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Epizootiology and Ecology of Anthrax

Contributor

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Table of Contents

Summary

Anthrax is an acute, febrile disease of warm-blooded animals, including humans. Anthrax is caused by *Bacillus anthracis*, a gram-positive, non-motile, spore-forming bacterium, and it occurs most commonly as a rapidly fatal septicemia in animals. Anthrax outbreaks in animals in nearly 200 countries are recorded by The World Anthrax Data Site, a World Health Organization Collaborating Center for Remote Sensing and Geographic Information Systems for Public Health. Anthrax is a globally distributed disease, having been reported by all continents that are populated densely with animals and humans.

The number of cases of anthrax in animals has decreased significantly during the latter half of the twentieth century due to enhanced control and prevention programs which include efficacious vaccination. Approximately 25 outbreaks in the US were listed on The World Anthrax Data Site during the seven-year period from 1994 to 2000. Most of the outbreaks were in bovine species. The number of livestock deaths due to anthrax during any given year is typically one per one million animals-at-risk. However, anthrax is among the list of pathogens that could be used as a bioweapon. As such, there has been resurgence of interest in anthrax of humans and animals. Also, as the incidence of anthrax in livestock has dwindled, there have been fewer opportunities for veterinary health professionals to have personal encounters with anthrax and to maintain familiarity with various aspects of the disease.

This paper describes what is known about the ecology of anthrax, and it reviews several significant outbreaks of anthrax in livestock populations in the US and abroad. The epizootics reviewed herein included a period of at least 30 years (1971 to 2001), three continents (North America, Australia, and Africa), four countries/provinces (the United States, Canada, Namibia, Victoria) and two broad classifications of animals (domesticated livestock and wildlife). Anthrax epizootics in livestock and wildlife are restricted to specific geographical regions, regardless of continent, country, or geopolitical unit within a given country. Epizootics in livestock in the US are restricted to states west of the Mississippi River. Similarly, epizootics of anthrax in livestock and bison in Canada have been restricted to the western provinces.

Observations of the role of climatic factors such as season of year, ambient temperature, and drought in promoting anthrax epizootics have been made for several decades. The commonality of summer months, high ambient temperatures, drought and anthrax epizootics are non-contentious. The roles of environmental factors such as soil types and soil disturbances via excavation are poorly defined despite attempts to evaluate these potential factors. Formal analyses of risk factors for anthrax epizootics in the US have been only weakly informative due possibly to problems of recall bias, small sample size, and non-participation by livestock producers, among others. Quantitative analysis of risk factors for epizootics is one aspect of anthrax that can be pursued more aggressively.

A novel and potentially significant face of anthrax in the US is the host species. Mortality in exotic species was not evident in reports of anthrax prior to 2001. However, proportional mortality in white tail deer and other exotic species was very high during the epizootic in Texas in 2001. This change in affected host species appears to be driven by a social factor, not a biological factor. Its long-term epizootiological and epidemiological implications, if there are any, may be undeterminable at this time.

List of Figures

List of Tables

Introduction

Anthrax is an acute, febrile disease of warm-blooded animals, including humans. Anthrax is caused by *Bacillus anthracis*, a gram-positive, non-motile, spore-forming bacterium (Aiello, 1998). The name of the bacterium is derived from “anthrakis”, the Greek word for coal, because anthrax in humans causes black, coal-like lesions on the skin at the site of inoculation (Inglesby et al., 1999). Anthrax occurs most commonly as a rapidly fatal septicemia in animals. Thus, synonyms are charbon, splenic fever, and Milzbrand.

Anthrax outbreaks in animals in nearly 200 countries are recorded by The World Anthrax Data Site, a World Health Organization Collaborating Center for Remote Sensing and Geographic Information Systems for Public Health (Hugh-Jones, 2005). The types of data recorded by The World Anthrax Data Site are: country-of-origin, anthrax status, vaccination program, species affected, year of outbreak, number of outbreaks during the year, number of cases, number vaccinated, and total livestock population. Anthrax is a globally distributed disease, having been reported by all continents that are populated heavily with animals and humans. The anthrax status of a given country may be classified into one of six categories: hyperendemic/epidemic, endemic, sporadic, probably free, free and unknown. The countries with hyperendemic/epidemic status are more frequent in Africa, although the status of Egypt is “Probably free”. Examples of regions with unknown anthrax status are the polar extremes, the Arctic and the Antarctic.

The number of cases of anthrax in animals has decreased significantly during the latter half of the twentieth century due to enhanced control and prevention programs which include efficacious vaccination. Approximately 25 outbreaks in the US were listed on The World Anthrax Data Site during the seven-year period from 1994 to 2000. Most of the outbreaks were in bovine species; the ovine, caprine, and swine species were affected less frequently, and reports in horses are even rarer. The number of livestock deaths due to anthrax during any given year is typically one per one million animals-at-risk. However, anthrax is among the list of pathogens that could be used as a bioweapon (Noah et al., 1998; Inglesby et al, 1999; Inglesby et al, 2002; Centers for Disease Control and Prevention, 2005). *B. anthracis* is one of a limited number of organisms that can be used to inflict great biological and psychological harm on an affected human population. As such, there has been resurgence of interest in anthrax of humans and animals. Also, as the incidence of anthrax in livestock has dwindled, there have been fewer opportunities for veterinary health professionals to have personal encounters with anthrax and to maintain familiarity with various aspects of the disease. The objective of this paper is to describe what is known about the ecology of anthrax, and to review several significant outbreaks of anthrax in livestock populations in the US and abroad.

