

Reanalysis of the Anthrax Epidemic in Rhodesia, 1978-84

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ABSTRACT

In the mid-1980s, the largest epidemic of anthrax of the last 200 years was documented in a little known series of studies by Davies in *The Central African Journal of Medicine*. This epidemic involved thousands of cattle and 10,738 human cases with 200 fatalities in Rhodesia during the Counterinsurgency. Grossly unusual epidemiological features were noted that, to this day, have not been definitively explained. This study performed a historical reanalysis of the data to reveal an estimated geographic involvement of 245,750 km², with 171,990 cattle and 17,199 human cases. Geospatial time series analysis is suggestive of multiple, independent geotemporal foci of anthrax introduced via an unknown mechanism rather than re-emergence from native endemic foci.

INTRODUCTION

Anthrax is a potentially lethal disease caused by *Bacillus anthracis*, an aerobic spore-forming bacterium that exists in a complex ecological cycle predominantly involving herbivorous mammals and man. Persistence of anthrax in the environment is due to soil borne spores that remain viable for decades. It is an Office International des Epizooties (OIE) List B disease, for which obligatory reporting is requested of all OIE Member States. The ancient origin of *Bacillus anthracis* is sub-Saharan Africa, specifically the region encompassing Kruger National Park and the North Cape Province of South Africa. Over the centuries, anthrax has been exported throughout the world predominantly through the trade of domestic herbivores.¹

Anthrax was first recognized in colonial times in southern Africa in 1842 and was placed on the list of scheduled diseases in South Africa in 1891. Europeans noted the persistent spread of

anthrax in their cattle herds and eventually pushed for control programs that yielded the development of vaccines from 1920 onward. In 1923, South Africa had reported the death of 30,000-60,000 cattle, however once vaccination was initiated, a dramatic reduction in bovine anthrax was noted. The culmination of an effective vaccination program in South Africa was realized with the Sterne vaccine in 1937, where knowledge of South Africa's success in controlling anthrax was shared with Rhodesia, now known as the country of Zimbabwe.^{1,2}

In 1978, during the context of the Rhodesian Counterinsurgency, an unprecedented anthrax epidemic in livestock and humans began in Rhodesia. The epidemic progressed largely unchecked until the mid-1980s as the largest known anthrax epidemic in history. This event was documented by Davies in a three-part descriptive study from 1982 to 1985 that was limited to three of five involved provinces³⁻⁵. Additional primary source documentation was limited that provided greater insight into the location, case count, spread, and etiology of this epidemic.

A total of 10,738 human cases and approximately 200 deaths (1.9% fatality rate) were reported by JCA Davies in *The Central African Journal of Medicine* across Midlands, Matabeleland, and Mashonaland Provinces from January 1979 through December 1980. The majority of reported human cases were the cutaneous form of the disease, however all known clinical forms of anthrax infection were documented during the course of the epidemic. The universally acknowledged source of human cases was cattle infected in rural, noncommercial farming areas known as Tribal Trust Lands.³⁻⁵ The majority, if not all of the cases were among indigenous African farmers living on Tribal Trust Lands. This was an important observation given that approximately 85% of the country was considered agricultural land, of which half was Tribal Trust Lands.⁶ Because of the extent of agricultural land involved, the epidemic was described as “an economic disaster”.⁷

The two primary agricultural land classifications of Rhodesia in the late 1970s were Tribal Trust Lands and commercial farming areas. Tribal Trust Lands covered 16.4 million hectares of rural Rhodesia and were designated by the government for subsistence agriculture by indigenous ethnic groups. These areas were known to be of generally marginal agricultural value because of climatic conditions (sporadic rainfall and lengthy droughts), soil quality, and farmers' lack of resources (e.g., lime) to improve and maintain soil quality. Over 4 million people lived in the Tribal Trust Lands, 800,000 of them belonging to indigenous African farming families; this represented the majority of the rural indigenous population of the country at twice the population density of the Europeans living on the commercial agricultural areas. The majority of cattle raised on the Tribal Trust Lands were used for agricultural labor, personal consumption, trade, and sources of fertilizer. Commercial agricultural areas included farms owned by European farmers who raised cattle for profit, as well as tobacco and other cash crops. The land appropriated for these farms was roughly equal in size to the Tribal Trust Lands and was located in fertile areas.^{6, 8, 9}

African lineages of cattle were comprised of *Mashona*, *Matabele*, *Zansi*, *Amabula*, *Kavuvu*, *Amabowe* types and had been present in Rhodesia since at least the early 1800s, which was the limit of documented history in this region of the world. Cattle were an integral part of migrant indigenous peoples of the region during this period. These breeds intermingled with European stock introduced during the colonial period.² Loosely considered, the African lineages resided on the Tribal Trust Lands, whereas the European lineages resided on commercial agricultural lands.

Prior to 1960, Rhodesians had destroyed much of the indigenous wildlife during the expansion of cattle ranching. However, after 1960, concerted effort was directed to the re-establishment of game and wildlife both on private ranches and in national parks. Private ranches engaged in

profitable trade with the tanning industry, where elephant, antelope, and zebra skins garnered the bulk of the industry's income in the mid-1970s.¹⁰

Prior to the war, the national anthrax control program was considered one of the most advanced and effective in Africa. This was a program that had been in place since approximately the mid-1950s. During the war, vaccination for anthrax was maintained largely on commercial as opposed to Tribal Trust Land farms due to chronic distrust of indigenous Africans directed towards European veterinary practice.² The disruption of standard veterinary services on the Tribal Trust Lands during the war was associated with a dramatic resurgence of multiple diseases in cattle, such as various tick-borne diseases, trypanosomiasis, rabies, and two outbreaks of vaccine-resistant hoof-and-mouth disease.^{3, 11}

Anthrax was rare in Rhodesia prior to 1978, as shown in Figure 1. In 1898, 41 cattle died of anthrax in Matabeleland Province, which was the first documented appearance of anthrax in Rhodesia. In 1912, 14 pigs and one donkey in Ardbennie and six cattle in Umganin, Bulawayo, died of anthrax. At Mount Hampden, nine cattle died in a limited outbreak in 1917. A larger epidemic in Shamva two years later resulted in the deaths of 102 head of cattle on 18 farms. In 1920, 18 cattle died in Hartley, Mtoko, and Shamva. All of these outbreaks were in western Rhodesia, north of Hartley.¹² Other limited outbreaks in bovines and humans were reported in Chipinga (1952–54), Mhondoro Tribal Trust Land (1974), and Mount Darwin (1965 and 1970–71).^{3, 5, 11, 13, 14} There were 6 human cases and 2 deaths (33 % fatality) reported in the Mhondoro outbreak; the high fatality rate was attributed to delays in seeking timely medical attention.⁸ Data on human cases were unavailable for the Chipinga and Mount Darwin outbreaks. No human or bovine anthrax cases were reported in all of Rhodesia from 1972 to 1977, and no outbreaks of anthrax were documented in Rhodesian wildlife until 2004.¹⁵

FIGURE 1. Historical anthrax human cases reported from 1926 to 1977. Vaccination for anthrax in cattle was not available until the mid-1950s.

Human anthrax was rare, as Davies noted, “the majority of doctors in Rhodesia had never seen a case of anthrax”.⁵ The majority of reported human cases in these earlier outbreaks were cutaneous and, to a much lesser degree, gastrointestinal; infections were acquired from handling or eating infected livestock.³⁻⁵ The approximate annual number of human cases reported nationally was 6 per year, for a total of 322 cases and 20 fatalities from 1926 to 1977.³

The purpose of this study was to re-examine the 1979-80 anthrax epidemic in Rhodesia in light of new data and analytic insights gained in the years since this important event.

MATERIALS AND METHODS

We conducted literature searches using PubMed (United States National Library of Medicine) and AGRICOLA (United States National Agricultural Library) for all references to anthrax in southern Africa from 1970 to the present. We also reviewed all available veterinary and agricultural literature published in Rhodesia from 1975 to 1985. Historical land classification maps published for Rhodesia from 1975 to 1981 were obtained from the United States Library of Congress¹⁶. All available original manuscripts regarding anthrax in Rhodesia were reviewed, and a reanalysis of the epidemiological data contained therein was performed. We examined epidemic associations by evaluating historical (pre-epidemic) data, route of infection, severity of disease, meteorological data and seasonality, host animals, potential vectors, and vaccination coverage.

In order to assess the spread of anthrax outbreaks in Rhodesia in space and time, we performed a simple geospatial analysis using ArcGIS [ESRI, Redlands, CA, USA]. We defined an “outbreak” as an incipient focus of human anthrax cases that appeared in a district level hospital, which was the finest spatial resolution available in the data. These district hospitals had a patient catchment area of approximately 20 km².³ These cases were reported to a monthly temporal resolution. We georeferenced hospital locations using Wikipedia [<https://www.wikipedia.org>] and Google Maps [<https://www.google.com/maps>]. We then calculated the centroid (arithmetic mean) of outbreak locations for each time step in our series.³⁻⁵

Geospatial cluster analysis was performed using the Kulldorff space-time permutation model resident in SaTScan v9.3, where the objective was to identify space-time clusters within the context of the epidemic itself.¹⁷ Default settings were used for the analysis.

