



Anthrax Epizootic in Zimbabwe, 1978–1980: Due to Deliberate Spread?

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The largest recorded outbreak of anthrax in humans occurred in Zimbabwe during its civil war, in 1979 to 1980. There were a number of unusual features of the epizootic. The disease spread over time from area to area, until six of the eight provinces were affected. Yet anthrax usually appears as a point source outbreak, without significant geographic spread. Only the African-owned cattle in the Tribal Trust Lands were affected; cattle belonging to whites were uninvolved. A critical review of the scientific explanations proposed to account for these events is presented. The possibility that the epizootic could have been a biological warfare event is evaluated. Finally, suggestions are advanced for further investigations into the origin of this epizootic. [PSRQ 1992;2:198-209]

The largest recorded outbreak of anthrax among humans, and possibly the largest among animals, occurred over a decade ago in Zimbabwe, formerly Rhodesia, during the time of its civil war [1]. The outbreak was reported in a series of articles by J. C. A. Davies and others in the *Central African Journal of Medicine* [2-8]. Little was written about it outside of Africa. Over 10,000 human cases and 182 human deaths were documented [9]. Human cases

were secondary to an unprecedented outbreak in cattle [5,10]

UNUSUAL FEATURES OF THE EPIZOOTIC

There were a number of surprising aspects of this epizootic. First, the large number of cases was unusual. Ten thousand seven hundred thirty-eight human cases were documented in Zimbabwe from January 1979 through December 1980 [9]. According to Mandell's *Principles and Practice of Infectious Disease*, published in 1979, "about 7,000 cases are reported in the world annually" [11]. The large number of human cases was particularly unusual in light of the historically low prevalence of anthrax in Zimbabwe [5]. In the 29-year period preceding the epidemic (1950-1978), the period for which records are

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available, a total of 334 human cases were reported in Zimbabwe. By comparison, during the same period (1950–1978) in the United States, 459 human cases were reported [12]. Clearly, anthrax was a rare disease in both countries. According to Davies, "At the beginning of what was to be a major epidemic, it is safe to say that the majority of doctors in Zimbabwe had never seen a case of anthrax" [5]. Yet during the war, anthrax became one of the country's major causes of hospital admissions.

Second, the geographic scope of this outbreak was highly unusual for anthrax. Most outbreaks are characterized by a high degree of focality [11]. Cases occur in limited areas only. Yet in Zimbabwe from 1978 to 1980, the disease spread from area to area, until six of the eight provinces were affected [13]. Anthrax needs very specialized conditions to grow in soil. Alkaline soil containing adequate nitrogen, calcium, and organic matter is required, in conjunction with extreme weather changes, such as a drought followed by heavy rains [14–19]. When these conditions are met, the organisms are thought to undergo a vegetative cycle in soil and then re-sporulate. This process could generate sufficiently high soil concentrations of anthrax spores to cause disease in grazing animals, producing the occasional outbreaks separated by long disease-free intervals that have been observed. However, although this theory accounts for the periodic outbreaks exhibited by anthrax, it has yet to be proved.

Humans generally acquire the infection by handling meat or other products from infected animals. Butchering, preparing, and eating meat from an animal infected with anthrax are frequent causes of the disease in humans and accounted for many cases in Zimbabwe.

There is believed to be no significant spread from animal to animal (except through consumption of infected meat by carnivores, which are relatively resistant to infection) and no significant spread from human to human. Epizootics are generally limited both geographically and temporally [14–16]. Animal cases tend to appear in limited areas over a period of weeks to months, and the epizootic ends naturally as soil conditions change and the density of anthrax spores declines, with no spread to distant areas.

Many of the Zimbabwe cases occurred in areas where anthrax had not been recorded before. Yet in the rest of the world, epizootics generally occur in areas that are known to have produced anthrax

outbreaks in the past, where there is assumed to be low-density contamination of the soil. Anthrax spores in soil may retain their virulence for decades. Epizootics do not spread beyond these areas, although sporadic cases are seen in areas that lack a history of anthrax. The exception to this occurs when an area has become newly contaminated by animal remains or material made from tainted animal products, such as fertilizers or animal feeds made from contaminated bone meal. Such products were not generally used by rural blacks in Zimbabwe [1].

