationals, the World Health Organization, the National Cancer Institute and the AIDS pandemic.

But what about the green monkey? Some of the best virologists in the world and many of those directly involved in

(continued page 2)

**Reprinted from September 1987 issue, THE CUTTING EDGE, Dr. William Campbell Douglass, Publisher. For a continuing update on the AIDS epidemic subscribe to, "The Cutting Edge", $49/yr—$36 CRC members: 2470 Windy Hill Rd, Suite 440, Marietta, GA 30067. (Courtesy, "Health Freedom News")*

[1] *Allison, et al.* Bull. WHO *1972, 47:257-63 and Amos, et al.* Fed. Proc. *1972, 31:1087.*

WHO (continued)

AIDS research, such as Robert Gallo and Luc Montagnier, have said that the green monkey may be the culprit. You know the story: A green monkey bit a native on the ass and, bam—AIDS all over central Africa.

There is a fatal flaw here. It is very strange. Because Gallo, Montagnier and these other virologists know that the AIDS virus doesn't occur naturally in monkeys. *In fact it doesn't occur naturally in any animal.*

AIDS started practically *simultaneously* in the United States, Haiti, Brazil, and Central Africa. (Was the green monkey a jet pilot?) Examination of the gene structure of the green monkey cells proves that *it is not genetically possible* to transfer the AIDS virus from monkeys to man by natural means.

Because of the artificial nature of the AIDS virus it will not easily transfer from man to man until it has become very concentrated in the body fluids through repeated injections from person to person, such as drug addicts, and through high multiple partner sexual activity such as takes place in Africa and among homosexuals. After repeated transfer it can become a "natural" infection for man, which it has.

Dr. Theodore Strecker's research of the literature indicates that the National Cancer Institute in collaboration with the World Health Organization made the AIDS virus in their laboratories at Fort Detrick (now NCI). They combined the deadly retroviruses, bovine leukemia virus and sheep visna virus, and injected them into human tissue cultures. The result was the AIDS virus, the first human retrovirus known to man and now believed to be 100 percent fatal to those infected.

The momentous plague that we now face was anticipated by the National Academy of Sciences (NAS) in 1974 when they recommended that "Scientists throughout the world join with the members of this committee in voluntarily deferring experiments (linking) animal viruses."

What the NAS is saying in carefully guarded English is: "For God's sake, stop this madness!"

The green monkey is off the hook. How about the Communists?

Communists are in the process of *conducting germ warfare* from Fort Detrick, Maryland against the free world, especially the United States, *even using foreign communist agents within the United States Army's germ warfare unit* euphamistically called the Army Infectious Disease Unit.

You don't believe it? Carlton Gajdusek, an NIH bigshot at Detrick *admits it:* "IN THE FACILITY I HAVE A BUILDING WHERE MORE GOOD AND LOYAL COMMUNIST SCIENTISTS FROM THE USSR AND MAINLAND CHINA WORK, WITH FULL PASSKEYS TO ALL THE LABORATORIES, THAN THERE ARE AMERICAN. EVEN THE ARMY'S INFECTIOUS DISEASE UNIT IS LOADED WITH FOREIGN WORKERS NOT ALWAYS FRIENDLY NATIONALS."[2]

Can you imagine that? A UN-WHO communist trojan horse in *our* biological warfare center with the full blessing of the U.S. government?

The creation of the AIDS virus by the WHO was not just a diabolical scientific exercise that got out of hand. It was a cold-blooded successful attempt to create a killer virus which was then used in a successful experiment in Africa. So successful in fact that most of central Africa may be wiped out, *75,000,000 dead* within 3-5 years.

It was not an accident. It was deliberate. In the Federation Proceedings of the United States in 1972, WHO said: "In the relation to the immune response *a number of useful experimental approaches can be visualized.*" They suggested that a neat way to do this would be to put their new killer virus (AIDS) into a vaccination program, sit back and observe the results. "This would be particularly informative in sibships," they said. That is, give the AIDS virus to brothers and sisters and see if they die, who dies first, and of what, just like using rats in a laboratory.

They used smallpox vaccine for their vehicle and the geographical sites chosen in 1972 were Uganda and other African states, Haiti, Brazil and Japan. The present or recent past of AIDS epidemiology coincides with these geographical areas.

