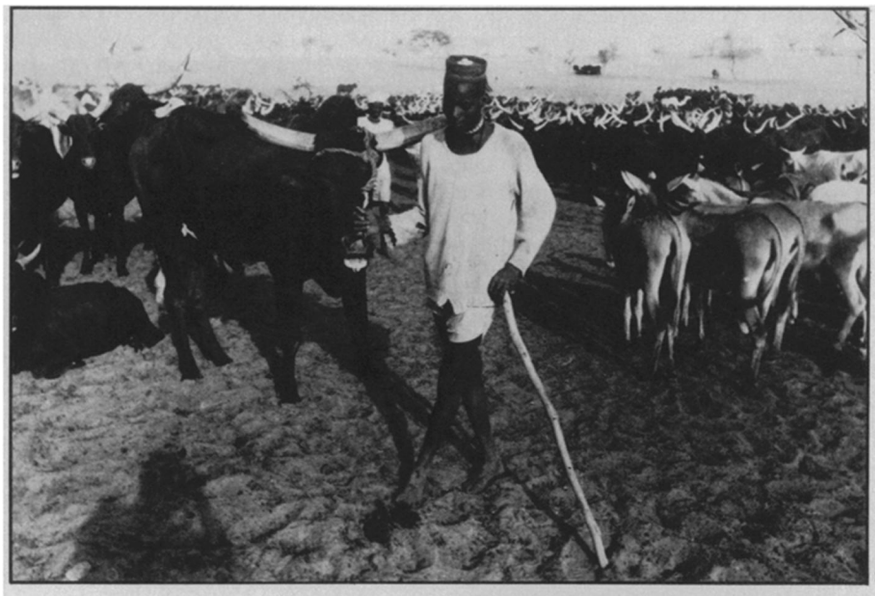
# Taming the Tsetse

With new weapons, including a sterile-male "fly factory," researchers are closing in on one of Africa's deadliest pests

BY WILLIAM J. BROAD



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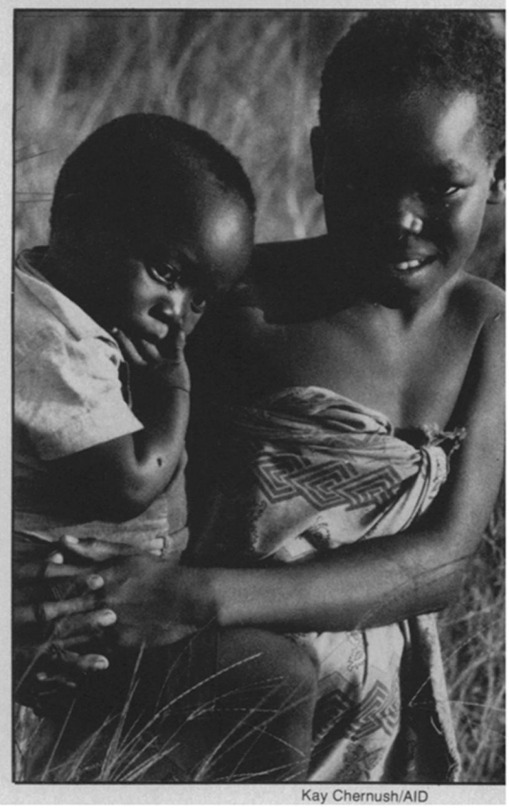
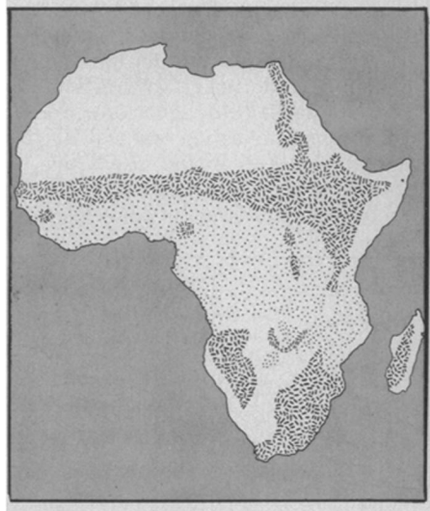


Kay Chernush/AID



*A close look at the boy's eyes (right) reveals numerous tsetse at work. These particular flies carry a parasite harmful to cattle, not humans. A glance at the map of Africa shows that cattle-raising areas (dark) and tsetse-infested areas (light) have almost no overlap.*

