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The Wild, Wild Pest.

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A gang of crude yet deadly viruses, on the run from domestic animals, is slaughtering wild species worldwide

IT WAS AS IF THERE WERE A WAR among the lions," says Melody E. Roelke-Parker of the events on the Serengeti Plain in the early months of 1994. "I saw lions who had been killed by other lions, lions with crushed skulls and hideous infected wounds." Roelke-Parker had been in Tanzania for barely a year at that point, hired by a Swiss foundation to set up veterinary services for the Tanzanian national parks. She knew that lions kill each other on occasion, when defending territory, or when males take over other prides and slaughter the cubs and juveniles, thereby bringing the females back into heat. But she had "an ill feeling" about these deaths. Many of the lions killed that year were mature, but they weren't overcrowded enough to suggest territorial disputes.

Then one day Roelke-Parker got a call from a friend who worked as a balloon pilot. He had been flying a sight-seeing couple around, he said, when they spotted a lion that suddenly fell over and began to thrash its limbs senselessly. Roelke-Parker tracked down the sightseers on their way to the next lodge. There, in the dust and exhaust, she watched the lion's recorded image on their video camera: the lion had suffered a grand mal seizure.

Later, she heard more eyewitness accounts, including one that explained the attacks: Two lions were walking together on the plain when one began to twitch and grimace so strangely that the other attacked him. The afflicted lion could neither defend himself nor escape.

That was the sixth year of what turned out to be a wildlife plague of global proportions. Between January and October of 1994, wildlife biologists estimate, more than a third of all the lions in the Serengeti and the neighboring Masai Mara Plain died of a disease they weren't supposed to get: canine distemper. And the lions weren't the only local animals to suffer. Hyenas and bat-eared foxes also died, and investigators suspect that wild dogs in the region were previously

decimated by the disease, as well as by rabies. A few years earlier, tens of thousands of seals, dolphins and porpoises died from Russia to the Gulf of Mexico. All those losses have been attributed to outbreaks of viruses of the same genus: Morbillivirus.

IN THE PAST DECADE MORBILLIVIRUSES have challenged the received wisdom and accepted policy concerning wildlife diseases. The sheer number of deaths seems to cry out for dramatic measures: the lions should be treated, the hyenas vaccinated, the disease contained just like any human epidemic. But since the victims are wild animals, it is unclear whether anything can or should be done. No one knows much about the diseases that afflict wild populations, and conservation biologists have traditionally taken a hands-off approach. Pathogens and parasites are natural parts of an animal's environment, they reason; disease may even help control some populations. Although an epizootic--as an epidemic among animals is called--may claim thousands of victims, it rarely kills off every member of a species.

Morbillivirus may be the plague to turn such laissez-faire attitudes. For the animals involved--from the lions in Tanzania to the seals in Russia--were not infected by their own kind or even by diseases that are common in the wild. Their distemper came from domesticated animals. Moreover, the conditions that made them susceptible to canine distemper--pollution, overfishing, habitat loss--were hardly what One would call "natural." As the distinctions blur between wild and domestic, savanna and farm, biologists and veterinarians are beginning to ask themselves, When exactly is a disease worth containing?

VETERINARY SCIENCE OWES ITS beginnings to economics rather than compassion. The first veterinary school, founded in Lyons, France, in 1762, was established not to prevent anima] suffering, but to protect the investments of French farmers, whose cattle were being killed by yet another Morbillivirus. The disease it caused, known as rinderpest, was so lethal that its victims sometimes died overnight, before they had time to develop mouth ulcers and diarrhea (the classic symptoms of the disease). The virus originated in southern Asia, slipped into Europe via trade and warfare, and then proceeded to wipe out herds of cattle throughout Europe.

Medical science, of course, was then in its infancy. Although the principle of infection was understood, the agents of infection were not--theorists imagined them as minute insects or "animalcules." Great Britain and France managed to control rinderpest temporarily by quarantining herds or slaughtering sick animals and burning or burying their carcasses. Other countries, notably the Netherlands, tried to save their cattle--and thereby became reservoirs for recurrent infections.

Farmers had to wait until the 1930s before an effective vaccine became available against rinderpest, and it eradicated the virus from Europe. By that time, unfortunately, the virus had escaped into animal populations that people took no

interest in protecting. In 1889, for instance, when the Italian army used oxen to drag ordnance into Ethiopia, rinderpest hitched a ride. Rinderpest is not fastidious in its choice of hosts: any beast with cloven hooves will do. Gazelles, eland, kudu, buffalo, giraffes and even warthogs were fair game, and rinderpest infected them all. In less than ten years the virus raced like brush fire across 3,500 miles of landscape and completely rearranged the map of African wildlife. In southern Africa, Morbillivirus infected every available victim, leaving only a few immune survivors before dying out.