Dr. Strecker points out that even if the African green monkey could transmit AIDS to humans, the present known amount of infections in Africa makes it *statistically impossible* for a single episode, such as a monkey biting someone, to have brought this epidemic to this point. The doubling time of the number of people infected, about every 14 months, when correlated with the first known case, and the present known number of cases, prove beyond a doubt that *a large number of people had to have been infected at the same time.* Starting in 1972 with the first case from our mythical monkey and doubling the number infected from that single source every 14 months you get only a few thousand cases. From 1972 to 1987 is 15 years or 180 months. If it takes 14 months to double the number of cases then there would have been 13 doublings, 1 then 2, then 4, then 8, etc. In 15 years, *from a single source of infection* there would be about 8,000 cases in Africa, *not 75 million AIDS infected people.* We are approaching World War II mortality statistics here—without a shot being fired.

Dr. Theodore A. Strecker is the courageous doctor who unraveled this conundrum, the greatest murder mystery of all time. He should get the Nobel prize but he'll be lucky not to get "suicided." ("Prominent California doctor ties his hands behind his back, hangs himself, and jumps from 20th floor. There was no evidence of foul play.")

Strecker was employed as a consultant to work on a health proposal for Security Pacific Bank. He was to estimate the cost of their health care for the future. Should they form a health maintenance organization? (HMO) was a major issue. After investigating the current medical market he advised against

William Campbell Douglass, M.D. is one of the nation's leading authorities on health and medicine. His provocative and entertaining lectures on Nutrition, The Drugging of America, New Horizons in Wholistic Medicine, Pre-menstrual Syndrome, Chelation Therapy, and his book, The Milk of Human Kindness, *have been presented all over the United States.*

Dr. Douglass has four books to his credit. His latest, The Milk of Human Kindness is Not Pasteurized, *is unique in the field of nutrition. He is former Florida State President of the American College of Emergency Physicians and former editor of the Journal of the Sarasota County Medical Society. He has been practicing nutritional medicine for 10 years and has extensive experience lecturing and discussing orthomolecular medicine on radio and television.*

[2]Omni Magazine, *March, 1986, p. 106.*

WHO (continued)
the HMO because he found that the AIDS epidemic will in all probability *bankrupt the nation's medical system.*

He became fascinated with all the peculiar scientific anomalies concerning AIDS that kept cropping up. Why did the "experts" keep talking about green monkeys and homosexuals being the culprits when it was obvious that the AIDS virus was a man-made virus? Why did they say that it was a homosexual and drug-user disease when in Africa it was obviously a heterosexual disease? If the green monkey did it, then why did AIDS explode practically simultaneously in Africa, Haiti, Brazil, the United States and southern Japan?

Why, when it was proposed to the National Institute of Health that the AIDS virus was a combination of two bovine or sheep viruses cultured in human cells in a laboratory, did they say it was "bad science" when that's exactly what occurred?

As early as 1970 the World Health Organization was growing these deadly animal viruses in human tissue cultures. Cedric Mims, in 1981, said in a published article that there was a bovine virus contaminating the culture media of the WHO. Was this an accident or a "non-accident"? If it was an accident why did WHO continue to use the vaccine?

This viral and genetic death bomb, AIDS, was finally produced in 1974. It was given to monkeys and they died of pneumocystis carni which is typical of AIDS.

Dr. R. J. Biggar said in *Lancet.*[3] "... The AIDS agent . . . could not have originated de novo." That means in plain English that it didn't come out of thin air. AIDS was engineered in a laboratory by virologists. It couldn't engineer itself. As Doctor Strecker so colorfully puts it: "If a person has no arms or legs and shows up at a party in a tuxedo, how did he get dressed? Somebody dressed him."

There are *9,000 to the fourth power* possible AIDS viruses. (There are 9,000 base pairs on the geneome.) So the fun has just begun. Some will cause brain rot similar to the sheep virus, some leukemia-like diseases from the cow virus and some that won't do anything. So the virus will be constantly changing and trying out new esoteric diseases on hapless man. We're only at the beginning.

Because of the trillions of possible genetic combinations there will never be a vaccine. Even if they could develop a vaccine they would undoubtedly give us something equally bad as they did with the polio vaccine (cancer of the brain), the swine flu vaccine (a polio-like disease), the smallpox vaccine (AIDS), and the hepatitis vaccine (AIDS).