RESULTS

First recognition of the epidemic was in Nkai District, Matabeleland Province, in November 1978, with a low number of human cases reported until June 1979. All of the cases were associated with the butchering and skinning of local cattle. Nkai Hospital would later report over 500 cases from January 1979 to October 1980. Approximately half of these cases required hospitalization, with 17 fatalities (case fatality rate 3.3%). Eight of the fatalities were due to respiratory anthrax (47%). The remainder died of sepsis that followed a preceding cutaneous lesion. This may be considered the first phase of the epidemic.³⁻⁵

The epidemic smoldered until mid-1979, when a second phase of the epidemic was apparent. Case counts abruptly increased from September to November 1979 (Figure 2) ahead of the rainy

season peak in December-January (Figure 3).¹⁸ Geotemporal variation in epidemic progression was observed at the provincial level (Figure 4), where epidemic peaks were sequential across Matabeleland (November 1979), Midlands (December 1979), and Mashonaland Provinces (February 1980). Midlands Province was noted to begin peaking in October 1979 (Figure 5).³⁻⁵ This was suggestive of a general southwest to northeast progression. Evidence to support correlation of the peak of the rainy season with the provincial level peaks of human cases, which reportedly followed cases in cattle, was not apparent in the data (Figure 3). The patients were dominantly Tribal Trust Land inhabitants, with rare documentation of cases among those in the tanning industry.^{4,5} Over the course of five months (November 1979 to March 1980), explosive spread of anthrax in Mashonaland Province had encompassed 13 districts and 120,000 km².³⁻⁵

FIGURE 2. Rhodesian human anthrax cases from January 1979 to December 1980 for the provinces of Matabeleland, Midland, and Mashonaland.

FIGURE 3. Rhodesia climatology based on data from 1960-80. The anthrax epidemic in Rhodesia began in cattle during the dry season, followed by a dramatic surge in human cases observed ahead of the peak of the rainy season.

FIGURE 4. Human anthrax cases by province, noting the temporal shift in the peak of cases as the epidemic progressed across the country. A second wave of cases is observed in the data for Mashonaland Province.

FIGURE 5. Human anthrax cases in Que Que District, which experienced a prolonged peak from October 1979 to February 1980.

The Beatrice Road Infectious Diseases Hospital, located in Salisbury (now called Harare), saw 712 cases from January 1980 to June 1982 (Figure 6). These patients were referred to the hospital from across Mashonaland Province. All were Tribal Trust Land inhabitants except one individual from a tanning facility. Resource strain at the hospital was reflected in demand for

hospitalizations, was noted in January 1980 and again in July 1980, which was the result of two waves of patients who were hospitalized for two to five weeks. By March 1980, the Beatrice Road Infectious Diseases Hospital reported that the most common reason for admission to their hospital was anthrax. The number of cases seen at Beatrice Road was considered enough to prompt concerns about contamination of the hospital with anthrax spores, which prompted heavy use of masks, gowns and gloves until supplies were unable to meet the demand. However, no infection was noted among healthcare providers or between patients. Although many medical facilities reported abrupt, significant strain on their resources, fatality rates were considered very low and manageable. There was no report of antimicrobial resistance; rather, the vast majority of the patients were effectively managed with penicillin. There was no indication of a penicillin shortage.⁵

FIGURE 6. Human anthrax cases seen at the Beatrice Road Infectious Disease Hospital. This hospital, by March 1980, reported that anthrax was the leading reason for hospital admission. The facility experienced resource strain in two periods that coincided with two waves of patients in February 1980 and again in December 1980.

Inhalation, gastrointestinal, and meningitis presentations were documented at the Beatrice Road Infectious Diseases Hospital as well as additional hospitals.³ Five cases of anthrax meningitis, all fatal, out of 18 total cases seen were reported over the course of 12 months at Parirenyatwa General Hospital, also in Salisbury. This facility had not previously seen a single case of anthrax from 1970 to mid-1979. All of these fatal cases had cutaneous lesions that followed contact with cattle upon presentation. Several of these cases had reported a painful insect bite that preceded development of the classic anthrax eschar lesion. Death was observed within one week of initial symptoms. This experience prompted several case report publications because these presentations had previously been “extremely rare” in Rhodesia.^{5, 19, 20, 21} The unusual volume

and variety of clinical presentations led to comparisons at the time to the Sverdlovsk, U.S.S.R. outbreak of anthrax.⁷ The Sverdlovsk outbreak was later shown to be an accident of a biological weapons laboratory.²²

Figure 7 displays where human cases were first recognized, at the finest temporal and spatial resolution the epidemiological data allowed (i.e. monthly and district hospital coordinate). After June 1979, the epidemic spread eastward to Que Que District, Midlands Province, then erratically in the context of a “puzzling hop[ping]” phenomenon, whereby anthrax cases appeared in multiple foci with non-contiguous involvement of the land between.³ We attempted to further elucidate the “hop[ping]” phenomenon by using a combination of centroid distance measurements and the Kuldorff statistic. The Kuldorff space-time permutation highlighted five clusters of increased relative risk, as shown in Table 1 and displayed in Figure 7. Under the Kuldorff statistic, clusters associated with non-significant P-values are still associated with increased relative risk for high anthrax activity. The map corresponding to November 1979 in Figure 7 is an example of hops, which involved non-contiguous spread to Filabusia, Umzingqane, and Bembezi Districts from Nkai and Que Que Districts. Another example of a hop was in March 1980, with spread to Chilimanze, Charter, and Seluweke. These hops involved distances of 40-50 km. According to Davies, “The intervening commercial and communal areas did not report any cases at this time.” The context of these observations was during the second phase of the epidemic, when explosive spread was noted to involve 120,000 km² within 5 months in Mashonaland Province.³

FIGURE 7. Geospatial time series progression of the anthrax epidemic in humans. The mean of outbreak coordinates is indicated as a centroid coordinate, where each time step is associated with spatial movement of that coordinate. Geospatial time cluster periods associated by the Kuldorff statistic with higher relative risk for high anthrax case counts are shown and denoted with a

capital letter (A-E). According to Davies, the spread of anthrax in humans involved a non-contiguous, heterogeneous distribution pattern.

Coordinates	Radius	Time Frame	Number of Outbreaks	Expected Outbreaks	Observed / Expected	Test Statistic	P-Value
18.469056 S, 29.444797 E	59.37 km	Sep-79	3	0.26	11.5	4.67	0.055
18.999396 S, 28.901210 E	32.95 km	Nov-78 to Dec- 78	2	0.087	23	4.4	0.24
17.507240 S, 30.975851 E	58.94 km	Jan-80	4	0.61	6.57	4.27	0.25
20.916667 S, 28.466667 E	83.34 km	Feb-80	3	0.43	6.9	3.3	0.79
19.674956 S, 30.003541 E	85.84 km	Jun-80	2	0.22	9.2	2.69	0.992

TABLE 1. Results of the Kuldorff space-time permutation analysis. The first geospatial time cluster identified in the data was September 1979, which was statistically significant. The remaining clusters, while not statistically significant, were associated with increased relative risk.

Mapping of the data neither supported nor refuted Tribal Trust Lands as the dominant areas involved, however may be inferred based on the 20 km² patient catchment area and report that the cases originated on the Tribal Trust Lands.³ Urban hospitals, such as Beatrice Road Infectious Diseases and Salisbury Hospitals, were referred patients from distant Tribal Trust Lands, which was indicative of the resource strain placed on the district hospitals.⁵

Up to the point of the epidemic, an average of 20 cases were seen in livestock annually.¹¹ In the pre-1980 time period, systematic animal disease surveillance was not performed, especially on the Tribal Trust Lands. The epidemic among humans began nearly exclusively through contact with cattle across all areas of involvement in Rhodesia. In Lupane, within the epicenter, greater than 5,000 head of the total cattle population (5%) had died.^{11, 23} National statistics for the total number of anthrax-related cattle deaths were unavailable, but one report cited “many thousands”.¹¹ Up to 50% of cattle herds died in some communities.²⁴ After conducting a full literature review, we were unable to document reports of anthrax in any of the neighboring countries during the time of the Rhodesia epidemic. There was no report of any other animal species involved in the epidemic, with the exception of the rare, occasional domestic goat²³. The

first documented appearance of anthrax in the wildlife of Zimbabwe was in 2004.¹⁵ Because no point source could be identified, introduction of anthrax to cattle via feed or fertilizer was hypothesized.²³ Access to safe drinking water on the Tribal Trust Lands for both humans and cattle became a serious concern.⁴

During the course of the epidemic, veterinary control measures had waned in the context of the Rhodesian Counterinsurgency. Anthrax vaccines were acquired in response to the epidemic, but supply was limited.¹¹ The response campaign was considered ineffective due to poor utilization by the effected communities, due to pressure from insurgent forces to not cooperate with the national veterinary service.²⁴ After the war in 1981, vaccination services resumed and reached 70% of the national cattle herd by 1985.¹¹

Substantial underreporting of human cases by as much as 50% was inferred given the number of district hospitals and medical facilities who responded to Davies' survey and the likelihood that many patients either chose not to be evaluated by a medical facility or did not survive long enough for evaluation, as was observed in prior anthrax outbreaks in Rhodesia.^{3,13} Ongoing conflict over much of the anthrax-involved areas also impeded communications.²⁵ This represents an important source of potential bias when examining the results of the space-time permutation model described above.