In the late 1930s, to protect southern Africa from the virus, a fence was erected between Lake Tanganyika and Lake Malawi, and guards were ordered to shoot all game that came within twenty-five miles of it. Still, pockets of rinderpest persist to this day in East and West Africa. Every ten years or so, the virus jumps from a resistant host species to a new generation of susceptible animals. Whenever it runs out of wild species it simply returns to cattle.

Morbillivirus is the kudzu, the zebra mussel, of the microbial world: adaptable, opportunistic and voracious. None of the seven or so species is particularly clever, as viruses go. Simple spheres of lipids and protein with RNA inside, they hardly differ one from the other: a test for distemper will come back positive in the presence of any Morbillivirus. Immunity, if achieved before an animal succumbs, is complete--morbilliviruses are not good at evasion. But morbilliviruses never want for new victims. Camine distemper virus goes even farther afield than rinderpest virus: besides killing any carnivore--whether dog, ferret or seal--it has killed javelinas (a kind of wild boar) as well as Japanese macaques. In 1994 a related virus made the jump from fruit bats into sixteen Australian racehorses, as well as into the horses' trainers. (No human had ever been known to contract Morbillivirus from an animal before, nor has anyone since.)

Morbillivirus doesn't care what kind of cell it enters. If a dog sniffs an infected raccoon's nose, the virus will begin to grow in the dog's nose and lungs, causing rhinitis and pneumonia. If a gazelle eats a blade of grass that has been licked by an infected buffalo, the gazelle's mouth and intestines will ulcerate and slough. Once inside a body, Morbillivirus also attacks immune cells known as lymphocytes, opening the gates to secondary bacterial and fungal infections. Many morbilliviruses can target nerve cells, and so even if an animal survives the initial onslaught, it will suffer from trembling limbs and crippling seizures for the rest of its abbreviated life.

MORBILLIVIRUSES NO LONGER pose much of a threat to domestic animals. Thanks to effective vaccines, few dogs or cattle in wealthy countries ever get the disease. The Food and Agriculture Organization of the United Nations (FAO) even maintains that rinderpest could be eradicated from cattle much as smallpox was eradicated from people in the 1960s and 1970s, and the organization has a detailed plan for doing so.

FAO's boast may never be put to the test--public apathy could see to that. At any rate, protecting wild animals is a far more formidable task than protecting cattle. Wild animals don't hold still for treatment or diagnosis, and neither do their diseases. When alive, the animals are hard to touch, much less sample; when they die, their carcasses are quickly scavenged, and any disease evidence disappears with them.

Transfer those obstacles to the sea, and the difficulties for investigators grow exponentially. "Sick seals only come ashore when things are really starting to go wrong, not just at the first sign of disease," says Joseph R. Geraci, a veterinarian and marine biologist with the National Aquarium in Baltimore, Maryland. "A stranded sea] is ill, depressed, dehydrated and showing secondary infections, complicating problems from bacteria and fungi." Under those circumstances, not only is it hard to find samples for testing; it is hard to be sure that what you find in the samples is the real initiator of disease and not just some tagalong.

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IN 1987, IN WHAT AT THE TIME seemed an isolated event, several thousand seals died in Siberia. The victims, living in and along landlocked Lake Baikal, were stricken with diarrhea, eye inflammations and paralysis. Sampling proved that the underlying cause of death was canine distemper: people in the area hunted seals, their dogs were rarely vaccinated, and the virus had simply jumped into another carnivore. Still, though the size of the Lake Baikal epizootic was alarming, it seemed unlikely to spread.

Then, in 1988, a die-off of startling proportions took place. It began quietly off the coast of Denmark, in the Kattegat Strait, with an increase in the number of aborted harbor seal pups. But soon the bodies of dead and dying adults were washing ashore. The phenomenon quickly spread to the shores of Germany, the Netherlands and Great Britain. Both harbor and gray seals became ill, but the harbor seals were the hardest hit: some 80 percent of those infected died. Within a year nearly 18,000 seals were dead, their carcasses burned or buried in an attempt to destroy whatever agent had caused the destruction. The virologist Albert D.M.E. Osterhaus of Erasmus University in Rotterdam, the Netherlands, soon fingered the culprit: a strain of Morbillivirus that proved to be distinct from but closely related to canine distemper. It was named phocine distemper virus, from the Latin word for "seal."