There are precedents. *This is not the first time* the virologists have brought us disaster. SV-40 virus from monkey cell cultures contaminated polio cultures. Most people in their 40's are now carrying this virus through contaminated polio innoculations given in the early 60's. It is known to cause brain cancer which explains the increase in this disease that we have seen in the past ten years.

This is the origin of the green monkey theory. The polio vaccine was grown on green monkey kidney cells. Sixty-four million Americans were vaccinated with SV-40-contaminated vaccine in the 60's. An increase in cancer of the brain, possibly multiple sclerosis, and God only knows what else is the tragic result. The delay between vaccination and the onset of cancer with this virus is as long as 20-30 years. 1965 plus 20 equals 1985. Get the picture?

The final piece of the puzzle is how AIDS devastated the homosexual population in the United States. It wasn't from smallpox vaccination as in Africa because we don't do that any more. There is no smallpox in the United States and so vaccination was discontinued.

Although some AIDS has been brought to the United States from Haiti by homosexuals, it would not be enough to explain the explosion of AIDS that occurred simultaneously with the African and Haitian epidemics.

The AIDS virus didn't exist in the United States before 1978. You can check back in any hospital and no stored blood samples can be found anywhere that exhibit the AIDS virus before that date.

What happened in 1978 and beyond to cause AIDS to burst upon the scene and devastate the homosexual segment of our population? It was the introduction of the hepatitis B vaccine *which exhibits the exact epidemiology of AIDS.*

A Doctor W. Schmugner, born in Poland and educated in Russia, came to this country in 1969. *Schmugner's immigration to the U.S. was probably the most fateful immigration in our history.* He, by unexplained process, became head of the New York City blood bank. (How does a *Russian trained* doctor become head of one of the largest blood banks in the world? Doesn't that strike you as peculiar?)

He set up the rules for the hepatitis vaccine studies. Only males between the ages of 20 and 40, *who were not monogamous,* would be allowed to participate in this study. Can you think of any reason for insisting that all experimentees be promiscuous? Maybe you don't believe in the communist conspiracy theory but give me some other logical explanation. Schmugner is now dead and his diabolical secret went with him.

The Centers for Disease Control reported in 1981 that four percent of those receiving the hepatitis-vaccine were AIDS-infected. In 1984 they admitted to 60 percent. Now they refuse to give out figures at all because they don't want to admit that *100 percent of hepatitis vaccine receivers are infected with AIDS.*

Where is the data on the hepatitis vaccine studies? FDA? CDC? No, the *U.S. Department of Justice* has it buried where you will never see it.

What has the government told us about AIDS?

- It's a homosexual disease—wrong. (The homosexuals certainly spread it but the primary responsibility wasn't theirs.)
- It's related to anal intercourse only—wrong.
- Only a small percentage of those testing positive for AIDS would get the disease—wrong.
- It came from the African green monkey—wrong.
- It came from the cytomegalovirus—wrong.
- It was due to popping amyl nitrate with sex—wrong.
- It was started 400 years ago by the Portugese—wrong. (It started in 1972.)
- You can't get it from insects—wrong.
- The virus can't live outside the body—wrong.

The head of the Human leukemia Research Group at Harvard is a veterinarian. Dr. O. W. Judd, International Agency for Research on Cancer, the agency that requested the production of the virus in the first place, is also a veterinarian. The leukemia research he is conducting is being done under the auspices of a school of veterinary medicine.

[3] *Jan. 11, 1986.*

WHO (continued)

Now there is nothing wrong with being a vet but, as we have pointed out, the AIDS virus is a human virus. You can't test these viruses in animals and you can't test leukemias in them either. It doesn't work. So why would your government give Judd, a veterinarian, eight and one-half million dollars to study leukemia in a veterinary college? As long as we are being used as experimental animals maybe it's appropriate.

The *London Times* should be congratulated for uncovering the smallpox-AIDS connection.[4] But their expose was very misleading. The article states that the African AIDS epidemic was caused by the smallpox vaccine "triggering" AIDS in those vaccinated.

Dr. Robert Gello, who has been mixed up in some very strange scientific snafus, supports this theory. Whether the infection of 75 million Africans was deliberate or accidental can be debated but there is no room for debate about whether the smallpox shots "awakened the unsuspected virus infection." There is *absolutely no scientific evidence* that this laboratory-engineered virus was present in Africa before the World Health Organization descended upon these hapless people in 1967 with their deadly AIDS-laced vaccine. The AIDS virus didn't come from Africa. It came from Fort Detrick, Maryland, U.S.A.