Third, if weather conditions were particularly favorable for the growth of anthrax in soils throughout much of Zimbabwe and often near its borders, then other anthrax outbreaks in adjoining countries might have been expected to occur as well. Yet none of the countries that are contiguous to Zimbabwe reported increased anthrax activity during this period [20].

Fourth, the epizootic was almost entirely confined to the Tribal Trust Lands. These were areas that had been assigned to Zimbabwe's blacks when the country was divided into distinct areas for black and white habitation by the Land Apportionment Act of 1930. Originally termed reserves, the name was changed to Tribal Trust Lands in 1969, and after 1981 they were renamed Communal Farming Areas. By the end of 1979, one-third of Tribal Trust Lands were affected with anthrax, approximately 17% of the land area of the country [10].

Davies noted that "the commercial (white-owned) farming areas appear to have been almost completely spared" [5]. Only four small outbreaks, with 11 cattle deaths, were reported in the commercial farming areas of Zimbabwe by early 1980 [10]. There are no reports of anthrax in white Zimbabweans during this period.

Fifth, the timing of the epizootic coincided with the final months of a long and particularly brutal guerrilla war. Some guerrilla activity had begun in the late 1960s, but the war did not escalate significantly until the mid 1970s. The war ended in late February 1980, when elections were held, and ZANU and ZAPU, the parties affiliated with the two guerrilla armies, won an overwhelming victory.

Human anthrax case reports by month are available for the provinces of Matabeleland, Midlands, and Mashonaland [5]. In Matabeleland and Midlands, cases peaked in November and December 1979, respectively, and decreased thereafter. In

Mashonaland, there were two peaks, the first in February 1980 and a second in December 1980. After the war ended in late February 1980, only sporadic cases were seen in previously unaffected areas, and there appeared to be no further geographic spread of the epizootic. However, anthrax has remained enzootic in Zimbabwe since the war ended, a not surprising finding, given the persistence of the spores in nature.

ANALYZING THE EXPLANATIONS

Owing to the rarity of an outbreak such as this, a number of hypotheses were put forward to account for the epizootic. These hypotheses included spread by insect vectors and contamination of new areas by infected meat that was transported long distances by the rural people. Veterinary services broke down almost completely in many communal farming areas during the war, and this lack of veterinary services was felt to contribute to the outbreak.

It was also suggested that anthrax had been more prevalent prior to the onset of the epizootic than was recorded, but, because of limited reporting from rural areas, the cases had not been documented.

Five questions that address the underlying issues in the explanations listed above have been identified. A review of the experimental and epidemiological literature on anthrax transmission was undertaken to answer these questions as precisely as possible. Many questions about the natural history of anthrax still exist [21,22]. However, despite limitations in the literature, this review permits a critical evaluation of the issues involved.

QUESTION 1: *What role did blowflies, horseflies, stable flies, and mosquitoes (the insects reported to be likely vectors) play in the transmission of anthrax to cattle?*

Observations Opposing Vector Transmission

Sparing of Young Animals. Several authors have noted the relative sparing of calves by anthrax [16,23]. This observation has been interpreted as supportive evidence for oral ingestion as the primary portal of entry of anthrax infection; young animals are thought to be spared by feeding primarily on their mothers' milk, thus ingesting less grass and soil.

Cutaneous Versus Systemic Disease. The anthrax syndrome seen in cattle is almost always systemic

and hyperacute, unlike the disease in horses and humans, both of which tend to develop localized areas of swelling and have a more prolonged disease course [16,24-26]. On this empirical basis alone (the absence of localized areas of swelling), most authors dispute insect vector transmission to cattle, even when they believe that it occurs in other species [26]. However, a few sources do report the existence of a cutaneous anthrax syndrome in cattle and link it to fly bites [27,28]. Still other authors report the presence of localized swelling in cattle but suggest that it is due to the systemic disease rather than local inoculation [16].

Volume of Blood Needed. Transmission of anthrax to cattle by any species of fly would require the inoculation of organisms through the skin. However, in the hands of several investigators, cattle have proved notoriously difficult to infect with anthrax by parenteral injection [24]. Using intravenous, intramuscular, subcutaneous, and intradermal routes, Schlingman and colleagues gave cattle injections of up to 600 million spores but were unable to produce a fatal infection or determine an LD₅₀ [29]. In contrast, infection by the oral route was easier to induce.