We found that Davies' text description of the total human cases equating to 10,738 to be in discrepancy with the epidemiological data presented in his manuscripts' tables for Midlands, Matabeleland, and Mashonaland Provinces from January 1979-December 1980, which collectively reported a total of 17,068 cases and 101 fatalities.³ In terms of overall case counts for the epidemic, we found this to underestimate the true extent of the epidemic as well, given:

- Data reported by Davies did not include summary statistics for the involved areas of Manicaland and Victoria Provinces, however was briefly discussed in his Beatrice Road manuscript;³⁻⁵
- Beatrice Road Infectious Diseases Hospital in Harare, Mashonaland Province reported 131 additional cases and 4 fatalities (0.03% fatality rate) from January 1981-June 1982;⁵
- Kubuch et al reported anthrax cases in Lupane extending through 1984;²³ and
- Statistics reported by Davies for Mashonaland Province indicated a second epidemic peak in December 1980 without evidence of transmission resolution (i.e. incomplete data).³⁻⁵

Based on inclusion of this new data we found an overall discrepancy of 10,738 versus 17,199 cases and over 200 fatalities versus 105 fatalities if only the data tables from Davies' manuscripts are considered. We were unable to account for those patients on the rural Tribal Trust Lands who were infected, unable to seek appropriate medical care, and be documented in the epidemiological reports. A larger number of subclinical cases and exposures were likely based on observations of the low relative infectivity of anthrax spores in humans.²⁶ Compared to annual baselines prior to this epidemic, this was at least more than 1,400 times above expected annual baselines in the half-century prior to this epidemic. Based on the observation that the majority of these human cases were Tribal Trust Land residents, we estimate the attack ratio to have been 17,199 cases in the rural farming population of 800,000 (2.1%) versus the overall Tribal Trust Land population of at least 4 million (0.43%). Based on an estimated 1 human cutaneous case to 10 infected cattle carcasses, we estimate the true number of infected cattle to be 171,990.²⁶ When attempting to discern the duration of the event, it is apparent the epidemic began in November 1978 and extended beyond 1980, to at least December 1984. This too raises questions regarding additional unreported cases that occurred beyond the purview of Davies' original reports.

Overall, we estimate the epidemic covered approximately 245,750 km², using a minimum convex polygon around all outbreak locations in our dataset. We acknowledge the actual land area was likely less due to the non-contiguous spread reported by Davies, where true involvement of the land was not spatially contiguous.

DISCUSSION

The unusual nature of the epidemic and how it became manifest in Rhodesia was the subject of several papers. Davies emphasized the pattern of case counts in relation to seasonal precipitation and tabanid (horsefly) counts as a way of explaining the abruptness and magnitude of the epidemic's spread possibly due to a combination of flooding and biting arthropods.⁵ This hypothesis was supported by multiple accounts by patients of having received a painful insect bite that later evolved into an eschar.⁴ There were no available observations of tabanids biting cattle, however this may be assumed because tabanid blooms coincided with the appearance of bovine anthrax.²⁷ This initial observation has been met with a high degree of debate over the years, with Kobuch et al remarking in 1989:

*In Zimbabwe, the Tabanidae multiply seasonally during the rainy summer months (October to April). They will settle and feed on carcasses of dead animals or on open wounds of the living and they readily bite humans, horses, cattle, and other livestock. The data for the tabanids derive from a detailed study carried out in 1973 and it has to be assumed that similar patterns occur annually. While the rise-fall-rise patterns of tabanid species counts parallel the numbers of anthrax cases, the concept of an association between biting flies and the incidence of anthrax remains a subject of controversy.*²³

In 2004, blowflies were later shown to be an important vector in Rhodesia during the context of a multi-species epidemic. The evidence that supported this hypothesis was the observation that browsers (kudu) versus grazers were predominantly infected. This was due to blowflies feeding on anthrax-infected carcasses and then regurgitating live *Bacillus anthracis* on leaves at grazing height versus directly on the ground.¹⁵ Davies suggested the possible involvement of vultures in the transmission cycle, but this hypothesis was not supported by contemporary studies.^{5,28}

An alternate hypothesis was proposed by Meryl Nass, an American physician living in Zimbabwe at the time who suggested the epidemic was propagated intentionally. She emphasized the unusual features of the epidemic: large numbers of cases, geographic extent and involvement of areas that had never reported anthrax before, lack of involvement of neighboring countries, specific involvement of the Tribal Trust Lands versus European-owned agricultural land, and coincidence with an ongoing civil war.²⁹ Despite others supporting Nass' provocative debate^{30,31}, no testimony from actors directly involved in the Rhodesian Counterinsurgency has been offered to support this assertion. However, witness testimony from Tribal Trust Land inhabitants revealed a belief that "poisoning" by anthrax occurred during the Counterinsurgency.^{32,33}

A review of Figure 7 shows the epidemic generally avoided what Rhodesian forces considered Vital Asset Ground during the Counterinsurgency however this observation was not universal. Vital Asset Ground was generally defined as the center ovoid area of the country consisting of non-Tribal Trust, European-owned land that ran from the southwest to the northeast. Joseph Nkomo's Soviet and Cuban-backed ZIPRA armed forces were based to the west of Rhodesia, and began an escalation of attacks into Matabeleland Province in 1978. In September 1978, a Soviet ground missile launched by ZIPRA forces downed a civilian Air Rhodesia Viscount. Rhodesian media had reported that 10 of 18 survivors were massacred on the ground by ZIPRA forces after the plane crashed, provoking a dramatic escalation in the conflict. Soon thereafter, Maoist ZANU

forces led by Robert Mugabe in turn escalated their attacks to the east in Mashonaland Province. All of Rhodesia was subsequently involved in conflict with ZANLA and ZIPRA guerilla forces that expanded their infiltration of the country from 8,952 to 11,183 personnel (25% increase) from December 1978 to January 1979. Infiltration routes spanned nearly every sector of border in the country during this period and exploited access to the resources and support of the Tribal Trust Lands as the insurgents ranged inwards towards the center of the country.²⁵ The first phase of the epidemic of anthrax began in November 1978 in Nkai, Matabeleland Province. The second phase of the epidemic, which was focused on Mashonaland Province, escalated dramatically in late 1979.³⁻⁵ The Kuldorff statistic identified clusters of high anthrax activity in November 1978, June and September 1979, and January and February 1980.

Bovine and human anthrax had been reported in years past in Nkai, Que Que, and many other districts involved in the epidemic.³ There are notable observations, however, that confound an explanation regarding the unusual nature of this epidemic within the context of modern history:

- There was no report of anthrax in wildlife in Rhodesia / Zimbabwe until 2004.¹⁵
- No point source of anthrax was identified.²³
- The volume of human anthrax cases reported here has not been previously reported anywhere in the world.
- The scale of cases in cattle and humans and associated morbidity and mortality was suggestive of low indigenous herd immunity.^{1,34}
- Anthrax was previously not observed to transmit so pervasively in Rhodesia as far back as 1898, when neither antibiotics nor vaccines were available.¹² This is despite the well documented presence of both cattle and humans dating back to the early 1800s.²

- There has, to-date, been no recorded instance in global history of a combined bovine-human anthrax epidemic of this magnitude attributed to an interruption of a vaccination control program.
- The observation of all clinical forms of anthrax within such a small time period has not been previously documented in world history.^{3-5, 7}
- There were no reports of unusual population densities of tabanid or other families of candidate vector arthropods present at the time.⁵
- There were no reports of unusual meteorological activity at the time.^{23, 35}
- Multiple physicians, veterinarians, public health and biodefense professionals focused their attention on an explanation of the unusual epidemiological features of this event.^{3-5, 7, 11, 12, 19-21, 23, 27, 29}
- Multiple independent sources reported the use of chemical and biological weapons by Rhodesian forces.²⁹⁻³³ The appearance and clustering of anthrax in humans geotemporally mirrored escalation in conflict with ZIPRA and ZANLA forces.²⁵

The sole possible exception to some of the above observations is the purported anthrax epidemic of 1770 in Haiti (then-Saint Domingue) that killed 15,000 indigenous and European people over a period of six weeks. The dominant clinical form of anthrax in Haiti was hypothesized to be gastrointestinal following the distribution of contaminated meat.³⁶ In comparison, the Rhodesia epidemic occurred over a longer time frame, with involvement of non-contiguous areas, and with several waves of transmission (see Figure 7).