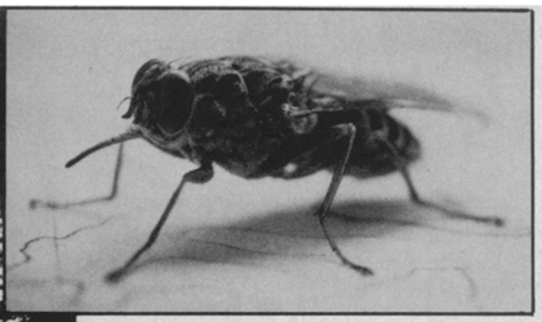
The Tswana tribesman shook his head and spat out a curse. One of his cows lay helpless on the ground, not far from death. Its flesh was limp and watery, and a discharge oozed from its eyes and nose. The hind legs, always first to show signs of paralysis, were already rigid. There was nothing the tribesman could do. He was careful to keep his cattle out of "fly country," but this cow had wandered—and was now paying the price.

Fly country, or the breeding range of the 22 species of tsetse fly, covers an area of Africa larger than the continental United States. Hardly anyone there finds it necessary to mention the tsetse fly by name. The word itself, which comes from the language of the Tswana people, is pronounced *Tseh-tseh*. Spit with proper vehemence, it mimics the buzzing sound made by the insect's wings in flight. There are plenty of other buzzing flies in Africa, but when talk turns to the ravages of "the fly," no one has to ask *which* fly.

The bite of the tsetse can carry a tiny parasite that causes trypanosomiasis, known as sleeping sickness in humans and nagana in cattle, sheep and goats. Ten thousand new cases of sleeping sickness are diagnosed each year. Those victims who don't die may suffer permanent brain damage.

No one knows how many cattle are stricken with nagana. But a look at the map of Africa shows virtually no overlap between major cattle-raising areas and tsetse-infested areas. The reason is obvious. Wherever the tsetse fly abounds, large-scale cattle raising is not feasible. This is most unfortunate, for as beasts of burden and as a source of food, cattle are crucial to Africa's economic development. Yet it is estimated that the threat of nagana has kept 4.5 million square miles of potential grazing land, capable of supporting 125 million head of livestock, out of productive use—in the heart of a protein-starved continent.

A number of recent developments, however, promise to open up vast areas of African grassland. In Tanga, Tanzania, researchers supported by the U.S. Agency for International Development have succeeded in setting up a "fly factory" that produces thousands of sterile male tsetse flies each week. Tests are now underway at a 100-square-mile cattle ranch in Tanzania to see if the sterile males can infiltrate and sabotage the wild tsetse population. The International Atomic Energy Agency, meanwhile, has come up with a skin-like silicon membrane that promises to ease the feeding of captive flies, making for faster "manufacturing" of sterile males. And last year, researchers working at the International Laboratory for Research on Animal Diseases in Nairobi, Kenya, were able to rear the infective form of the trypanosome (tryp for short) outside of fly or host—an essential step in developing a vaccine or drug against the tiny parasite (SN: 4/23/77, p. 261). It was the first such suc-



Kay Chernush/AID

*Worker sprays tree at edge of clear-cut area near Tanzanian ranch, helping keep wild tsetse out of the 100-square-mile test site.*

cess in more than 50 years of efforts. The tsetse, in short, has come under siege.

Conquest may be close, but the battle has been dreary and long-drawn. The first efforts were environmental ones — destruction of parasite-carrying wild game, clearing of woodlands and periodic burning to prevent growth of brush. More recently the trapping of flies, control by natural parasites and application of insecticides to livestock have been tried, but without great success. And newer methods often require huge sums of money. Residual insecticides have been dropped from planes, producing good results. But the tsetse is quickly becoming resistant, and the insecticides cause environmental damage. Traps baited with sex pheromones had been envisioned, but studies showed that male and female tsetse have no way of finding each other except when, by chance, they meet on the back of an animal.

Clashing armies and guerrilla activity, moreover, have forced the suspension of spraying with insecticides in many African countries. The tsetse fly is returning to areas from which it had been eradicated and is infecting regions where it had never been known. "The tsetse invasion is expanding," says Ian McIntyre, a British veterinary scientist who spoke last year in Nairobi at a United Nations conference on tsetse control. "The overall picture in Africa is not a rosy one."

The situation, however, may be easing. The tsetse research project at Tanga, Tanzania, is working with a proven weapon — the sterile male technique that successfully eliminated screwworms, a major livestock pest, from the southern United States in 1965. If the technique can be adapted to Africa, it will be a boon. In Tanzania alone, more than 60 percent of the land is infested with tsetse, making it all but useless for agriculture, grazing or human settlement.

With few pieces of equipment and lots of tropical heat, a team of five scientists, headed by entomologist Leroy Williamson, a hard-talking Texan with a penchant for cowboy boots and country music, have created a fly factory. When Williamson arrived at the project site in Tanga, a sleepy seaside town five hours north of the capital city of Dares-Salaam, all he found were several huts and one ramshackle building near collapse. "It was traumatic," he recalls.