In all animal species studied, the level of terminal bacteremia of anthrax organisms ranged from about 100 million to 1 billion colony forming units/ml [30-32]. This suggests that, for a biting fly to induce anthrax infection by parenteral inoculation of blood to a cow, on the order of 1 ml of blood would need to be transferred, a herculean task for a fly. Furthermore, loss of large blood volumes to second hosts is unlikely in flies, since it would be unfavorable for survival.

There are two caveats to this analysis. Every study that specified the inoculum sizes employed for parenteral inoculation of anthrax used spores. Yet theoretically, insects can transmit either spores or vegetative forms of the organism. No studies exist that provide estimates or measurements of inoculum sizes needed to induce infection with the vegetative form of anthrax. Moreover, the size of the infectious dose depends on the anthrax strain employed. Yet most of the studies cited here did not identify the anthrax strain used.

Literature Review

The most complete review of more than 100 years of literature on experimental vector spread of an-

thrax was performed in 1971 [33]. In his summary of the data, Greenberg emphasized our lack of knowledge and concluded that the issue of vector transmission remained unresolved.

Individual Analyses by Vector

Blowflies. The presence of hundreds of bubbles of blood resulting from insect bites, which are commonly seen on carcasses of animals that died of anthrax, has been mentioned as supporting insect vector transmission. Blowflies (*Chrysomya*) are the only flies that feed on carcasses that have been suggested as anthrax vectors in Zimbabwe. Tabanids, muscids, and mosquitoes (the other suggested insect vectors in Zimbabwe's epizootic) are unable to feed on dead animals, although they may attempt to do so and acquire a small number of organisms in this fashion [34].

Because blowflies do not feed on living creatures, they cannot transmit infection to animals or humans directly via a bite. It is reported that blowflies regurgitate infectious droplets onto the leaves of trees or bushes where they perch, at a height between 1 and 3 m above ground level [35,36]. It has been further suggested that kudu develop infection after feeding on leaves contaminated with anthrax by blowflies [36,37]. With respect to cattle, however, leaves have never been implicated in anthrax outbreaks.

Sen and Minett did transmit anthrax to goats through the blowfly (*Calliphora erythrocephala*) and the housefly (*Musca domestica*) [38]. Their procedure entailed bringing the flies in contact with incisions made in anthrax-infected carcasses and then transferring the flies to the cauterized skin of uninfected goats. Although it is not made explicit in the report, this transmission appears to have occurred as a result of deposition of anthrax spores present on the body parts or feces of the vectors, rather than via a bite. Twenty-four to 50 flies per goat were employed in these experiments. The authors reported that they used goats because goats are more susceptible than cattle to experimental inoculation.

Discussion of anthrax transmission to cattle by blowflies is hypothetical; no experimental evidence documents such transmission.

Stable Flies and Mosquitoes. Sen and Minett attempted to transmit anthrax to goats through the use of stable flies (*Stomoxys calcitrans*) by feeding

the flies on infected goats and transferring them to the healthy skin of uninfected goats [38]. They then attempted to feed flies on incisions made in goats that died of anthrax and transferred the flies to the cauterized skin of healthy goats. Finally, they exposed the cauterized skin of healthy goats to anthrax-contaminated fly feces. In none of these experiments was anthrax transmission achieved. It was noted that application of an anthrax culture suspension to goat skin that had been cauterized or had recently received *Stomoxys* bites led to infection, while the same suspension applied to healthy skin caused no disease.

In 1987, Turrell and Knudson did produce anthrax by vector transmission in mice and guinea pigs [32]. Their work was initiated partly in response to the Zimbabwe epizootic to resolve whether insect vectors could account for the massive spread of disease. Mosquitoes (*Aedes aegypti* and *Aedes taeniorhynchus*) and stable flies (*S. calcitrans*) were the vectors studied. Even under optimal conditions (feeding insects on animals immediately prior to the animals' deaths, interrupted feedings with forced transfer to a second host, shaving to remove hair from guinea pigs, and transfer of more than one infected fly to the second host), there was only occasional transmission of the disease. Twelve percent and 17% transmission rates were reported.