At around the same time, bottlenose dolphins began washing onto the Atlantic coast, of the United States, and harbor porpoises, already endangered, began washing onto the coast of Northern Ireland from the Irish Sea. Both populations were infected by morbilliviruses (though by different strains). The bottlenose dolphins lost as much as half their inshore population in the epizootic. In 1990, yet another previously unknown strain of the virus, similar to the one that struck the bottlenose population, infected striped dolphins off the coast of Catalonia.

Named dolphin morbillivirus, it eventually spread to the coast of Turkey and claimed more than a thousand victims.

If the past is any indication, more epizootics may still be on the way--and marine mammals in the Pacific may be the next targets. Antibodies to dolphin and porpoise morbillivirus have been found already in Hawaiian monk seals and in California sea lions, and phocine distemper virus may have reached the Pacific as well. Many populations could be as vulnerable as the hoofed animals of Africa were in 1889, before rinderpest virus struck.

NEW AS THEY SEEM TO US BIOLOGISTS, most emerging diseases have been around for a while. They simply don't become apparent to us until they discover new routes from one host to another. HIV, for instance, jumped from chimpanzees to humans when new logging roads were built in Africa, enabling the animals to be hunted and sold for meat on a broader scale. Morbilliviruses probably took advantage of similar opportunities. Antibodies to canine distemper have been found in blood and tissue samples from various marine mammals, as well as in archived samples of serum from lions and hyenas in the Serengeti. Although the viruses were new to biologists, in other words, they were not new to the animals.

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Could it be, then, that recent epizootics are part of an old pattern, one that simply passed unnoticed before? No, Geraci says. As early as 1828, the History of British Animals by John Fleming noted that seals "are occasionally subject to epizooty." In the early 1800s, for instance, half a million Cape fur seals died off the coast of Namibia, presumably from a disease. But few of the earlier die-offs were as large as the epizootics of the past ten years, and few took place over so short a period.

"Something is happening," Geraci says, but he can't say whether it results from global warming, shifts in ocean currents or the spread of man-made contaminants. "There is no one hypothesis that has emerged with greater favor." Tentative explanations abound, though, and some of them are relatively reassuring. In the years before the phocine distemper outbreak, for instance, a moratorium was imposed on hunting in the North Sea. As a result, the seal populations grew substantially, and so did their chances of spreading infection. The same forces were operating among the lions of the Serengeti: when a prolonged drought killed off 17,000 buffalo, the lions came together in close, contagious quarters to feed on them. The epizootics, in other words, may have been part of a natural boom-and-bust population cycle.

Yet behind such seemingly natural equations lurk more malignant factors. As seal populations grew, for instance, they had to compete with the ever-increasing human appetite for fish. Overfishing in the North Sea had severely depleted fish

stocks, forcing seals to work harder to survive; the extra work alone stressed them, and the need for wider foraging helped spread the virus further. When seals didn't get enough to eat, their body fat was often mobilized, releasing stored contaminants such as polychlorinated biphenyls (PCBs) into their bloodstreams. Such contaminants inhibited the seals' ability to fight off diseases.

Industrial farming and clear-cutting may also be partly to blame for marine epizootics. As agricultural runoff pumps the oceans full of fertilizer, it triggers vast algal blooms, some of them toxic. When Geraci examined the bottlenose dolphins that died off the Atlantic coast of the United States, he found traces of brevetoxin, one of the organic poisons generated by red tide, which could have helped make the dolphins susceptible to Morbillivirus.

LIKE A CONSPIRACY that spirals up through every level of government, growing more tenuous and entangled the higher it goes, an epizootic can be exceedingly hard to unravel. The events in the Serengeti were a case in point. Once Roelke-Parker decided that the tons had an infectious disease of some sort, she went to work identifying it. She observed more lions having seizures, tracked down carcass after carcass, collected tissue and serum samples from the bodies, and shipped her precious samples to pathology laboratories in Switzerland and the United States.

Then, suddenly, the situation exploded. Craig Packer, a behavioral ecologist from the University of Minnesota in Saint Paul, had studied the lions for fifteen years. Hoping to drum up some help, he sent out a press release: Lions on the Serengeti were dying of a mysterious disease. Soon headlines worldwide were announcing that the lions had AIDS or mad cow disease.