Contiguous, local spread of anthrax in humans was observed to be due to a stressed African indigenous population slaughtering their cattle for food and engaging in local commerce with the contaminated meat.^{3, 4, 15} This was a contributing factor in the context of food insecurity during

the war. The areas of anthrax involvement spanned multiple indigenous tribes across areas of intense conflict where the roadways were mined and travel was restricted for the general public. ZANLA and ZIPRA forces often fought each other as well as Rhodesian forces and were supported by different indigenous communities.¹⁵ In all of the district hospitals, reported human cases had slaughtered local cattle prior to noting their own illnesses, where the majority of cases were initially cutaneous presentations.³⁻⁵ An apparent high fatality rate among cattle is suggestive of either exposure through inhalation or gastrointestinal routes.¹ It remains unclear how the cattle were infected across such vast geography. It may be hypothesized that transport of live cattle, as was often observed when indigenous African owners sought to avoid official scrutiny during peacetime, could have also contributed to spread. We propose this was unlikely to account for the distances of the different novel foci that appeared in the various locations of the country due to a combination of 1) tribal boundaries and ownership and 2) restrictions to population movement due to land mines and enforcement of movement controls by authorities during the war. Therefore, while local trade of infected meat or cattle movement likely played some role in human exposures, it does not explain the spread of anthrax across such vast geography.

Based on the evidence provided by Davies and the results of this study, we hypothesize multiple, independent geotemporal foci of anthrax introduced via an unknown mechanism rather than re-emergence from native endemic foci, as shown in Table 1 and Figure 7. This supports the hypothesis of introduced anthrax through feed or fertilizer proposed by Kobuch et al.²³ We propose that anthrax did exist in Rhodesia in small endemic soil borne foci, likely introduced via cattle during the colonial period.² From 1978 to 1980, anthrax was introduced at multiple times and places through an unidentified mechanism. Humans were predominantly exposed through direct contact with cattle in the context of food insecurity. Local trade of contaminated meat and tabanids contributed to further localized spread. Blowflies and vultures likely played a minimal role at that time. Widespread environmental contamination of soil and water sources has resulted

in latter-day outbreaks in wildlife, facilitated to a minor extent by blowflies, tabanids, and vultures.

In the days following Mugabe's 1980 ascent to power in Zimbabwe and the departure of European leadership, ongoing anthrax activity has been noted in livestock and humans, with first documentation of a multi-species epizootic in wildlife.¹⁵ It is curious why anthrax has not exhibited explosive transmission again, as observed in the late 1970s and early 1980s, particularly when other recent epidemics such as cholera in 2006 demonstrate the chronic state of public health infrastructure collapse in Zimbabwe.³⁷ Indeed, it is curious why anthrax has not exhibited explosive transmission anywhere in the world as was observed in Rhodesia.

No definitive explanation to-date has been offered to explain the many unusual features of this epidemic. We acknowledge that lack of data regarding several of the observations related to this epidemic provides important sources of bias in this study. Despite the noted limitations of the data associated with this important historical event, we propose a need for further scrutiny of the etiology of this epidemic, which contributed to substantial human suffering and the destruction of a once-vibrant national agricultural economy.

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DISCLOSURES

The authors have no conflicts of interest to disclose.

REFERENCES

1. De Vos V and PCA Turnbull. Anthrax. Coetzer JAW and Tustin RC, eds. *Infectious Diseases of Livestock*. Oxford, UK: Oxford University Press, 1788-1818.
2. Mwatwara W. A history of state veterinary services and African livestock regimes in colonial Zimbabwe, c. 1896-1980. Dissertation presented for the degree of Doctor of Philosophy (History) in the faculty of Arts and Social Sciences at Stellenbosch University. Available at <http://scholar.sun.ac.za/handle/10019.1/86424>. Accessed on December 24, 2015.
3. Davies JCA. A major epidemic of anthrax in Zimbabwe (Part I). *The Central African Journal of Medicine*, 1982. Vol. 28, No. 12, 291–98.
4. Davies JCA. A major epidemic of anthrax in Zimbabwe (Part II). *The Central African Journal of Medicine*, 1983. Vol. 29, No. 1, 8–12.

5. Davies JCA. A major epidemic of anthrax in Zimbabwe: the experience at the Beatrice Road Infectious Disease Hospital, Harare. *The Central African Journal of Medicine*, 1985. Vol. 31, No. 9, 176–80.
6. World Bank, 1986. Report No. 5878-ZIM: Zimbabwe Land Subsector Study.
7. Turner M. Anthrax in humans in Zimbabwe. *The Central African Journal of Medicine*, 1980. Vol. 26, No. 7, 160–61.
8. Whitlow JR. Land use, population pressure and rock outcrops in the tribal areas of Zimbabwe Rhodesia. *Zimbabwe Rhodesia Agricultural Journal*, 1980. Vol. 77, No. 1, 3–11.
9. Whitlow JR. Environmental constraints and population pressures in the tribal areas of Zimbabwe. *Zimbabwe Rhodesia Agricultural Journal*, 1980. Vol. 77, No. 4, 173–81.
10. Mossman SL and Mossman AS. Wildlife utilization and game ranching. IUCN Occasional Paper No. 17. Morges, Switzerland: 1976.
11. Lawrence JA, Foggin CM, Norval RAI. The effects of war on the control of diseases of livestock in Rhodesia (Zimbabwe). *The Veterinary Record (Rhodesia)*, 1980. Vol. 107, 82–85.
12. MacAdam I. Putting anthrax into perspective. *The Zimbabwe Rhodesia Science News*, 1980. Vol. 14, No. 2, 38–41.
13. Roberts CJ, Chambers, PG. An outbreak of anthrax in the Mondoro Tribal Trust Lands. *The Central African Journal of Medicine*, 1975. Vol. 21, No. 4, 73–76.
14. Mwenye KS, Siziya S, Peterson D. Factors associated with human anthrax outbreak in the Chikupo and Ngandu villages of Murewa district in Mashonaland East Province, Zimbabwe. *The Central African Journal of Medicine*, 1996. Vol. 42, No. 11, 312–15.
15. Clegg SB1, Turnbull PC, Foggin CM, Lindeque PM. Massive outbreak of anthrax in wildlife in the Malilangwe Wildlife Reserve, Zimbabwe. *Vet Rec.* 2007 Jan 27;160(4):113-8.
16. *Rhodesia 1:1,000,000 Land tenure map*. 3rd ed. Salisbury, Maryland: Surveyor-General, 1978.

17. Kulldorff M, Athas W, Feuer E, Miller B, Key C. Evaluating cluster alarms: A space-time scan statistic and brain cancer in Los Alamos. *American Journal of Public Health*, 1998; 88:1377-1380.
18. World Bank, 2015. Climate Change Knowledge Portal. Available at http://sdwebx.worldbank.org/climateportal/index.cfm?page=country_historical_climate&ThisRegion=Africa&ThisCCCode=ZWE. Accessed on December 24, 2015.
19. Levy LM, Baker B, Meyer MP, Crosland P, Hampton J. Anthrax meningitis in Zimbabwe. *The Central African Journal of Medicine*, 1981. Vol. 27, No. 6, 101–104.
20. Latif AS, Nathoo KJ. Scrotal anthrax in a child: a case report. *Ann Trop Paediatr*. 1983 Mar;3(1):47-9.
21. Jena GP Intestinal anthrax in man: a case report. *Cent Afr J Med*. 1980;26:253–4.
22. Meselson M, Guillemin J, Hugh-Jones M, Langmuir A, Popova I, Shelokov A, Yampolskaya O. The Sverdlovsk anthrax outbreak of 1979. *Science*. 1994 Nov 18;266(5188):1202-8.
23. Kobuch, W. E., Davies, J., Fleischer, K., Isaacson, M. and Turnbull, P. C. B., 1990. A clinical and epidemiological study of 621 patients with anthrax in western Zimbabwe, Proceedings of the international workshop on anthrax, *Salisbury Medical Bulletin*, 68 (supplement), 34–38.
24. Anonymous. Rhodesian Anthrax Spreads. *Daily News-Record*. October 16, 1979.
25. Cilliers JK. *Counter-Insurgency in Rhodesia*. Kent: Croom Helm Ltd, 1985.
26. Turnbull TCP. Guidelines for the surveillance and control of anthrax in humans and animals. Third edition. World Health Organization WHO/EMC/ZDI/98.6, 1998 [online]. Available from: http://www.who.int/csr/resources/publications/anthrax/WHO_EM_C_ZDI_98_6/en/. Accessed December 24, 2015.