The situation is extremely desperate and the medical profession is too frightened and cowed (as usual) to take any action. Dr. Strecker attempted to mobilize the doctors through some of the most respected medical journals in the world. The prestigious *Annals of Internal Medicine* said that his material "appears to be entirely concerned with matters of virology" and so try some other publication.

In his letter to *The Annals* Strecker said, "If correct human experimental procedures had been followed we would not find half of the world stumbling off on the wrong path to the cure for AIDS with the other half of the world covering up the origination of the damned disease. It appears to me that your *Annals of Internal Medicine* is participating in the greatest fraud ever perpetrated."

I guess they didn't like that so Strecker submitted his sensational and mind-boggling letter with all of the proper documentation to the British journal, *Lancet*.

Their reply: "Thank you for that interesting letter on AIDS. I am sorry to have to report that we will not be able to publish it. We have no criticism" but their letter section was "overcrowded with submissions."

They're too crowded to announce the end of western civilization and possibly all mankind? It doesn't seem reasonable.

What can we do?

The first thing that should be done is *close down all laboratories in this country that are dealing with these deadly retroviruses.*

Then we must sort out the insane, irresponsible and traiterous scientists involved in these experiments and try them for murder. Then maybe, just maybe, we can re-staff the laboratories with loyal Americans who will work to save a remnant of people to repopulate and re-civilize the world.

*London Times. Front page, May 11, 1987.

(From the Front Page of THE TIMES, London, 11 May 1987

SMALLPOX VACCINE 'TRIGGERED AIDS VIRUS'

by Pearce Wright, Science Editor

The Aids epidemic may have been triggered by the mass vaccination campaign which eradicated smallpox.

The World Health Organization, which masterminded the 13-year campaign, is studying new scientific evidence suggesting that immunization with the smallpox vaccine *Vaccinia* awakened the unsuspected, dormant human immuno defence virus infection (HIV).

Some experts fear that in obliterating one disease, another disease was transformed from a minor endemic illness of the Third World into the current pandemic.

While doctors now accept that *Vaccinia* can activate other viruses, they are divided about whether it was the main catalyst to the Aids epidemic.

But an adviser to WHO who disclosed the problem, told *The Times:* "I thought it was just a coincidence until we studied the latest findings about the reactions which can be caused by *Vaccinia.* Now I believe the smallpox vaccine theory is the explanation to the explosion of Aids."

Further evidence comes from the Walter Reed Army Medical Centre in Washington.

While smallpox vaccine is no longer kept for public health purposes, new recruits to the American armed services are immunized as a precaution against possible biological warfare. Routine vaccination of a 19-year-old recruit was the trigger for stimulation of dormant HIV virus into Aids.

This discovery of how people with subclinical HIV infection are at risk of rapid development of Aids as a vaccine-induced disease was made by a medical team working with Dr. Robert Redfield at Walter Reed.

The recruit who developed Aids after vaccination had been healthy throughout high school. He was given multiple immunizations, followed by his first smallpox vaccination.

Two and a half weeks later he developed fever, headaches, neck stiffness and night sweats. Three weeks later he was admitted to Walter Reed suffering from meningitis and rapidly developed further symptoms of Aids and died after responding for a short time to treatment.

There was no evidence that the recruit had been involved in any homosexual activity.

In describing their discovery in a paper published in the *New England Journal of Medicine* a fortnight ago, the Walter Reed team gave a warning against a plan to use modified versions of the smallpox vaccine to combat other diseases in developing countries.

Other doctors who accept the connection between the anti-smallpox campaign and the Aids epidemic now see answers to questions which had baffled them. How, for instance, the Aids organism, previously regarded by scientists as "weak, slow and vulnerable", began to behave like a type capable of creating a plague.

Many experts are reluctant to support the theory publicly because they believe it would be interpreted unfairly as criticism of WHO.

(continued page 5)

TIMES (continued)

In addition, they are concerned about the impact on other public health campaigns with vaccines, such as against diptheria and the continued use of *Vaccinia* in potential Aids research.