Ecology of Anthrax Spores

“For the control of [anthrax] epidemics, it is important to understand not only the pathogenesis and interactions of *B. anthracis* with host animals but also the **ecology of the spores**.” (Dragon and Rennie, 1995). Two microenvironments of geographic regions in which repeated outbreaks of anthrax occurred were described by Van Ness (Van Ness, 1971). These microenvironments, also referred to as incubator areas, were characterized by: (1) “low-lying depressions”, where standing water collected and devitalized plant life, and (2) “rocklands”, which are dried courses of water, or hillside seeps where organic matter accumulated during runoff. Geological studies of epizootic areas along the coastal regions of Louisiana and Texas indicated that outbreaks were associated most frequently with calcareous soils that also were rich in nutrients (Van Ness, 1971).

The ecology of anthrax spores is a controversial subject. The “incubator area” hypothesis is based on observations that alkaline soil pH, high soil moisture, high concentrations of organic matter, and ambient temperature in excess of 15.5^{o} C provides a microenvironment that promotes cycling of *B. anthracis* spores, the end result of which is an increase in the exposure of susceptible hosts to infective doses of spores and more outbreaks of anthrax (Van

Ness, 1971). However, the “incubator area” hypothesis has been challenged based on several pieces of evidence:

(1) Experimental investigations which show that the vegetative cells of *B. anthracis* have highly specific nutrient requirements (e.g., animal blood, viscera) and survive poorly outside of a viable host.

(2) The vegetative cells are highly susceptible to antagonism from other bacterial species that occupy the same environment.

(3) Growth of *B. anthracis* outside a host leads to a rapid decrease in its virulence. A decrease in its virulence would **not** favor an outbreak of anthrax.

There are numerous opportunities during an anthrax outbreak for spores to be dispersed over large geographic areas. Carnivores are less susceptible to anthrax than are herbivores. Consequently, carnivores may become sub-clinical carriers and disperse ingested spores (Dragon and Rennie, 1995). Avian scavengers (e.g. vultures, gulls, ravens) also may disperse widely the spores that adhere to their feathers as they feed on infected carcasses. Infected animals shed the spores in their feces and urine. Mosquitoes and tabanids have been implicated in mechanical transmission of spores from animal to animal, and from animal to vegetation. Water and wind are other vehicles of transmission.

Generally speaking, anthrax epizootics occur during the summer months in which there are dry periods that are punctuated by prolonged periods of intense rain. A proposed role of water in anthrax epizootics is the collection (aggregation) and concentration of spores in storage areas, formerly referred to as incubator areas (Dragon and Rennie, 1995). Prolonged rainfall promotes runoff and pooling of standing water. The surface of *B. anthracis* spores is highly hydrophobic. Thus, the spores are resistant to dissolution by water and may be transported in clumps of organic matter by runoff to standing pools of water. Dry weather causes evaporation of standing water and concentration of floating anthrax spores as the water pools shrink. The high buoyant density of *B. anthracis* spores provides an opportunity for the spores to adhere to vegetation as the vegetation resurfaces during evaporation. Dragon and Rennie (1995) summarized the effects of water on anthrax spores essentially in three steps: (1) successive cycles of run-off and evaporation concentrates anthrax spores in storage areas, (2) evaporation redistributes the spores from soil onto vegetation, and (3) susceptible herbivores consume the contaminated vegetation.

The unique physiology of the anthrax spore protects it from harmful chemicals, disinfectants, and degradative enzymes in the environment (Dragon and Rennie, 1995). In general, anthrax spores consist of an integument and a protoplast. The outer, middle, and inner layers of the integument are the exosporium, coats, and cortex, respectively. The outer and inner layers of the protoplast are the inner-membrane and the core, respectively. Changes in pH in the environment reduce the water content of spores which, in turn, increases the buoyant density of the spores and increases their resistance to the destructive effects of heat and ultraviolet light. Van Ness was able to correlate alkaline pH with the microenvironments in incubator areas (Van Ness, 1971). In Van Ness’s investigations, the alkaline pH was attributed directly to calcareous soils. Calcium cations participate in germination of anthrax spores as well as maintenance of the dormancy of spores. The high calcium concentrations in the soil may help to maintain viable spores in the environment for long periods of time and increase the chances that susceptible hosts could be exposed to these spores. Regardless of whether calcium is needed by the integument of the spore to aid in germination, or by the core of the spore to maintain dormancy and resistance, calcium is thought to be vital to the spore-vegetative cell-spore cycle of all *Bacillus* species.

Ecology of Anthrax in Bison in Canada

Approximately ten anthrax epizootics in which at least 1,098 bison died in northern Canada were recorded between 1962 and 1993 **(Figure 01)** (Gainer and Saunders, 1989; Dragon and Rennie, 1995; Dragon et al., 1999).

Figure 01. Anthrax enzootic regions and surrounding environs in northern Canada. 1, Hook Lake; 2, Grand Detour; 3, Park Central; 4, Lake One; 5, Slave Point; 6, Boulogne Lake; 7, Falaise Lake; 8, Calais Lake; 9, Mink Lake. (*Source:* Dragon et al., 1999).