Although Turrell and Knudson concluded that "various forms of evidence strongly suggest that flies play a role in the transmission of *Bacillus anthracis* to humans and domestic animals during an anthrax outbreak," they neglected to take into account the huge difference in susceptibility and infectious dose between mice and guinea pigs, on the one hand, and humans and cattle, on the other. Since the parenteral LD₅₀ for mice and guinea pigs is only 5 to 50 spores [31,39], but the LD₅₀ for cattle appears to be more than 100 million spores, the demonstration of vector transmission to mice and guinea pigs cannot be extrapolated to cattle. In fact, the opposite conclusion should be drawn: if small rodents are only occasionally infected, then cattle, with an inoculum size more than a million times greater, will rarely if ever be infected by the vectors studied.

Horseflies. Horseflies (tabanids) are larger than the mosquitoes and stable flies studied and ingest a larger volume of blood when feeding, perhaps 100 times the volume ingested by mosquitoes and 20 times the volume of *Stomoxys* [40]. Yet, given the

orders of magnitude involved, the likelihood of tabanid transmission of anthrax to cattle appears small.

Tabanids have been frequently reported as vectors in the transmission of tularemia. For transmission of tularemia, however, "as few as 10 to 50 organisms are sufficient to cause infection by cutaneous inoculation" [41].

QUESTION 2: *Can the human consumption and transport of infected meat be used to account for new infections in cattle?*

The transport of meat can explain the occurrence of human cases in sites distant from the original outbreaks. But it does not explain cases in cattle at the secondary locations.

First, no transmission from humans to cattle by direct contact has ever been reported.

Second, since cattle are herbivorous, they would not consume meat from infected carcasses. Although infected animal bones or body parts might be discarded at the secondary sites, the areas they could contaminate would be limited, and transmission to very few animals would be expected. This is even more true if a cycle of growth in soil is needed for anthrax organisms to achieve sufficient concentrations in soil to cause disease. Most anthrax experts believe that a soil growth phase is necessary [15,16,25,42,43], though it is disputed by some and has not been proved experimentally [44,45].

Third, although the human butchering and consumption of anthrax-contaminated meat has been reported from many countries [45-51], no secondary cases in cattle have been reported as a result.

Fourth, even prior to 1978, when animals were found dead from anthrax in Zimbabwe, the meat was often consumed by the rural people [10], yet large outbreaks did not result.

QUESTION 3: *Was there a precipitous increase in human and animal cases of anthrax beginning late in 1978?*

Anthrax is reported to have been a rare disease in Zimbabwe in both animals and humans prior to 1978 [5-8,52]. According to Zimbabwe Veterinary Research Laboratory scientists, only about 20 cases per year in livestock had been reported annually [10].

Dr. Max Sterne, South Africa's anthrax expert and originator of the animal vaccine used worldwide,

wrote that, when all animals that had died were screened for anthrax in a South African study, a fourfold rate of underreporting was found [53]. Extrapolating this figure to Zimbabwe, which may or may not be applicable, only an estimated 80 cases in livestock would have occurred annually, still a relatively small number. In Sterne's 1967 map of worldwide anthrax incidence, Zimbabwe was placed in the lowest incidence category for animal anthrax [53].

The human case numbers from the annual and monthly reports of the Zimbabwe Minister for Health from 1950 to 1985 (when publication ceased) are shown in the Table. Regular publication of yearly human anthrax cases began in 1950. The reported incidence of human cases during the period from January 1, 1979, to December 31, 1980, was more than 400 times the average incidence of the previous 29 years.

QUESTION 4: *How did the cessation of veterinary services in the Tribal Trust Lands contribute to the spread of anthrax?*

Routine anthrax vaccination of livestock was not

Table. Human Anthrax Cases in Zimbabwe, 1950-1985

Year	No of Cases	Year	No. of Cases
1950	6	1968	8
1951	5	1969	1
1952	4	1970	11
1953	2	1971	4
1954	27	1972	0
1955	—	1973	0
1956	—	1974	10
1957	8	1975	0
1958	5	1976	29
1959	15	1977	11
1960	0	1978	2
1961	0	1979	4,002
1962	1	1980	6,736
1963	1	1981	1,575
1964	*	1982	1,229
1965	65	1983	295
1966	60	1984	295
1967	59	1985	730

Source: Southern Rhodesia Report on the Public Health (reports for the years 1950 through 1959); Annual Report on The Public Health of The Federation of Rhodesia and Nyasaland (reports for the years 1960 through 1963); Ministry of Health for Southern Rhodesia, bulletin of diseases reported during months ended (bulletins for the years 1965 through 1977); and Report of the Secretary for Health (reports for the years 1978 through 1985)

— = no entry for anthrax; * = no report available for 1964

practiced to a large extent in Zimbabwe before 1979, according to local veterinary experts.