27. McKendrick DR. Anthrax and its transmission to humans. *Cent Afr J Med*. 1980 Jun;26(6):126-9.
28. Mundy PJ, Brand FE. An investigation of vultures and anthrax in Southern Africa. *Rhodesian Veterinary Journal*, 1978. Vol. 9, 36–39.
29. Nass M. Anthrax epizootic in Zimbabwe, 1978–1980: due to deliberate spread? *Physicians for Social Responsibility Quarterly*, 1992. Vol. 2, No. 4, 198–209.
30. Mangold T, Goldberg J. *Plague wars*. 1st ed. New York: St. Martin's Press, 1999.
31. Martinez I. Rhodesian anthrax: the use of bacteriological and chemical agents during the liberation war of 1965-80. *Indiana International and Comparative Law Review*. 13(2): 447-479.
32. Alexander J, McGregor J, and Ranger T, Violence and memory: one hundred years in the 'dark forests' of Matabeleland. Portsmouth, NH: James Curry, 144-5, 172-9.
33. White L. Poisoned food, poisoned uniforms, and anthrax: or, how guerillas die in war. *Osiris*. 2004;19:220-33.
34. Cizauskas CA, Bellan SE, Turner WC, Vance RE, Getz WM. Frequent and seasonally variable sublethal anthrax infections are accompanied by short-lived immunity in an endemic system. *J Anim Ecol*. 2014 Sep;83(5):1078-90. doi: 10.1111/1365-2656.12207.
35. Unganai LS, Mason SJ. Spatial characterization of Zimbabwe summer rainfall during the period 1920–1996. *South African Journal of Science*, 2001. Vol. 97, 425–31.
36. Morens DM. Epidemic anthrax in the eighteenth century, the Americas. *Emerg Infect Dis*. 2002 Oct;8(10):1160-2.
37. Chirisa I, Nyamadzawo L, Bandaiko E, Mutsindikwa N. The 2008/2009 cholera outbreak in Harare, Zimbabwe: case of failure in urban environmental health and planning. *Rev Environ Health*. 2015;30(2):117-24. doi: 10.1515/reveh-2014-0075.

Coordinates	Radius	Time Frame	Number of Outbreaks	Expected Outbreaks	Observed / Expected	Test Statistic	P-Value
18.469056 S, 29.444797 E	59.37 km	Sep-79	3	0.26	11.5	4.67	0.055
18.999396 S, 28.901210 E	32.95 km	Nov-78 to Dec- 78	2	0.087	23	4.4	0.24
17.507240 S, 30.975851 E	58.94 km	Jan-80	4	0.61	6.57	4.27	0.25
20.916667 S, 28.466667 E	83.34 km	Feb-80	3	0.43	6.9	3.3	0.79
19.674956 S, 30.003541 E	85.84 km	Jun-80	2	0.22	9.2	2.69	0.992

TABLE 1. Results of the Kuldorff space-time permutation analysis. The first geospatial time cluster identified in the data was September 1979, which was statistically significant. The remaining clusters, while not statistically significant, were associated with increased relative risk.

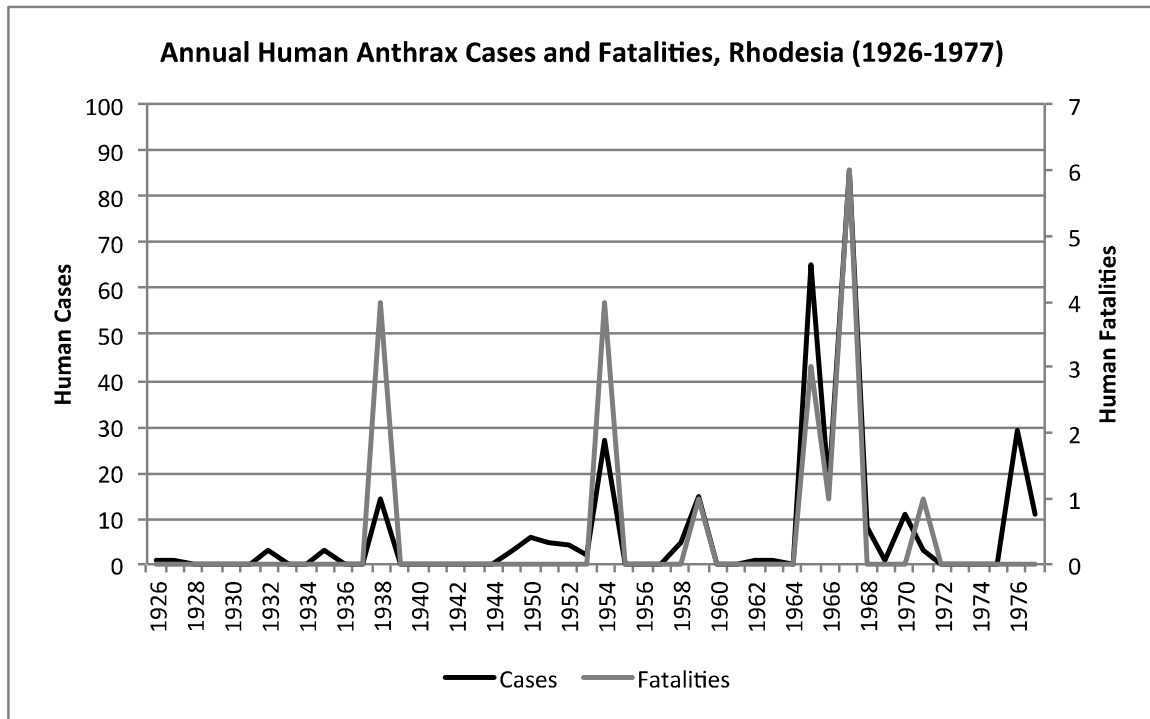


FIGURE 1. Historical anthrax human cases reported from 1926 to 1977. Vaccination for anthrax in cattle was not available until the mid-1950s.

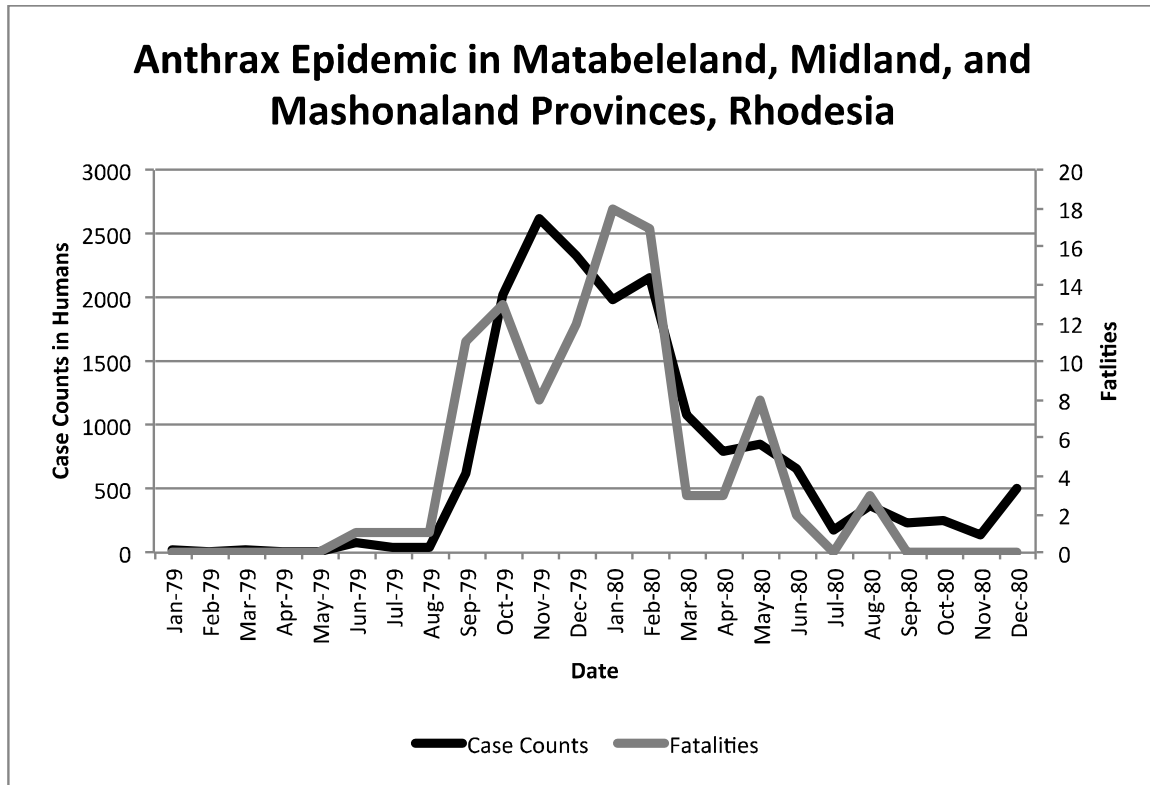


FIGURE 2. Rhodesian human anthrax cases from January 1979 to December 1980 for the provinces of Matabeleland, Midland, and Mashonaland.