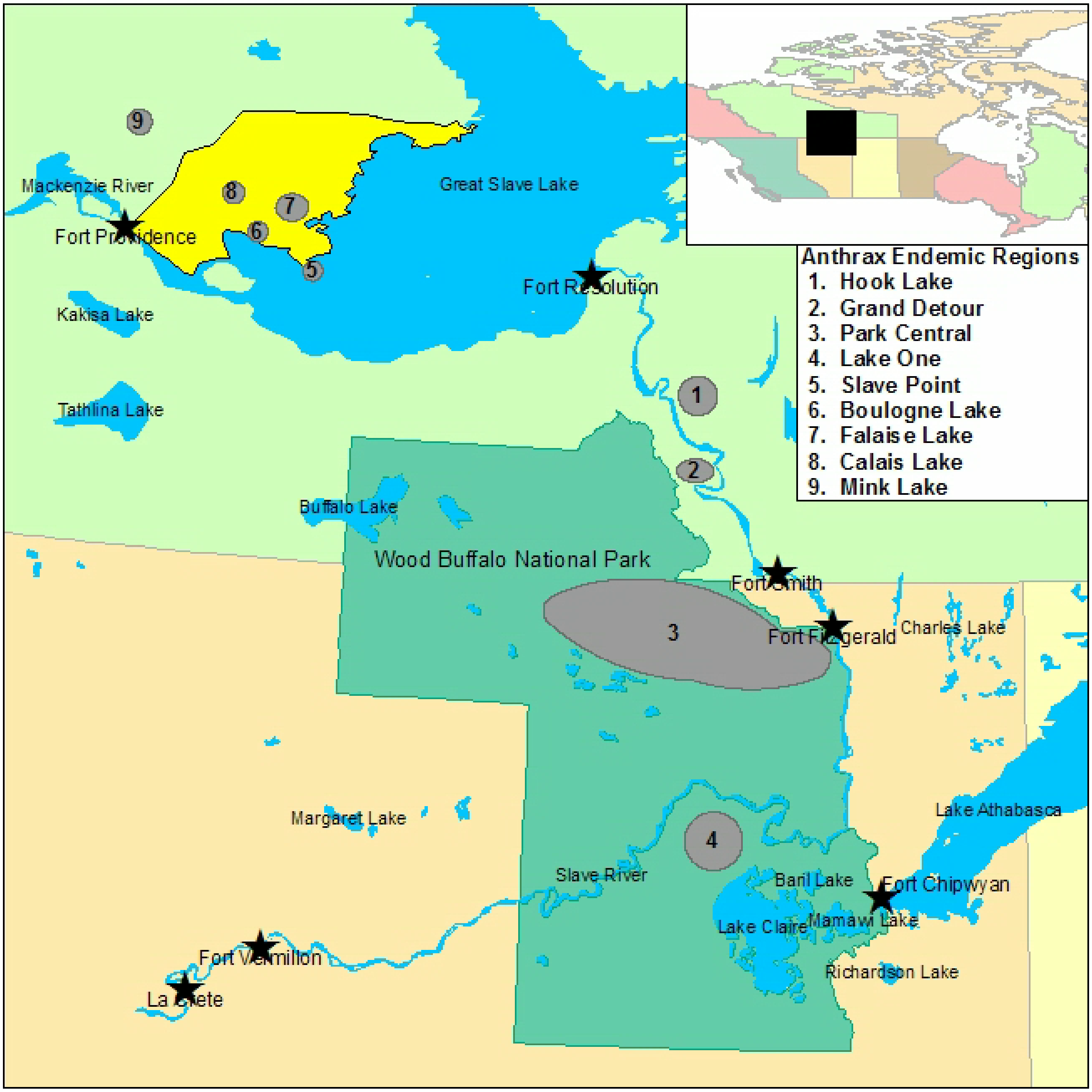


Figure 02. A bison engaged in wallowing during the bison rut, Custer State Park, South Dakota. (*Source:* http://www.igougo.com/photos/journal_photos/BisCus2(1).jpg).

The epizootics occur during hot, dry summer months of June through September, and are usually preceded by a wet spring. This period of time corresponds to the bison rut. Four factors that are associated rather consistently with these epizootics are high ambient temperatures, intense mating activity, high densities of insects, and high densities of bison as they congregate and compete for diminishing water and food supplies. Two hypotheses that are based on these four factors have been proposed to explain outbreaks of anthrax in bison in northern Canada: (1) “the modified host resistance hypothesis” and (2) “the wallow concentrator hypothesis” (Dragon et al., 1999). These two hypotheses are not mutually exclusive.

The modified host resistance hypothesis proposes that bison herds are persistently exposed to low levels of *B. anthracis* spores and they develop a subclinical infection prior to the outbreaks. It has been postulated that the four factors mentioned above may decrease the bison’s immunity to *B. anthracis*. Thus, a dose of spores that usually would be non-infectious not only may become infectious, but also may cause a lethal infection. If this hypothesis is true, then it also may explain the failed attempts to recover *B. anthracis* from the soil of anthrax-affected areas, both in Canada and in the U.S (Dragon et al., 1999). However, one other explanation for the failed attempts to recover *B. anthracis* from the soil is improper soil sampling techniques, not merely a low concentration of spores in the soil. An argument against the modified host-resistance hypothesis is that the epizootics generally <u>precede</u> the ruts and occur when bison bulls are in peak physical condition, after a summer of liberal grazing.

The wallow concentrator hypothesis proposes that anthrax spores in the environment are transported by spring run-off and floods to depressions in the earth’s surface, specifically “bison wallows” (Dragon et al., 1999). The bison wallows are circular depressions that are formed in open fields by bison bulls during the bison rut, and these wallows may be revisited by bison annually. During periods of drought, wallows are the last sites to retain standing water. The anthrax spores that become concentrated in the wallows would be encountered preferentially by mature bulls that use the wallows. Wallowing is a display of territorial behavior by bison, the frequency of which increases as the rut approaches **(Figure 02)**. In addition to the numerous acts of mating, bison bulls may be exposed to anthrax spores during aggressive acts such as stamping and wallowing, both of which may aerosolize the spores. Close grazing of sparse, devitalized vegetation is yet another possible route of exposure of bison to anthrax spores.