With the help of the Tanzanian Ministry of Agriculture, Williamson transformed the site and got the lab on its feet. The facility now includes three insectories with attached barns that hold 700 goats (for feeding the flies), a radiation building (for male fly sterilization), an emergency power generator, quarantine center, workshops and administrative offices. Even with the site in operating order, Williamson has to stay on his toes, anticipating things that might go wrong — and usually do. Vehicles break down regularly and spare parts can't be found. A cholera epidemic breaks out or trucks can't get through to deliver the goat feed. A record rainfall washes away the road to the ranch just as the fly releases are scheduled to begin. Williamson spent one night hosing down the roof of one of the insectories during a power failure in a frantic attempt to keep the building cool and the flies alive.

Through it all, Williamson, and more than 100 assistants, have made sure that 60,000 buzzing flies in hand-made cages were strapped to the sides of goats for their daily blood meal. And that's just the start. Each day newborn pupae have to be collected, weighed, then incubated for 30 days and separated by sex. And while immersed in liquid nitrogen to limit radiation damage, unhatched male pupae have to be sterilized with small doses of Cesium 137. Would the sterilized males fly as far, live as long and mate as often as tsetse males in the wild? The scientists were rightly concerned, for the healthy tsetse is one of nature's most robust blood-sucking machines.

It is a bristly, brownish insect, about the same size as its cousin, the common housefly. The mouth parts of the tsetse end in a long, thin snout which easily pierces the victim's skin. It feeds exclusively by sucking blood, a hungry fly swelling up to twice its original size during a blood meal.

Blood hunger makes the tsetse an ideal disease reservoir. As the fly feeds, trypanosomes swimming in an infected animal's blood are sucked into the fly's gut. Once infected, the fly becomes a carrier,

Kay Chernush/AID

*Flies swarm toward any dark moving object, even a car. After netting the flies off the black cloth, technicians at the ranch dissect the catch to see how well sterile males are infiltrating the wild fly population.*

passing the parasite while injecting saliva to keep an animal's blood from coagulating before the meal is finished. Any tryps swimming in the fly's saliva are thus swept into an uninfected animal's bloodstream.

Odd is the only word for tsetse reproduction. The female mates once in her life and fertilizes all her eggs with stored-up semen. Once every 10 days during her one-to-three month lifetime, she gives birth to a fully matured larva that burrows into the ground and pupates within an hour. After several weeks the adult emerges. The male tsetse, unlike the female, is a multiple mater.

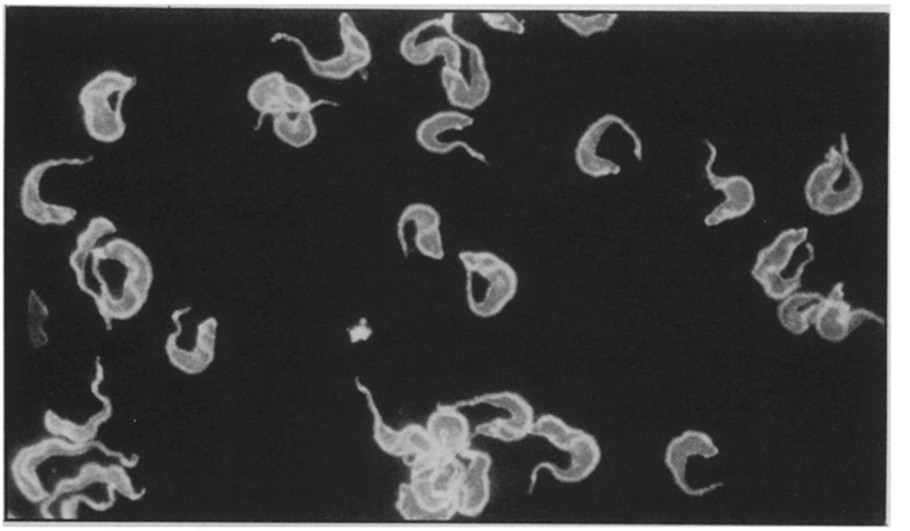
Mating habits could make a tsetse sterile-male campaign very effective once it got going, for one sterile stud could "seduce" many females and trick them into producing eggs that would not hatch. But the female's slow birthrate makes a tsetse "factory" hard to get started. Most insects lay tens of thousands of eggs. But with a captive female tsetse producing about four larvae in her lifetime, founding a colony is a slow, tedious chore.