Although vaccination certainly may have prevented spread to the commercial (white-owned) farms once the outbreak was underway, if vaccination had not been practiced widely prior to the epizootic, then it cannot be credited with keeping the earlier rates so low. One must instead assume that soil contamination by anthrax was previously not widespread in Zimbabwe.

Likewise, if vaccination had not been routinely practiced on a large scale, then the breakdown in veterinary services, which accompanied the turmoil of the war, was not a factor in the early development of the anthrax outbreak, as commonly believed.

Although the well-publicized breakdown in animal dipping contributed to the increase in tick-borne diseases of livestock and was thought by many lay people in Zimbabwe to have contributed to the anthrax outbreak as well, livestock dipping by itself does not contribute to anthrax prevention. The only role that the breakdown of dipping may have played in the anthrax epizootic was in the interruption of periodic contact between villagers and veterinary workers. As a result, the reporting of anthrax cases and of unexplained animal deaths stopped. In this way there was probably interference with the usual process of identifying anthrax outbreaks, with vaccinating in response to them, and with the appropriate disposal of carcasses.

To summarize, the absence of veterinary services does not seem to account for the onset of the epidemic or its geographic spread, but, had such services functioned in the communal farming areas, there should have been fewer cases in both humans and cattle.

QUESTION 5: *How can the spread to previously uninvolved areas be explained?*

Stein has published perhaps the most complete epidemiological assessment of anthrax outbreaks in nature. He surveyed the entire United States for anthrax incidence and endemicity. Describing his observations of anthrax occurrences, he wrote: "In the United States anthrax occurs in epizootic form in regions in which the soil is known to be seriously infected. However, it may occur sporadically anywhere at any time, and thus may appear where previously not identified or where it has been quies-

cent for a long period" [27] (emphasis added). Whitford, who analyzed many anthrax outbreaks, made a similar observation [54]. Later, Stein examined increasing anthrax outbreaks from 1945 to 1955 throughout the United States and addressed the issue of spread to previously unaffected areas. He reported that outbreaks occurring in cattle in new areas were mainly due to infections acquired by vaccination with improperly prepared batches of vaccine. In swine, they were mostly of suspected food origin [55]. Blood, Henderson, and Radostits concur, writing that "introduction of infection into a new area is usually through contaminated animal products such as bone meal, fertilizers, hides, hair, and wool, or by contaminated concentrates or forages" [25].

Inferred from these data is that Zimbabwe's anthrax epizootic is most consistent with the new introduction of the organism by some means into Zimbabwe.

WHAT DO THE ANSWERS TO THESE FIVE QUESTIONS INDICATE?

The pattern of acquisition of anthrax in humans was consistent with its natural pattern elsewhere: secondary to contact with infected animals or animal products [5]. The only unusual epidemiological feature with respect to humans was the massive number of cases. A small number of human cases might perhaps have been secondary to spread by insect vectors [55]. (The human parenteral infectious dose is unknown, so the likelihood of vector transmission is difficult to assess.)

The disease in cattle is another story. Anthrax spread in a way that cannot be accounted for either by insect vectors or transport of infected meat. Vectors and movement of meat may have accounted for occasional, sporadic cases but would not have been expected to play a major role in the development of Zimbabwe's massive outbreak.

Lacking an identified source of anthrax contamination, the massive epizootics in previously uninvolved areas are unprecedented. Although cases elsewhere are seen in areas with no history of prior anthrax infections, these cases have consistently been only occasional and sporadic, or, when seen on a large scale, they have been traceable to a newly introduced source of infection.

Weighing all available evidence, it is suggested

here that a plausible explanation for the sudden peak of anthrax in the Tribal Trust Lands beginning in November, 1978, is that one or more units attached to the Rhodesian military may have air-dropped anthrax spores in these territories. This action would expose cattle to the disease through ingestion or inhalation (or both) of anthrax spores. Humans would have acquired the disease from meat or meat products.