Insects have been implicated in transmission of *B. anthracis* from animal to animal, but their roles may vary depending on the host species. For example, the role of insects in transmission of *B. anthracis* among bison is thought to be minor because of the disparity in incident cases between bison bulls and bison cows. The incidence in bulls is higher than in cows. Because biting vectors are equally attracted to bulls and cows, the incidence of anthrax would be expected to be similar in bulls and cows, if vectors were a significant mode of mechanical transmission of *B. anthracis*.

Anthrax Epizootics in Livestock and Other Wildlife

Anthrax Epizootic in Louisiana, 1971

The Epizootic

An outbreak of anthrax in southeastern Louisiana during the summer of 1971 affected 10 parishes (Fox et al., 1973). The epizootic investigation was restricted to Ascension Parish, from which the earliest and greatest number of incident cases were reported **(Figure 03)**. Livestock morbidity and mortality data were acquired from the state livestock disease laboratory, interviews with veterinary practitioners, livestock producers, and county livestock agents. The first laboratory-confirmed case was reported on June 11, 1971. From June 11 through June 24, 75 farm operations within a five-mile radius of the index premises reported 700 livestock deaths that were suspected to be due to anthrax **(Figures 04, 05)**. The last case was reported on August 30, 1971. *B. anthracis* was isolated from 94 of the suspected 700 cases. After June 1, incident cases were more frequent outside Ascension Parish. Although the incident cases were

Figure 03. Geographical distribution by parish of 700 livestock deaths due to anthrax, Louisiana, 1971. *(Source:* Fox et al., 1973).

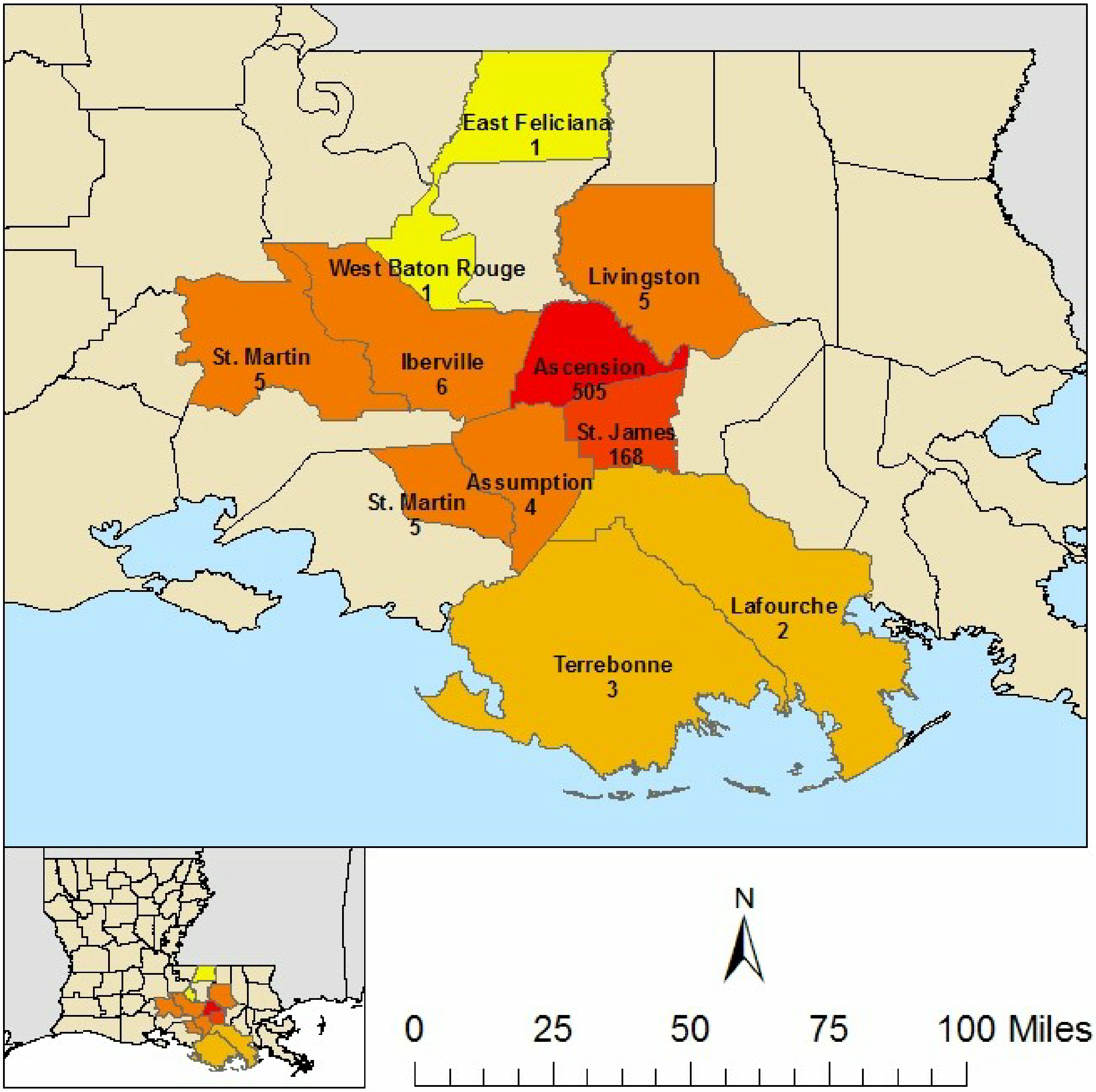


Figure 04. Week of onset of anthrax on 76 premises, Louisiana, 1971. *(Source:* Fox et al., JAVMA, 1973).