For some Tanzanian officials that was the last straw. The preceding rabies epizootic, they believed, had been abetted by vaccines administered by foreign veterinarians. Now Packer and Roelke-Parker were making local officials look incompetent. Worse, they were scaring off tourists, who might believe they could be infected by the lions. ("I don't know about you," Packer recently told me, "but if I was close enough to a lion to catch AIDS from it, that wouldn't be my biggest worry.") The Tanzanians even threatened to kick Packer and Roelke-Parker out of the country.

In the meantime, Roelke-Parker had received a fax from the wildlife pathologist Linda Munson, then at the University of Tennessee in Knoxville. The lion tissues Roelke-Parker had sent her looked familiar; Munson said. She had seen the same effects a year before in lions, tigers and jaguars in southern California. Those animals had died of canine distemper, which they had probably contracted from raccoons.

The mystery was solved. Ironically, by then the lion epizootic had moved north, toward the Masai Mara, and the crisis was subsiding on its own.

WHAT CAN BE DONE TO PREVENT similar disasters in the future? Should anything be done at all? The safest, smartest course of action, veterinarians seem to agree, is to begin monitoring wildlife diseases, gradually building a baseline of knowledge about each species. But though the public may be interested in such work, at least abstractly, and though the U.S. government assigns departments to carry it out, funding is hard to come by. Often, investigators are caught in a catch-22. In 1990, for instance, the wildlife veterinarian A. Alonso Aguirre of Tufts University in Medford, Massachusetts, wanted to study the increased incidence, worldwide, of certain tumors among green sea turtles, which were already endangered. But when he asked the National Cancer Institute (NCI) about a grant, he was told that the institute did not fund research for cancers in lower vertebrates. And when he went to the National Science Foundation, he was told that the foundation left all cancer research funding to NCI.

Even when veterinarians do secure funding, they are not always sure how best to use it. "Most of conservation came out of the disciplines of ecology and behavioral science, where scientists were traditionally supposed to play the role of observers," says William B. Karesh, a field veterinarian for the Wildlife Conservation Society. "They were not supposed to participate in the outcome of what they saw, as much as to report it." The epizootic in the Serengeti is a good example: until lions started dying, no one gave much thought to their infectious diseases. Although geneticists had been taking blood samples from the animals for years, they never bothered to run serology tests. As for hyenas, bat-eared foxes and wild dogs, the level of scientific neglect has been shocking. No one knows how many of the animals, or what percentage of their populations, were killed by the virus, because no one was studying them when the virus struck. When their bodies were found, they were left unexamined. Indeed, the only assurance that they died of canine distemper comes from the fact that they died at the same time the lions did and are far more susceptible to the disease.

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THE IRONY IS THAT EVEN IF THE Serengeti outbreak had been perfectly monitored, and its victims exhaustively documented, veterinarians might still have let the disease run its course. Vaccines to protect wild populations are not out of the question, but what is safe in a pet can be lethal for a wild animal. In 1971, for instance, veterinarians worried that canine distemper might kill the last remaining black-footed ferrets. But when they captured six members of what was then thought to be the last ferret colony and injected them with a vaccine developed for dogs, four of the six died. Canine distemper vaccine later proved fatal to red pandas and kinkajous as well.

In a few cases safe vaccines have been created and tested but never manufactured: the market for wildlife vaccines is minuscule. Osterhaus has put

together a vaccine against phocine distemper, made from components of canine distemper virus, that has proved safe and effective in harbor seals. Preliminary studies suggest it could protect monk seals in Hawaii as well, but do they really need the protection? Their biological cousins, the Mediterranean monk seals, show signs of immunity to dolphin morbillivirus: though traces of Morbillivirus and antigens of the virus have sometimes been found in their carcasses, they have no lesions or other signs of disease.

Another option for protecting wild animals is to vaccinate domesticated carriers of the disease. In Tanzania, for instance, domestic dogs around the Serengeti have been vaccinated to form a barrier to distemper. But is the strategy all that helpful? Roelke-Parker has her doubts. If the domestic population becomes healthier and more prolific, there will be more dogs to harass wildlife in the park. And if no one keeps up the vaccinations, the virus will spread that much more readily the next time.

It may be that humanity will opt to do little more than let animals in the wild confront their own predicament. But the spread of morbilliviruses, like the invasion of exotic species, is a problem we had a hand in creating. We may long for the days when wilderness was wilderness, but longing won't make it so: without decisive action, some species may soon be little more than nostalgic memories--victims of paralysis through analysis. In the end, as hard as it may be to protect wild animals from disease, it will be even harder to wait and watch them die.

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