ANTHRAX AND BIOLOGICAL WARFARE

For the above reasons, this epidemic may not have been a natural occurrence, and might instead have been the result of deliberate spread—employing anthrax as an agent of biological warfare. No proof exists for the deliberate use of anthrax; nor is there positive evidence of the origins of anthrax spores that may have been used or the technical means by which they may have been disseminated. The following discussion, however, attempts to put these issues into the context of what is known about anthrax and biological warfare.

Technologically, production of anthrax spores is not a difficult problem. Anthrax weapons were developed and tested by at least the Japanese, British, and United States governments during the Second World War [56–58], and it is suspected that a number of other nations have developed or acquired the technologies since [59]. The spores are stable under a wide range of conditions of temperature, pressure, and moisture. Many means exist for delivering viable anthrax spores [60,61]. Experiments on Gruinard Island (where the British tested anthrax weapons in 1942–1943) included release from exploding bombs and by airplane [62]. Either of these two methods, or other methods, could conceivably have been used in Zimbabwe. Deliberate contamination of animal feeds or fertilizers might have accomplished the same end, although these products were not widely used.

Although aerial release of anthrax spores is generally thought to result in an epidemic of inhalation anthrax in humans (which was not reported from Zimbabwe), inhalation anthrax is not necessarily what would have been seen had this method been used in Zimbabwe, for the following reasons.

Inhalation anthrax results from the intrapulmonary deposition of individual spores less than 5 microns in size. It is a rare disease, with 18 human cases reported in the United States between 1900

and 1980 [63]. This is despite the fact that some woolen mills, tanneries, and even laboratories researching anthrax have been shown to be contaminated with anthrax spores when air and surface sampling have been performed [63].

Little information is available regarding the factors that influence human susceptibility to anthrax. Besides susceptibility, the other factor that determines whether a person develops inhalation anthrax is the inoculum size to which he or she is exposed. For inhalation anthrax, this would be a function of the spore concentration, the amount of time the spores remain airborne, and spore size (larger spores are trapped before reaching the pulmonary parenchyma). Although the inhaled infectious dose for humans is unknown, studies in monkeys and estimates of spore numbers inhaled by workers in contaminated factories exist. These indicate that for 3-pound cynomolgus monkeys the LD₅₀ is over 17,000 inhaled spores, for chimps about 40,000 spores, for rhesus monkeys about 80,000 spores, and for humans something probably greater [39,63,64].

In Zimbabwe, where "protected villages" existed in many parts of the country (which entailed the creation of new population centers by removal of blacks from their rural farms to regulated areas) and the movement of rural blacks was in some areas strictly controlled, it may have been possible to accomplish airborne spraying and yet avoid populated areas. Or, had spores been prepared as pellets or cattle cakes (as was planned by the British for a possible raid on Germany) [57], inhalation anthrax would not have occurred.

Another consideration is that, by the war's end, many of the medical facilities in the rural areas were no longer functioning. Had cases of inhalation anthrax occurred, most of the patients would probably have been unable to reach a treatment facility prior to death, which usually occurs within 24 hours of the onset of symptoms significant enough to seek medical attention.

To manufacture anthrax weapons under ideal conditions, high-containment suites are employed. However, such facilities were not available to the nations that manufactured such weapons during World War II. As pointed out, even in settings highly contaminated with anthrax spores, only rare cases of inhalation anthrax have occurred. Cutaneous anthrax was more common but could be easily treated. Therefore, use of such suites, though desirable, is

not mandatory for production of anthrax weapons.

Since human anthrax vaccine has been available since the 1950s, is currently manufactured in at least three countries, and is recommended for persons with occupational exposures, procurement of the vaccine for workers who may have been engaged in researching or producing anthrax weapons should not have been difficult. Obtaining cultures of the organism is also not difficult; cultures might have been available from the American Type Culture Collection in Rockville, Maryland, or from the Centers for Disease Control [65]. Anthrax is also easily cultured from soil found in endemic areas or from some infected animal remains [19]. Methods for culturing the organism and inducing sporulation are described in the open literature.