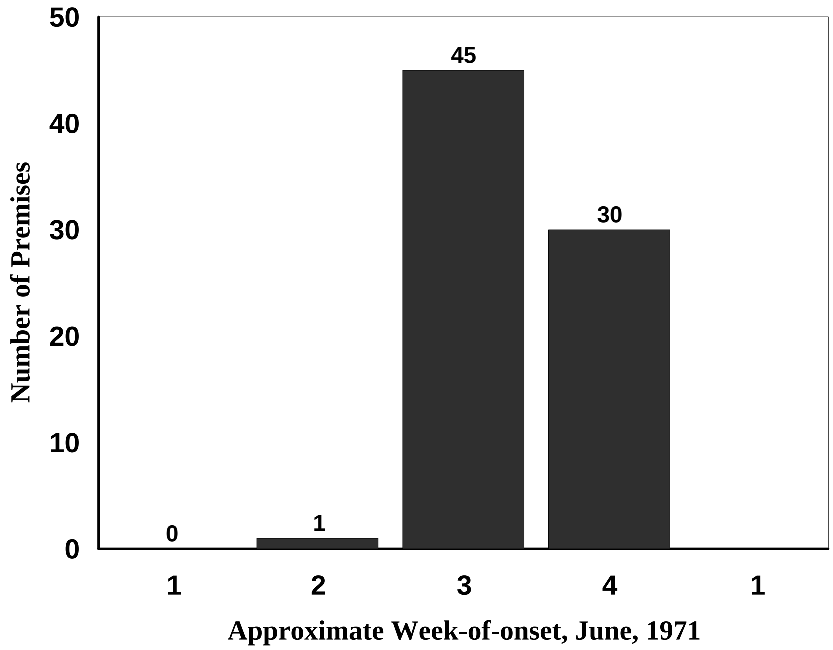
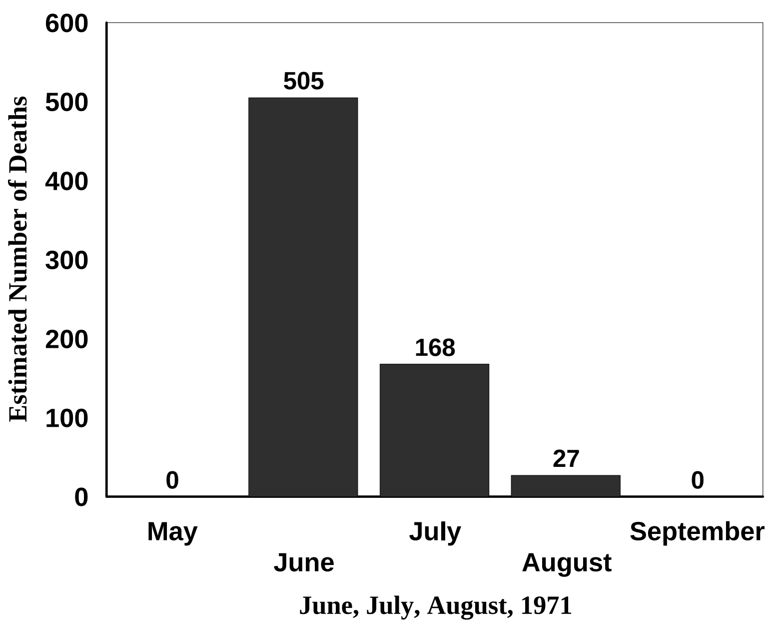


Figure 05. Monthly mortality due to anthrax on 76 premises, Louisiana, 1971. *(Source:* Fox et al., JAVMA, 1973).



reported from 10 different parishes, 96 percent of the cases were from two parishes only: (1) 72 percent from Ascension Parish, and (2) 24 percent from St. James Parish. Data were collected from the affected premises during three separate occasions: July, 1971, December, 1971, and August, 1972. The total number of confirmed and suspected cases was 636 cattle, 31 horses, 24 hogs, 4 mules, 3 dogs, and 2 goats. The species-specific mortality was greatest in horses at 12.5%, 17.4%, and 22.5% on each of the three separate occasions. The species-specific mortality in cows was 4.3%, 7.1%, and 9.1% on each of three separate occasions **(Figure 06)**. The proportional mortality was much greater in cattle than in horses **(Figure 07)**. Human morbidity and mortality data were acquired from the Louisiana State Department of Health. Two veterinarians who had done postmortem examinations on affected cattle contracted cutaneous anthrax. *B. anthracis* was isolated from one of the two veterinarians.

Management and Environmental Factors

Face-to-face interviews and telephone interviews were used to collect information about animal deaths, livestock management practices, insect populations, and other factors from producers on 23 affected premises. Similar information was collected from producers who occupied 16 non-affected premises **(Table 01)**. None of the factors were associated with the epizootic. In addition to these factors, neither feed supplements, nor antibiotics, nor sulfonamides had been fed to livestock on any of the premises. There were no herd additions to any of the premises within several months preceding the epizootic.

Ecological Factors

Geological data were obtained from the Soil Conservation Service, U.S. Department of Agriculture. Several distinct ecological zones are located in Ascension Parish. Anthrax mortality was associated with **all** soil types in the parish **except** the Calhoun-Olivier soils, for which the pH of the topsoil is weakly acidic (i.e., less than 6.0) and the pH of the subsoil is highly acidic. The epizootic area was adjacent to continuously-flooded swamps that originated at the Mississippi River. Much of the parish was occupied by natural levees which were comprised of soils that developed from loam and clay sediments. The pH of the topsoil of levees was weakly acidic to alkaline, and the pH of the subsoil was alkaline. The soil samples from six affected farm operations in Ascension Parish were cultured for *B. anthracis* during the peak of the epizootic and within a few weeks following the epizootic. The former specimens were positive; the latter, negative.