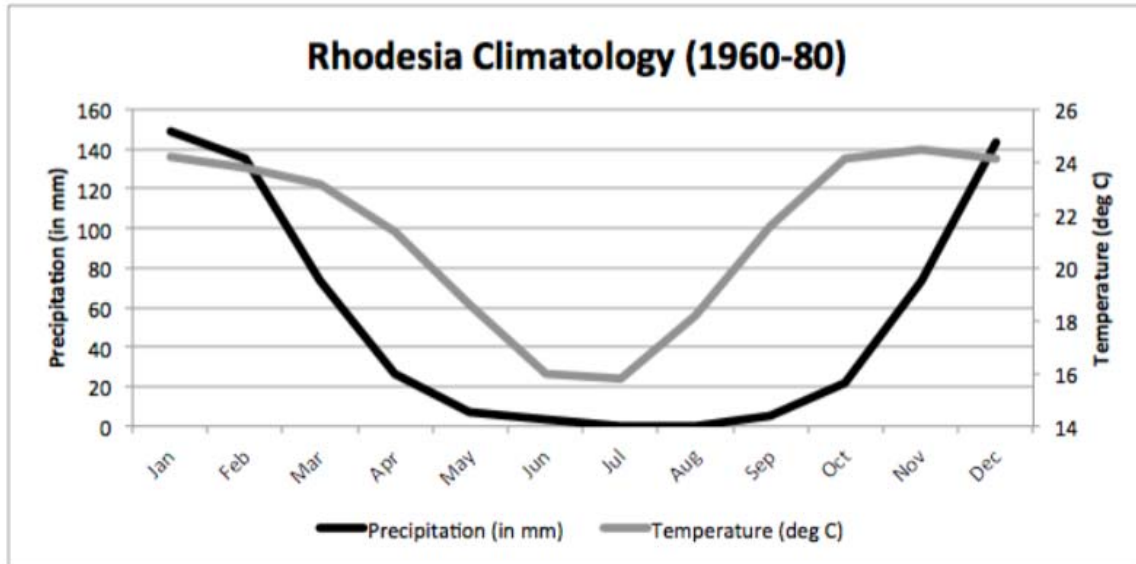


FIGURE 3. Rhodesia climatology based on data from 1960-80. The anthrax epidemic in Rhodesia began in cattle during the dry season, followed by a dramatic surge in human cases observed ahead of the peak of the rainy season.

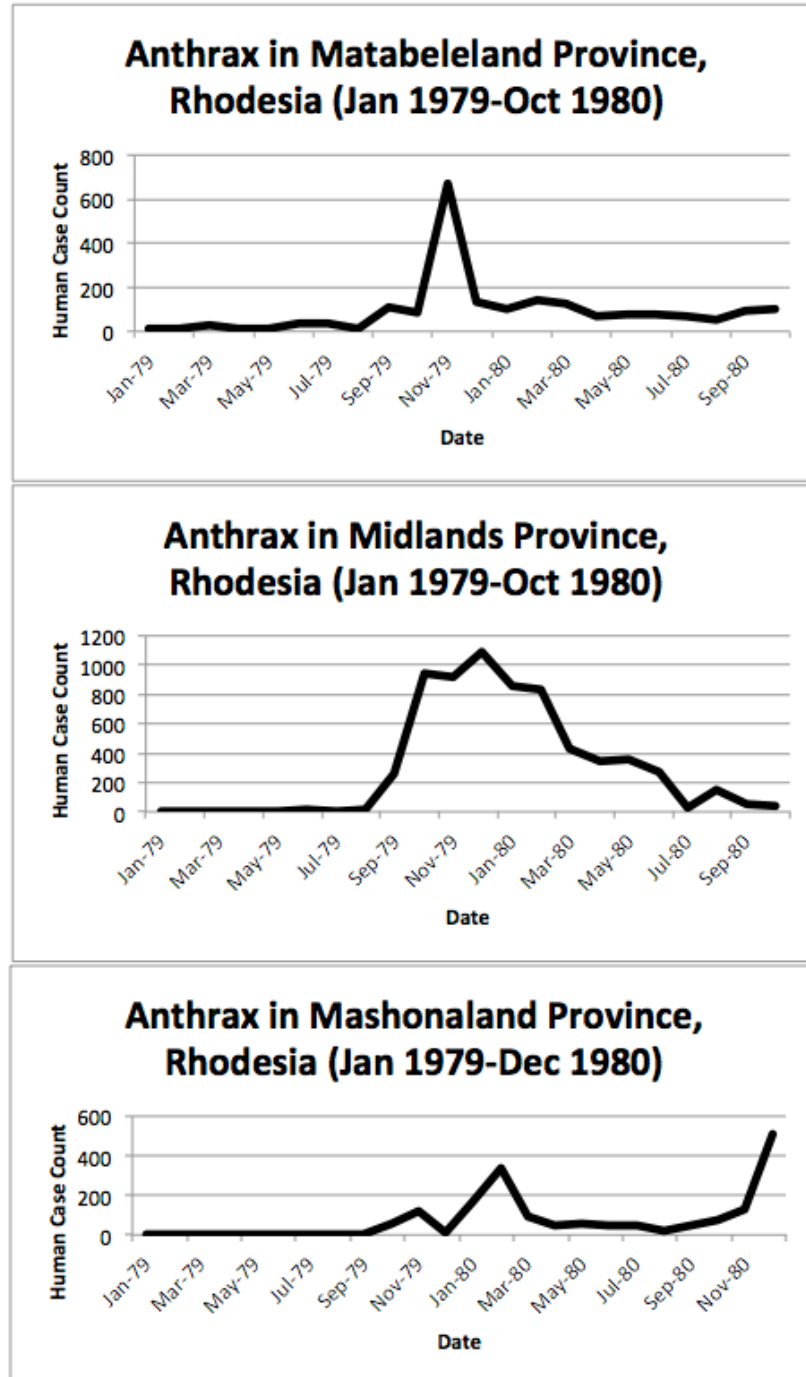


FIGURE 4. Human anthrax cases by province, noting the temporal shift in the peak of cases as the epidemic progressed across the country. A second wave of cases is observed in the data for Mashonaland Province.

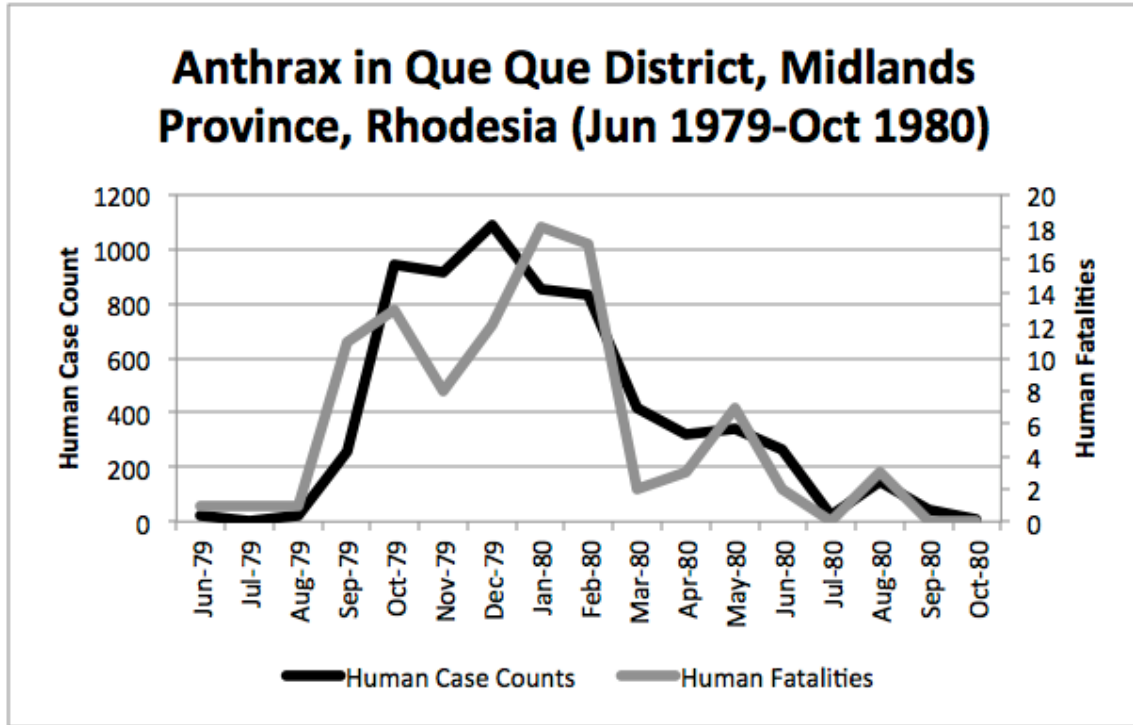


FIGURE 5. Human anthrax cases in Que Que District, which experienced a prolonged peak from October 1979 to February 1980.