The coincidence between the anti-smallpox campaign and the rise of Aids was discussed privately last year by experts at WHO. The possibility was dismissed on grounds of unsatisfactory evidence.

Advisors to the organization believed then that too much attention was being focused on Aids by the media. It is now felt that doubts would have risen sooner if public health authorities in Africa had more willingly reported infection statistics to WHO.

Instead, some African countries continued to ignore the existence of Aids even after US doctors alerted the world when the infection spread to the United States.

However, as epidemiologists gleaned more information about Aids from reluctant Central African countries, clues began to emerge from the new findings when examined against the wealth of detail known about smallpox as recorded in the *Final Report of the Global Commission for the Certification of Smallpox Eradication.*

The smallpox vaccine theory would account for the position of each of the seven Central African states which top the league table of most-affected countries; why Brazil became the most afflicted Latin American country; and how Haiti became the route for the spread of Aids to the US.

It also provides an explanation of how the infection was spread more evenly between males and females in Africa than in the West and why there is less sign of infection among five to 11-year-olds in Central Africa.

Although no detailed figures are available, WHO information indicated that the Aids league table of Central Africa matches the concentration of vaccinations.

The greatest spread of HIV infection coincides with the most intense immunization programmes, with the number of people immunized being as follows:

Zaire 36,878,000; Zambia 19,060,000; Tanzania 14,972,000; Uganda 11,616,000; Malawai 8,118,000; Ruanda 3,382,000 and Burundi 3,274,000.

Brazil, the only South American country covered in the eradication campaign, has the highest incidence of Aids in that region.

About 14,000 Haitians, on United Nations secondment to Central Africa, were covered in the campaign. They began to return home at a time when Haiti had become a popular playground for San Francisco homosexuals.

Dr. Robert Gello, who first identified the Aids virus in the US, told *The Times:* "The link between the WHO programme and the epidemic in Africa is an interesting and important hypothesis. "I cannot say that it actually happened, but I have been saying for some years that the use of live vaccines such as that used fo smallpox can activate a dormant infectio such as HIV.

"No blame can be attached t WHO, but if the hypothesis is correct i is a tragic situation and a warning th we cannot ignore."

Charity and health workers a convinced that millions of new Aic cases are about to hit southern Africa

After a meeting of 50 experts ne Geneva this month it was revealed tha up to 75 million, one third of th population, could have the disease withi the next five years.

Some organizations which hav closely studied Africa, such as War o Want, believe that South Africa's blac population, so far largely protected fro the disease, could be most affected migrant workers bring it into the countr from the worst hit areas further north

* * * * *

Aids was first officially reporte from San Francisco in 1981 and it w about two years later before Centr African states were implicated. It is no known that these states had become reservoir of Aids as long ago as the la 1970s.

* * * * *

Although detailed figures of Aid cases in Africa are difficult to collec the more than two million carriers, an 50,000 deaths, estimated by the Worl Health Organization are concentrate in the countries where the smallpo immunization programme was mos intensive.

1987 HIGHLIGHTS OF ACTIVITIES

Committee to Restore the Constitution, Inc.

JANUARY

Bulletin
"Dead Man Fuzing—The Real Meaning of the Reykjavic Summit"
"Spectatorship vs Participation"
"County Ordinance to Repeal Federal Reserve"

FEBRUARY

Bulletin
"Challenging the New Barbarians"

MARCH

Bulletin
"Why a Constitutional Convention"
"Plotting to Rewrite the U.S. Constitution"
"Norman Dodd"
"1986 Operational Highlights, CRC"

2 **Annual Meeting**
Committee to Restore the Constitution, Inc.

3 **Direct Mail Mission**
"Dear Publisher" letter with "Facts" ad to 2,38 media and organizations

4 **Radio Mission**
Taffy McCallum Show, WINZ, Miami, Florida
4430 NW 207th Drive—Roberts live—2 hours

9 'Notice Letter' w/envelope mailed to 26 CRC voti members who failed to return voting proxi

11 **Direct Mail Mission**
Fund-raising FESPA letter, w/enclosures, mailed t 19,612 foundations

19 Roberts meeting with Raymond White, book publishe Australia—re "Victory Denied"

27 **Direct Mail Mission**
"Dear Publisher" letter with "Facts" ad & Janua CRC bulletin to 3,311 weekly newspapers