Anthrax weapons produced by the military existed in the United States until about 1970, when President Nixon gave orders for their destruction. One cannot totally exclude the possibility that some biological munitions were transferred from the United States to other countries prior to their destruction. It is also not impossible to imagine that such weapons could have been produced by a nation that was not a complying party to the Biological Weapons Convention (which in any case only entered into force in 1975), or even by a renegade group, and could have found their way to Zimbabwe.

POLITICAL CONSIDERATIONS

Why would such an action be carried out? Could it possibly have benefitted the war effort? What political risks would have been faced by a possible perpetrator?

The net result of the anthrax epizootic appears to have been the impoverishment of the affected rural populations. Cattle were the major source of wealth for black farmers. Describing the effects of the outbreak 10 years later, Pugh and Davies paid testimony to its effects on a rural economy: "There is always hardship, but if cattle die, the family loses its source of wealth; without motive power for ploughing, crops cannot be planted, leading to no food, no money to purchase food, pay school fees, bus fares, taxes, or buy the essentials to life. The family is reduced to grinding poverty and malnutrition becomes rife" [13]. Most likely, cattle would have been the objects under attack, and human cases occurred only incidentally.

One can perhaps imagine that, as the war escalated with no end in sight, and with a black population becoming ever more polarized in favor of the guerrillas, a willingness developed to use any weapon that might lead to victory. Since the many programs that had tried to stop the local populations from providing the guerrillas with support had failed [66–68], the local population itself may have come to be seen as the enemy. According to J. K. Cilliers, who published his dissertation analyzing Rhodesian counterinsurgency strategy, "by the overly aggressive use of tactics . . . Security Force actions tended rather to be aimed against the local population than in defence of them" [67].

Point 1: Food Control

The rationing and limiting of food supplies to the black population was in fact a part of the military strategy for controlling the population and restricting their support to the guerrillas. In his analysis of military strategy, Cilliers provides details of this approach:

During the final months of 1976, food control measures were instituted . . . food in the Tribal Trust Lands had become less readily available to the insurgent forces owing to a general drought and the movement of locals into Protected Villages . . . The intention was to further limit even these supplies by rationing the farm laborers to that which was needed . . . Farmers were to ration their laborers on a day-to-day basis with only sufficient food for a particular day. No surplus would therefore be available to feed insurgent forces, even were this demanded by force of arms. Tight food control would force insurgents to spend much time seeking sustenance, which would hasten their location and eventual elimination. A further advantage could result from hostility between the local population and insurgents as demands on limited available foodstuffs increased [67].

In late 1977,

Intelligence reports indicated that ZANLA (Zimbabwe African National Liberation Army) morale in the area was on the verge of collapse because of their inability to obtain either food from the local population or water from points outside Protected Villages. These had largely been destroyed by Security Forces Operations [67].

Naturally, the local population felt the effects of these policies as well as the guerrillas. "Malnutrition and disease had always been features in black rural life for numerous decades. The concentration of people (into Protected Villages) tended to exacerbate

these problems" [67]. Discussing the situation in late 1978, Cilliers points out: "An increasing number of reports of malnutrition were reported by the few doctors that remained in rural areas" [67].

Point 2: Escalation of Tactics Aerial Bombing of Zambia and Mozambique

According to Ken Flower, head of the Rhodesian Central Intelligence Organization (CIO) during the war, the guerrillas escalated a terrorism campaign beginning in June 1978 that culminated in the shooting down of a civilian Viscount airliner and massacre of many survivors in September 1978 [68]. Their actions were met by the Rhodesian government with parallel escalation: regular airborne bombing raids into Zambia and Mozambique were initiated in October 1978, attacking training camps established by the guerrillas. This was begun despite warnings from the U.S. and Britain that taking the war outside the country in a significant way could lead to a superpower conflict in southern Africa. Thousands of men, women, and children were killed in these bombing raids.

It was in November 1978, a month after initiation of the bombing raids, that the first human anthrax cases were reported following an outbreak in cattle. Thus, the epidemic did coincide with a period of escalation of tactics by the Rhodesian military.

Point 3: Chemical and Poison Weapons

Was there a parallel, well-documented use of other weapons considered abhorrent during the conflict and a willingness to overlook civilian casualties? In his memoirs, Flower admits to the deliberate distribution of poisoned clothing, which killed hundreds of black guerrillas [68]. Yet clothing can be worn by anyone. Organophosphate poisoning from tainted clothing affected civilians as well, and poisoning by this means became documented in the Zimbabwe medical literature [69,70].