The weather data for the epizootic region were not available. Thus, an alternative data source was obtained from a weather station located 25 miles from the epizootic region. The mean precipitation for the 40-year period that was preceded by the epizootic, the months of January through June only, was 28.4 inches. In contrast, the mean precipitation in the affected region was 18.0 inches, or 37 percent lower than the normal 28.4 inches. There were anecdotal reports of no rain during the 5 weeks preceding the outbreak, followed by substantial rainfall within one week preceding the first reported anthrax case. The mean monthly ambient temperature during the first 6 months of 1971 was normal at the weather station most near the epizootic region.

Figure 06. Species-specific mortality during an anthrax epizootic, Louisiana, 1971. *(Source:* Fox et al., JAVMA, 1973.)

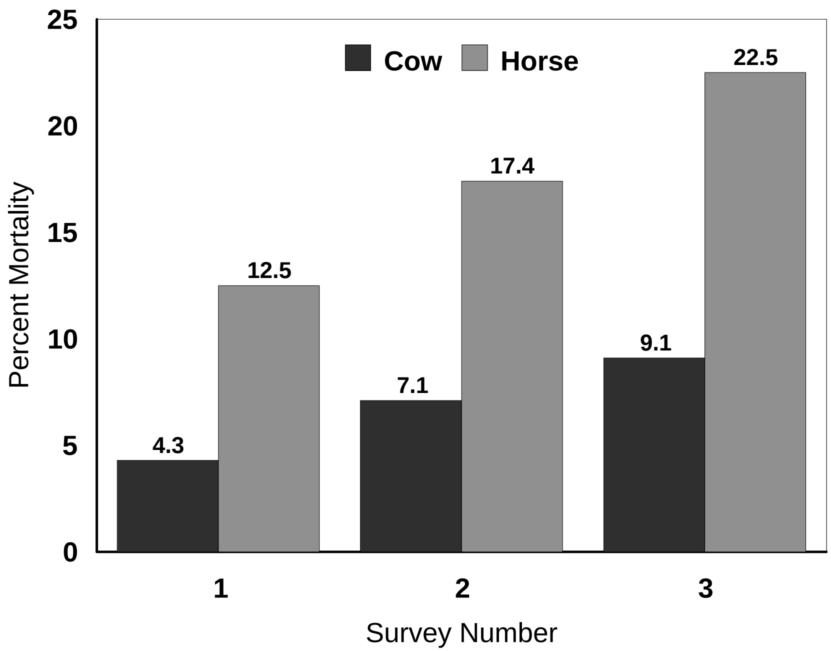


Figure 07. Proportional mortality during an anthrax epizootic, Louisiana, 1971. *(Source:* Fox et al., JAVMA, 1973).

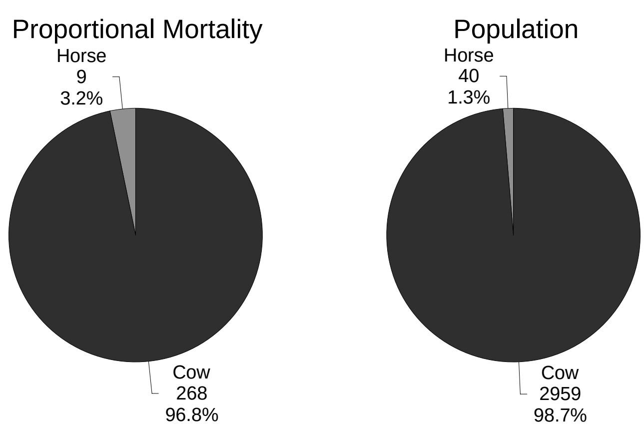


Table 01. Ecological, environmental and management factors investigated during an epizootic of anthrax involving 23 of 75 livestock premises in Ascension Parish, Louisiana in 1971, and the possible effects of the factors on the frequency of anthrax outbreaks.*

Ecological Factors	**Effects on Frequency of Anthrax Outbreaks ⌖**
Rainfall	
A "dry period" followed by a "wet period"	Possibly increased
Soil Types	
Loam and clay (alkaline pH)	Possibly increased
Calhoun-Olivier (acidic pH)	None
Management Factors	
Pastures	
Length of grasses in pastures	None
Spread fertilizer onto pastures	None
Spread lime onto pastures	None
Use inorganic fertilizers	None
Animal density in the pastures	None
Water (standing and flowing)	
Ponds located on pastures	None
Drainage canals located in pastures	None
Drainage tiles located in pastures	None
Constantly flowing streams in pastures	None
Pools of standing water during rainy weather	None
Supplements Fed	
Trace mineral salt	None
Others	
New additions to herd in 1971	None

*Sample size equals 23 premises with confirmed anthrax and 16 non-affected, neighborhood premises located within the epizootic region. ⌖Based solely on frequency data from a descriptive analysis. *(Source:* Fox et al., *JAVMA*, 1973.)