APRIL

Bulletin
"Americans Are An Endangered Species"
"The Safety of the State Begins With You"
"Law of Principal"
"The Hegalian Principle"
"County Ordinance to Repeal Federal Reserve"

14 Second Reprint
"The Most Secret Science", 5,000 copies

24 Direct Mail Mission
"Dear Farm/Ranch Organization President" letter with "Facts" ad & county ordinance to 3,528 farm/ranch organizations

30 Direct Mail Mission
"Most Secret Science" flyer w/business reply envelope to 5,000 American Patriotic Donor list

MAY

Bulletin
"Dear County Commissioner" letter w/text personal letter for member action
"County Government"
"Constitutional Authority"

4 Direct Mail Mission
"Most Secret Science" brochure w/business reply envelope to 5,000 doctors at home address

5 Direct Mail Mission
"Farm/Ranch" letter with "Facts" ad mailed to 3,558 organization presidents

7 Direct Mail Mission
"Most Secret Science" brochure mailed to 5,503 Antony Sutton bookbuyers list

8 Direct Mail Mission
"Dear County Commissioner" letter with "Silent Revolution" 4-page brochure to 13,239 county commissioners
"Dear State Senator" and "Dear State Representative" letters with "Silent Revolution"
4-page brochure to 7,481 state lawmakers

22 Radio Mission
Anthony Hilder Show, KCZN Radio, Oxnard, California—Roberts live—1 hour

23 Radio Mission
Leona & Frank Deisz, KURL Radio, Billings, Montana PO Box 962, 59103—Roberts live—1 hour

JUNE

Bulletin
"Reece Committee Revisited—Dodd Report to the American People on Tax Exempt Foundations"
"Penalty for Constitutional Ignorance is a Police State Society"
"The Symptoms of Revolution"
"How to Extend the Power and Expand the Scope of Your Patriotic Efforts"

11 Direct Mail Mission
May CRC bulletin & "Harness the Power of County Government" letter to 9,092 McAlvany Advisory list

JULY

Bulletin
"Nevada Lawmakers Challenge Constitutional Convention"
"Withdrawal Request for Constitutional Convention", Nevada Assembly Joint Resolution #25
"Resolution Expunging Nevada Call for Federal Constitutional Convention"
"Balancing the Federal Budget Requires Enforcement of the Constitution, Not Amendment of It"
"A Bill Finding that the Issuance of Federal Reserve Notes is a Violation of the Constitution"

6 Radio Mission
Anthony Hilder Show, KSTR Radio, Ventura, California, Roberts live—1½ hours

7 Radio Mission
Wes Templeton Show, "Your Opinion Counts", WKIZ, Bonita Springs, Florida, PO Box 1536—Roberts live—2 hours

10 Direct Mail Mission
"Harness the Power of County/State Governments" letter w/May CRC bulletin to 4,865 L. Patterson list

AUGUST

Bulletin
"Personal Survival" letter & "Montana Lawmakers Defeat Constitutional Convention Resolution", w/May CRC bulletin (1984)

4 Direct Mail Mission
FESPA fund raising letter w/June 1987 CRC bulletin to 11,228 'living donor' foundation list

9 Conference with Jeffrey Smith, New York media

26 Direct Mail Mission
August CRC bulletin w/FESPA flyer to 5,000 Sutton bookbuyers list

SEPTEMBER

Bulletin
"Whose Fingerprints: Colonel North Plot to Suspend Constitution", w/insert, "Here Are Suppressed Facts on the Secret Contingency Plan", & "Meese Suggests States Should Overrule Congress"

OCTOBER

Bulletin
"Supreme Court Confirms Rights of Property Owners"
"Court Order 85-1199"
"Petition to the County Commission" letter with personal Survival letter

14 Radio Mission
Cecil Johnson Show, KCRC Radio, San Bernardino, California—Roberts live—1 hour

NOVEMBER

Bulletin
"Model State Constitution Scheme—A Socialist Governance in Your Future"
"An Act to Establish a State Advisory Commission on Intergovernmental Relations"
"The Montana Ripoff"
"Model State Constitution Convention" insert

DECEMBER

Bulletin
"Vindication of Property Rights"
"Toward a Soviet America"
"A Bill to Provide for Enforcement of the United States Constitution with Regard to Federal Regionalism"
"The Most Secret Science", 2-color flyer insert