Just ask Danny Gates, an entomologist from the U.S. Department of Agriculture who spent two years in the Tanzanian bush scratching around, looking under logs, crawling down warthog holes in search of tsetse pupae. From the flies collected by Gates and his 10 Tanzanian co-workers, the Tanga tsetse project has built up a colony of more than 60,000 noninfected flies, which are now the backbone of the project.

But even the back-breaking effort of finding the flies pales in comparison with the preparation of a test site to release the sterile males. At Mkwaja, about 60 miles south of the Tanga laboratory, a huge chunk of ranch set in the heart of fly country has been turned into a tsetse test tube. It stretches for one hundred square miles, and is thick with tall trees, dense undergrowth and waving grasses that stand taller than a man. For two years 250 men with axes and machetes chopped out what amounts to a one-kilometer-wide no-man's-land that encircles the release area.

Into this artificial island, 10,000 sterilized tsetse males are released each week. Swinging nets through the high grasses, workers later round up and analyze a small sample of the tsetse population. Fortunately for the scientists, there are two species of tsetse on the test site. Only one is under attack by the release of sterile males. The other is the control. Since the site was sprayed with insecticide at the beginning of the experiment, both tsetse populations were initially knocked back. Three months after spraying, the control group was climbing back fast and the tsetse under siege were going down. It looks encouraging.

But problems still crop up. The fly round-up recently revealed an alarming increase in the wild tsetse population in one sector of the test site. At first, no one could account for the sudden upward shift. A herd of 20 giraffes was then discovered going back and forth across the clear-cut area, bringing a new supply of tsetse with them each time. Problems like these are no small thing for the scientists. As Gates candidly puts it: "If the project works, we'll be heroes. If it doesn't, it's going to be a heartbreaker. This is not like working in a lab where if one experiment fails you just try another. This is an all-or-nothing shot."



John Doyle/ILRAD

*Immunofluorescence test reveals the type of protein coat on the tryp.*

What has been demonstrated so far is that sterile males apparently *can* hold their own against the wild flies. They seem to fly as far, live as long and mate as often. Whether they will be able to totally outpace the wild tsetse males remains to be seen. The test will not be finished until December 1978. It has already shown, however, that tsetse can be mass-raised in Africa, and that the technology involved can be adapted to the conditions and resources of a developing nation — in itself no mean feat.

Attacking the tsetse fly, however, is only one side of the African skirmish. The battle against the parasite itself, a tiny saboteur that commandeers the tsetse for transport from one victim to another, is also heating up.

The tryp's role in nagana and sleeping sickness has been recognized since early in the century. Yet all attempts to develop a vaccine have failed. What makes the trypanosome such an evasive enemy is its ability to side-step the host's natural defenses. After invading humans or cattle, tryps show a chameleon-like ability to change their protein coating, whose molecular structure signals the host's immune system to produce antibodies against the invaders. As the immune system musters appropriate defenses, the parasites change their coats and force the immune system to mount a new counterattack. As this game of immunological hide-and-seek goes on throughout the course of the disease, the parasites become more and more deeply entrenched in the victim's brain and nervous system, and the patient progressively weakens.

In order to probe the tryp's talent as a quick-change artist, cell biologists have tried for more than 50 years to grow cultures of tryps in the lab—without success. Whenever researchers tried to culture the bug, it invariably reverted into a harmless form. Thus they were unable to learn much about the deadly parasite — to say nothing of devising weapons against it.

A small but significant step, however, has been taken in the conquest. Last year a team of scientists at ILRAD, using grants from the U.S. Agency for International Development and the Rockefeller Foundation, managed to grow in the test tube the long, slender, infective form of the single-celled parasite *Trypanosoma brucei*. That feat was accomplished by Hiroyuki Hirumi, a Japanese-born American cell biologist and John Doyle, a Scottish parasitologist. In effect, they fooled the parasite into acting as if it were in a natural host.

Now that the entire life-cycle of the parasite can be recreated and manipulated in the laboratory, it will be easier to study the tryp's habits and probe for weak points in its own defenses. The ILRAD team next hopes to understand the mechanism that orders the changes in the parasite's coating. That knowledge could perhaps be used to fashion an effective vaccine or more potent drug against nagana and sleeping sickness.

Yet the elusive tryp and its robust carrier, the tsetse, have evaded many attackers. Over the years legions of scientists, politicians and public health workers have heralded the demise of the fly, only to be disappointed. After a visit to Uganda in 1907, Winston Churchill wrote: "International commissions discuss him [tsetse] round green tables, grave men peer patiently at him through microscopes, active officers scour Central Africa to plot him out on charts. A fine spun net is being woven remorselessly around him." That was more than 50 years ago. The net is just now starting to close. □