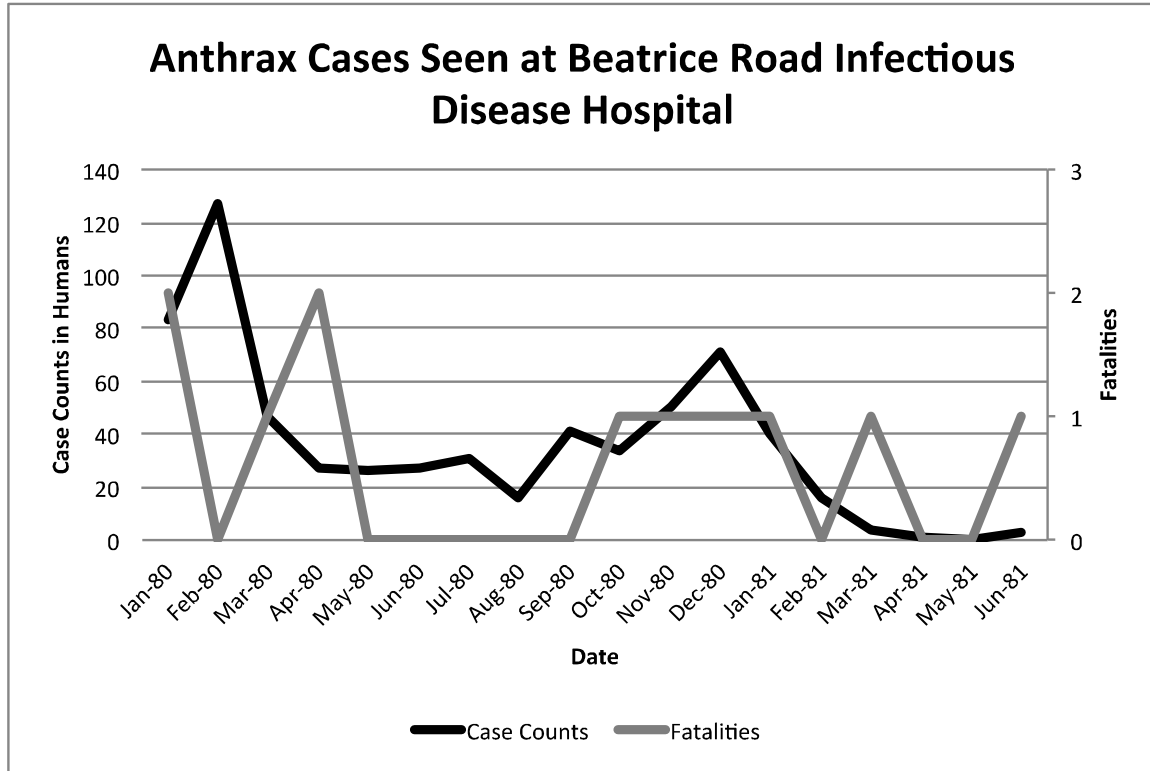
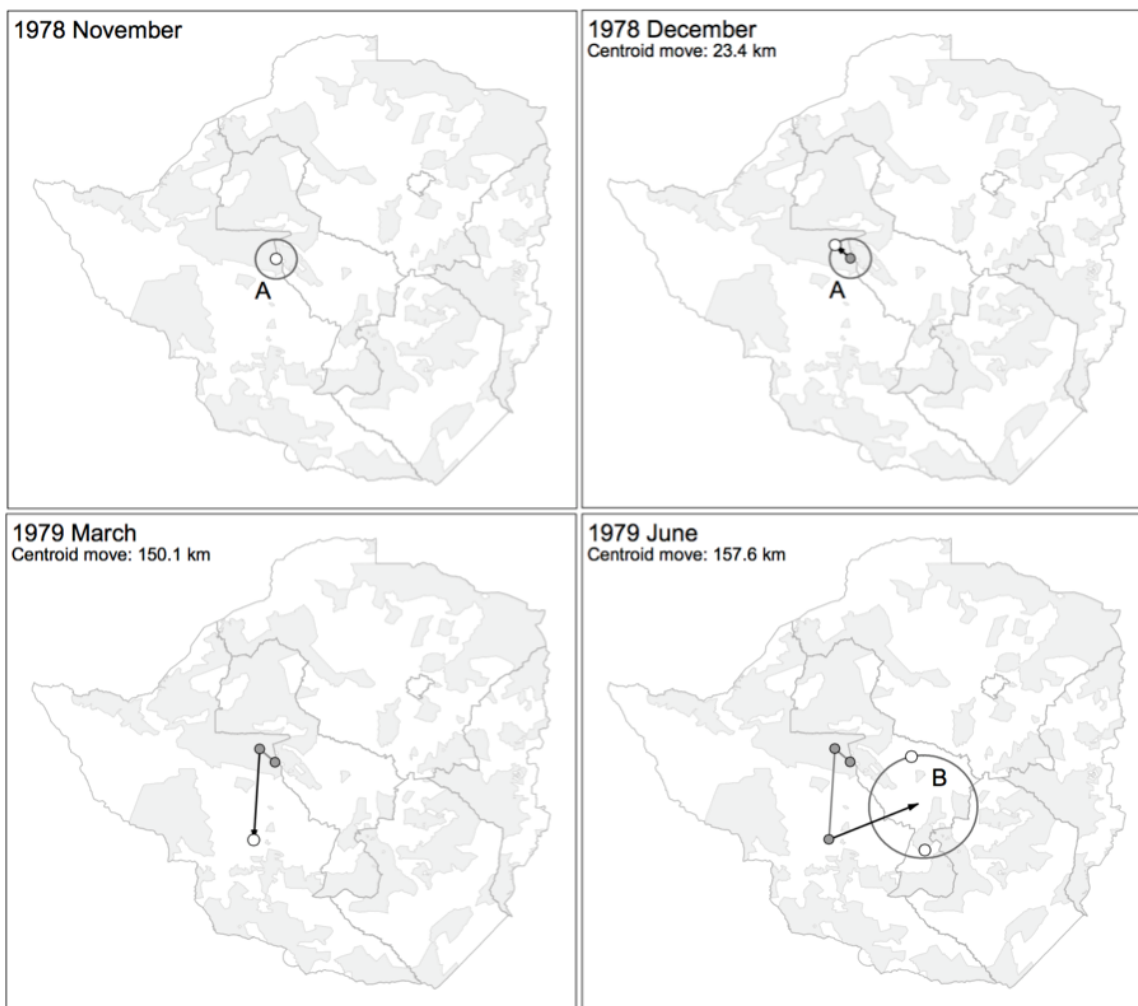
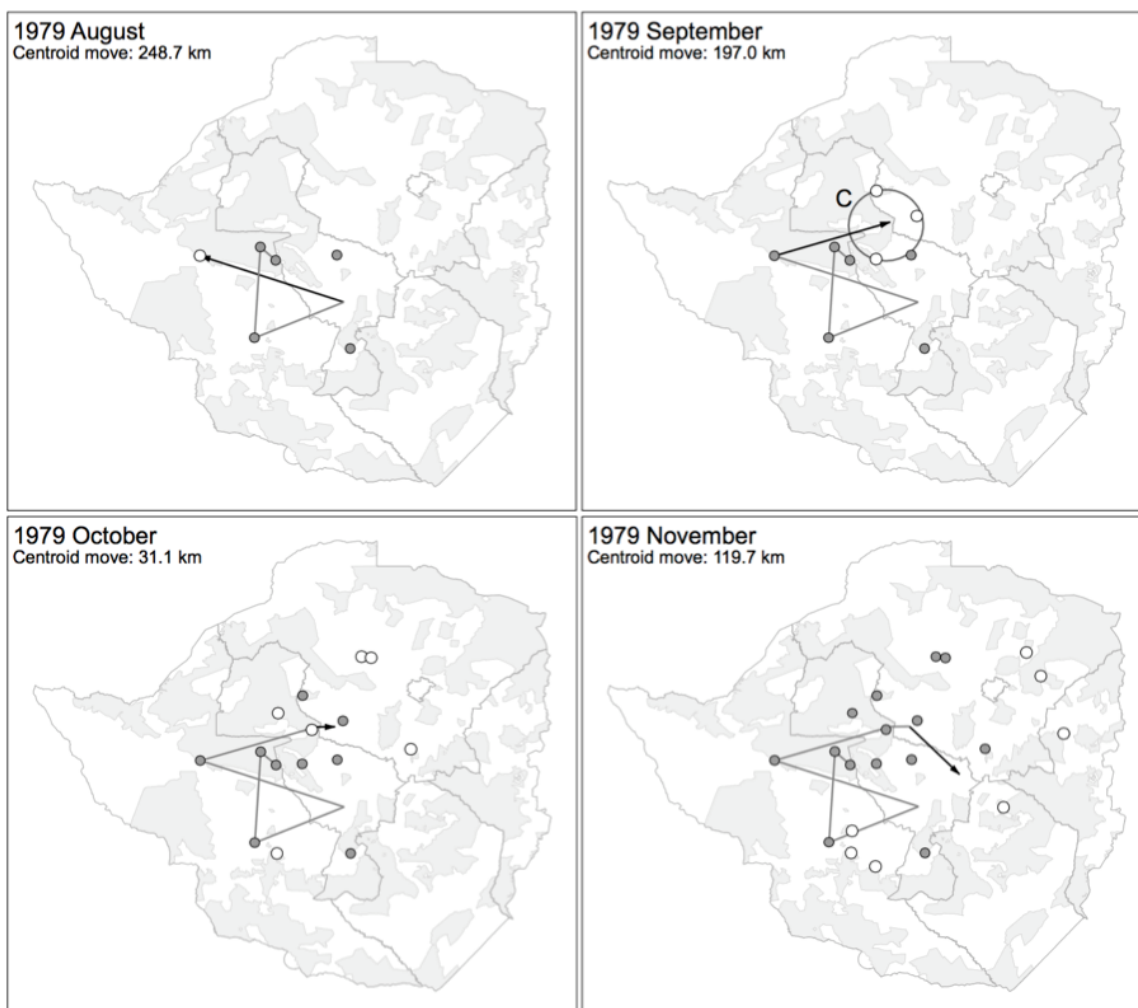
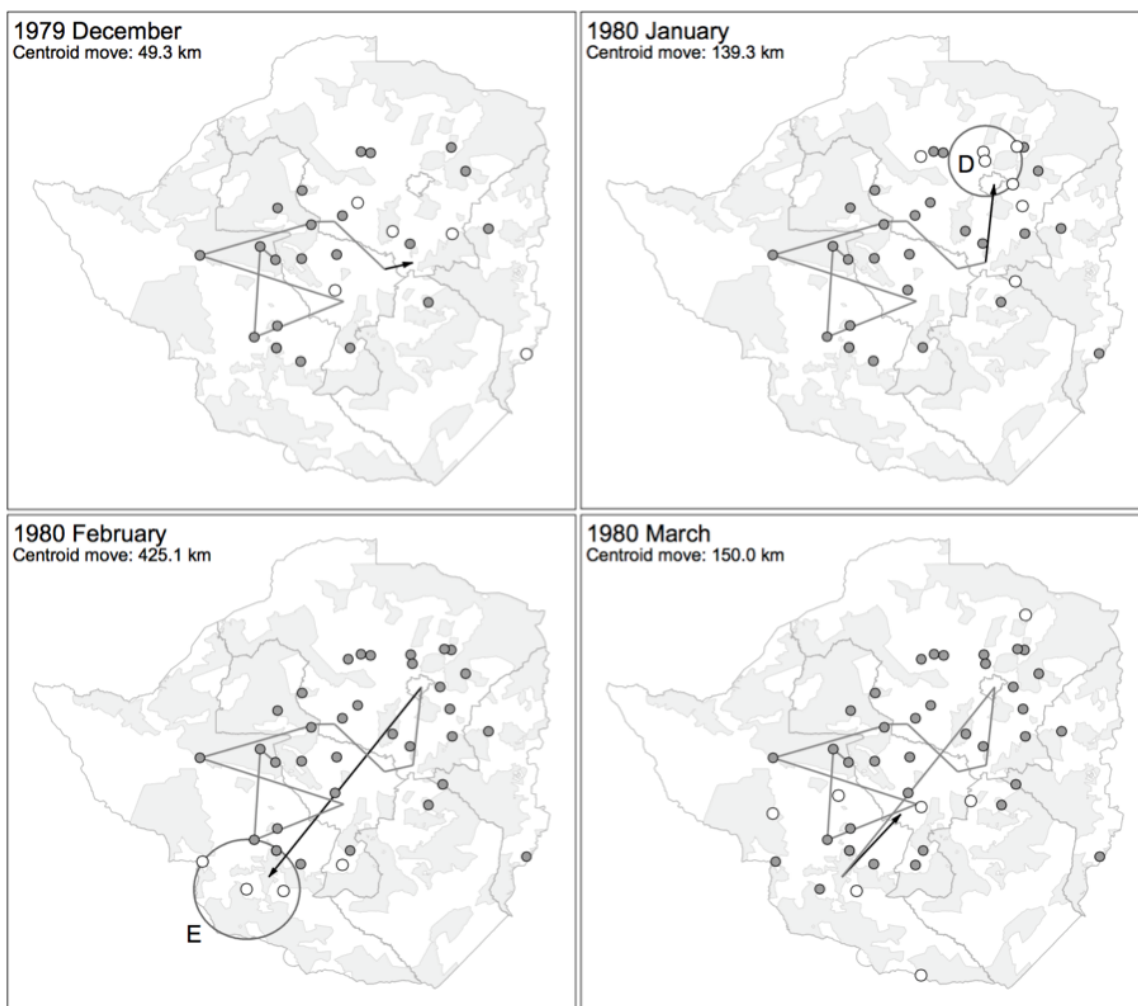