The conclusions from the ecological investigation were:

- It was concluded via process of elimination of other potential sources that contaminated soil in the pastures was a likely source of *B. anthracis*.
- Anthrax mortality was associated with all soil types in Ascension Parish, except Calhoun-Olivier soils.
- The pH of the soil types on anthrax-affected premises was neutral to alkaline.
- *B. anthracis* was cultured from soil specimens on two of six anthrax-affected farms from which specimens were collected in July (i.e., **during** the epizootic) .
- In August (i.e., **after** the epizootic), *B. anthracis* was **not** cultured from soil specimens from one of the two farms that was positive originally in July.
- The precipitation during the first 6 months of 1971 was 10 inches (35%) lower than the preceding 40-years mean of 28.4 inches at the weather station most near the epizootic.
- There were anecdotal reports of no rainfall during the 5 weeks preceding the epizootic, followed by substantial rainfall during the week preceding the first reported case.
- The mean monthly ambient temperature during the first 6 months of 1971 was normal at the weather station most near the epizootic.

Anthrax Epizootic in Texas, 1974

The Epizootic

On June 28, 1974, the Texas Veterinary Medical Diagnostic Laboratory isolated *Bacillus anthracis* from cattle on two neighboring premises. The animals had died on June 25 and June 26. An epizootiological investigation by the Centers for Disease Control and Prevention was begun on July 20 (Fox et al., 1977). Livestock producers were interviewed to collect data about animal populations, deaths, feeding practices, management practices, pastures, water supplies, and vaccine usage. Each dead animal was classified either as a confirmed anthrax death, a probable anthrax death, or a possible anthrax death. No more than five percent of the 192 animals that died between June 16 and July 16 were subjected to a bacteriological examination. However, specimens from most of the remaining 44 animals that died during the epizootic were examined, beginning on July 18. Anthrax was diagnosed on approximately 93 premises. If anthrax was the suspected cause of death, all mortalities were categorized further as "confirmed", "probable", or "possible" anthrax. Confirmed anthrax deaths occurred on 27 of the 93 premises. Probable anthrax deaths and possible anthrax deaths occurred on 67 other premises, most of which were located in Falls County, Texas. Data about the date of onset of the outbreak **(Figure 08)** and specific data about mortality **(Table 02)** were available for only 48 of the 93 premises. The duration of the outbreak was at least 7 weeks. Anthrax was diagnosed on the first premises during the second week of June, the greatest numbers of affected premises were reported during the second week of July, and the last affected premises were reported during the final week of July. A total of 236 livestock deaths were attributed to anthrax **(Figures 09, 10)**. The temporal distribution of the number of deaths was a similar to the temporal distribution of affected premises.

Management Factors

To determine the likely sources of infection, producers from 18 of 46 premises located in Falls County were interviewed. Each producer was asked to provide information about the management factors that were specific to their farming operation **(Table 03)**. Neither recently purchased herd additions, mineral supplements containing bone meal, water sources, fertilizers of animal origin, anthrax vaccination status, introduction to new pastures, nor animal density was associated with anthrax infection. By process of elimination, it was concluded that contaminated pasture soil was the most likely source of *B. anthracis*. When it is taken into consideration that a sample size of 18 was relatively small, it may be expected that no

Figure 08. Week of onset of confirmed anthrax cases on 27 premises, Falls County, Texas, 1974. *(Source:* Fox et al., JAVMA, 1977).

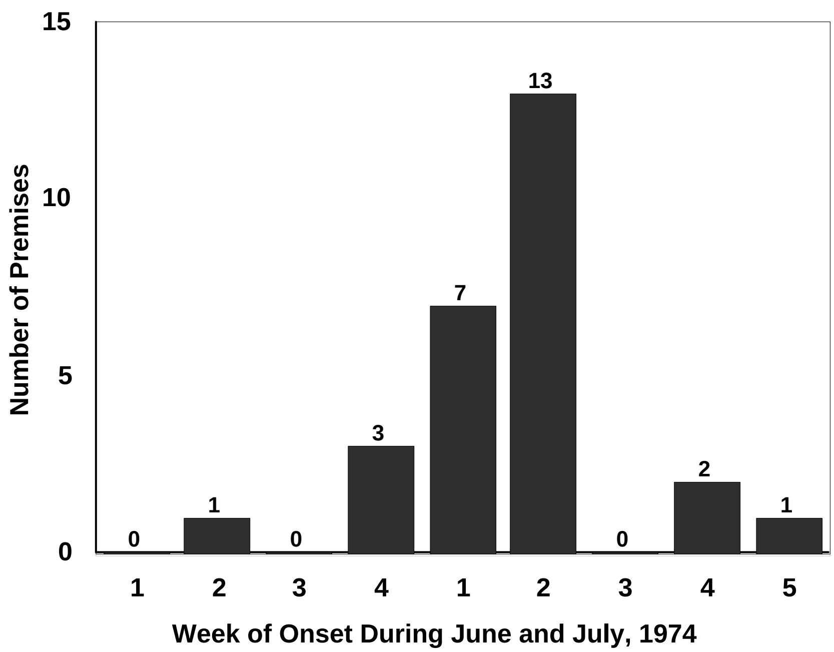


Table 02. Confirmed, probable, and possible deaths due to an epizootic of anthrax on 48 premises in Falls County, Texas in 1974.

	Confirmed Deaths		Probable and Possible Deaths		
	Number	Percent	Number	Percent	Totals
Species					
Cattle	24	10.5	204	89.5	228
Horses	2	40.0	3	60.0	5
Mules	1	50.0	1	50.0	2
Pigs	0	0.0	1	100.0	1
Totals	27		209		236

(Source: Fox et al., *JAVMA*, 1977.)

Figure 09. Weekly mortality due to anthrax on 48 premises, Falls County, Texas, 1974. *(Source:* Fox et al., *JAVMA*, 1977).