Dr. Paul Epstein, an American physician practicing in Mozambique for the Ministry of Health, with support from the American Friends Service Committee in 1978, treated large numbers of Zimbabweans, who had arrived from ZANLA training camps, for a bleeding disorder. Initially a viral hemorrhagic fever was suspected. But there were many deaths despite treatment. Eventually a fat biopsy was obtained and sent for toxin analysis; this analysis revealed the presence of warfarin [71]. Thus

another unconventional mode of warfare, warfarin poisoning, may have been employed by some within the Rhodesian military.

Although the use of bacteriological weapons and poisons such as organophosphates was and is prohibited by the 1925 Geneva Protocol, of which the United Kingdom is a party, the events described occurred after Rhodesia's Unilateral Declaration of Independence from the U.K. Thus, whether use of such agents would have been in actual violation of the treaty is arguable (Rhodesia was not a party to the Biological Weapons Convention, which bans the possession and use of biological weapons.) Since Rhodesia was already subject to an international embargo, which had been in force since 1965, fear of an international response to the use of chemical and biological weapons was probably not a significant deterrent.

Reporting recently in a TV documentary and magazine article on confidential interviews with former contractors for the Rhodesian military, Jeremy Brickhill, a Zimbabwe journalist and veteran of the conflict, claimed that the Rhodesian CIO and Selous Scouts (an arm of the Rhodesian military that employed blacks who successfully masqueraded as guerrillas) used anthrax, cholera, thallium-contaminated foodstuffs, and organophosphate-impregnated clothing in the later years of the war [72,73].

CONCLUSIONS

A case has been made for the possible deliberate use of anthrax as an agent of biological warfare, directed at African-owned cattle, in the final months of the Zimbabwe civil conflict.

The characteristics of Zimbabwe's anthrax epizootic are unusual. Outside Zimbabwe, outbreaks of animal anthrax have remained confined to enzootic areas or could be traced to contaminated animal products and have been generally self-limited. Zimbabwe's epizootic did not conform to this expected behavior, and the arguments put forward to explain it are unconvincing.

A military role for anthrax can be postulated, given the strategic control of food and other resources that existed at the time. Deliberate impoverishment of rural blacks may conceivably have been a strategy as well. Desperate tactics appear to have been used by the Rhodesian military elsewhere as the war drew to a close. Finally, there have been

recent reports attributed to confidential eyewitnesses that support the theory of the deliberate spread of anthrax

NEXT STEPS

During the past 45 years, no allegation of biological warfare has undergone careful scientific analysis and been brought to a satisfactory conclusion. There exists no generally accepted methodology to serve as a guide for the design of an investigation into the possible use of biological weapons [74-76]

A lot is known about the ecology of anthrax, and this knowledge could be employed to design studies that would help to resolve the issue of the origin of Zimbabwe's anthrax epizootic. Soil sampling could be used to detect the presence of anthrax in soils. The extent of anthrax found in communal versus commercial farming areas would be interesting. Finding high anthrax spore counts in unusual locations, such as in places that do not support its growth, would contribute to an understanding of the epizootic.

Recent developments in biotechnology can be used to resolve issues such as this. Characterization of the genetic structure of Zimbabwe anthrax strains can be used to estimate the likelihood that the strains found originated from locally occurring southern African strains, as opposed to strains that are found elsewhere or are held in laboratories

A third approach to this question might look at the documents that are available on the military actions and strategies used during the war. For example, one tactic used by the military was to assign areas of land to be "no go" or "frozen" for varying periods. This enabled certain military units to carry out special projects in the designated areas. During this time, no unassigned military units were allowed to operate within the areas and civilian access may have been restricted [67]. It would be useful to know whether land that was heavily affected by anthrax was "frozen" shortly before the first local cases of anthrax were seen.

During the time following the national elections and before transfer of power to the Mugabe government several weeks later, a large number of government records were destroyed [66]. Still, documents and sources remain. For instance, an archive of the papers retained by Ken Flower, who stayed on to work for the Mugabe government after the war,

exists in Harare. Other former military and intelligence officers continue to live and work in Zimbabwe.

It is now possible to design a careful and definitive investigation of this issue. The time has come for a thorough inquiry.

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