FIGURE 6. Human anthrax cases seen at the Beatrice Road Infectious Disease Hospital. This hospital, by March 1980, reported that anthrax was the leading reason for hospital admission.

The facility experienced resource strain in two periods that coincided with two waves of patients in February 1980 and again in December 1980.







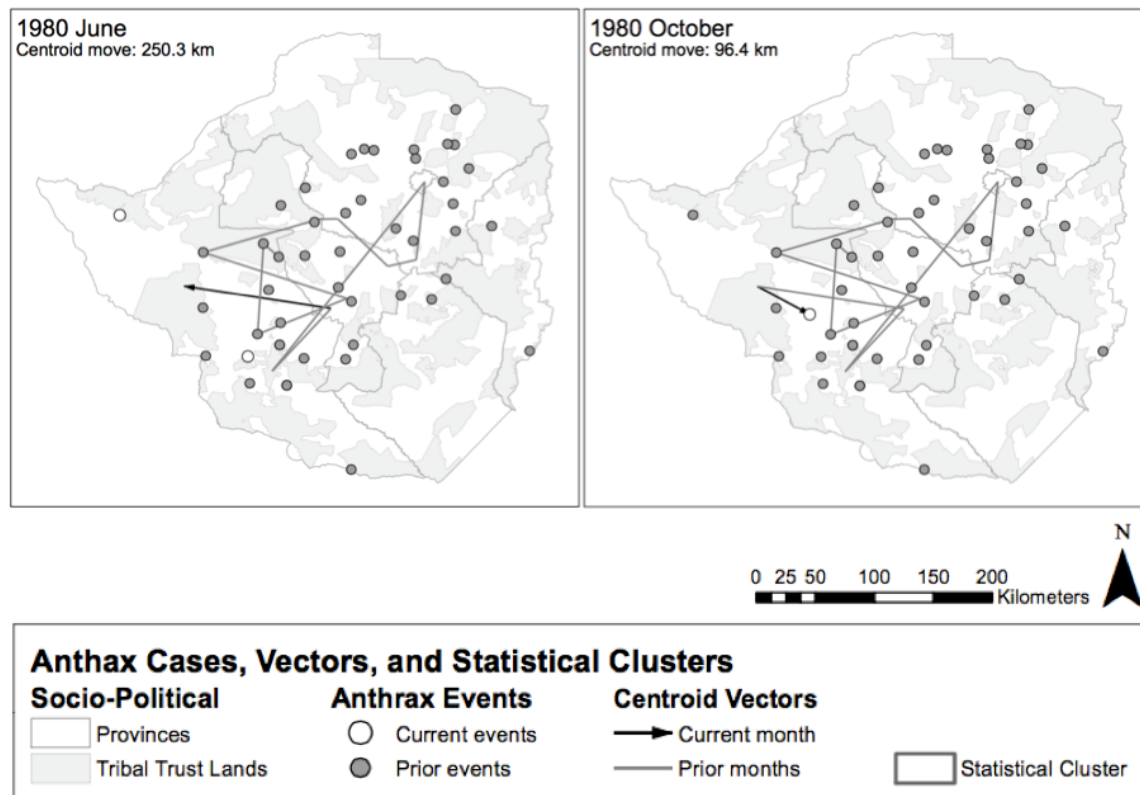


FIGURE 7. Geospatial time series progression of the anthrax epidemic in humans. The mean of outbreak coordinates is indicated as a centroid coordinate, where each time step is associated with spatial movement of that coordinate. Geospatial time cluster periods associated by the Kuldorff statistic with higher relative risk for high anthrax case counts are shown and denoted with a capital letter (A-E). According to Davies, the spread of anthrax in humans involved a non-contiguous, heterogeneous distribution pattern.