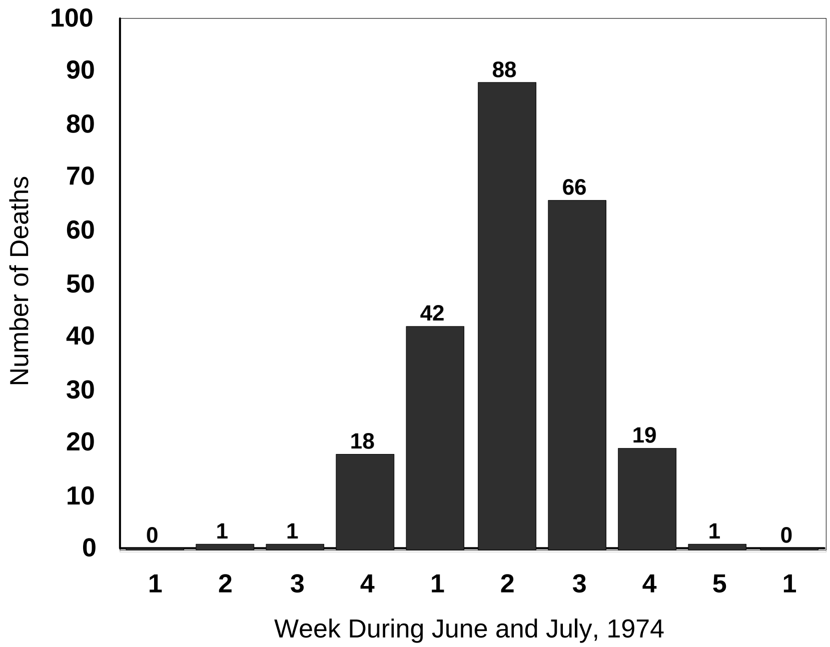


Figure 10. Epizootic curve of anthrax on 48 premises, Falls County, Texas, 1974. *(Source:* Fox et al., *JAVMA*, 1977.)

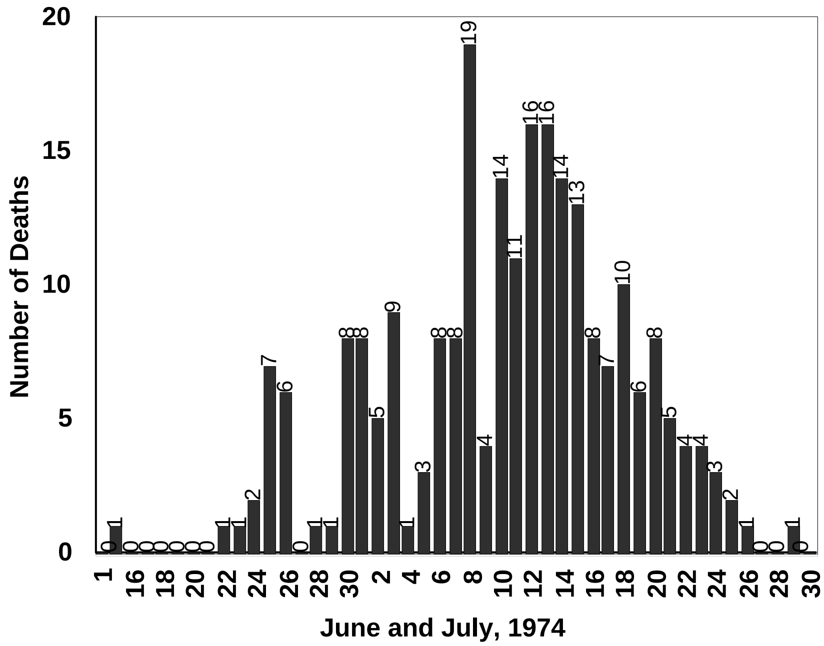


Table 03. Ecological factors and soil characteristics investigated during an epizootic of anthrax on 18 of 48 livestock premises in Falls County, Texas during year 1974, and possible effects of the factors on the frequency of anthrax outbreaks.*

Ecological Factors	**Effects on Frequency of Anthrax Outbreaks⌖**
Rainfall	
A “dry period” that was not followed by a “wet period”	Possibly increased
Soil Characteristic	
Texture	
Loam	Possibly increased
Clay	None
Sand	None
Topsoil pH (0 to 17 mm deep)	
Acidic to mildly acidic	Possibly increased
Alkaline	None
Subsoil pH (1.5 m deep)	
Acid	None
Alkaline	Possibly decreased
Surface Water Drainage	
Poor	Possibly increased
Good	None or possibly decreased
Water permeability	
15 to 500 mm per hour	Possibly increased
Less than 1.5 mm per hour	None
Herbage production	
More than 736 kg per hectare	None
Less than 736 kg per hectare	None

*Sample size equals18 premises with confirmed anthrax, 18 non-affected premises located outside the epizootic region, and 9 non-affected premises located within the epizootic region. ⌖Based solely on descriptive analysis. *(Source:* Fox et al., *JAVMA*, 1977.)

specific management factor could be linked statistically to the outbreaks.

Ecological Factors

Climatological data that had been recorded at the nearest official weather station were obtained from the National Climatic Center, U.S. Department of Commerce. The unofficial weather records were obtained from the county seat in Falls County, Texas. Falls County experienced a severe drought during the summer of 1974. The mean precipitation during January through June for the 77 years preceding 1974 was 48 cm. The mean precipitation during January through June of 1974 was only 23 cm, 48 percent lower than previous records. In contrast to the dry year of 1974, the annual precipitation during year 1973 was 28 cm above normal. The unofficial weather records from the county seat in Falls County were similar to the records from the National Climatic Center. Rainfall data were not available at the geographical center of the epizootic. However, there were anecdotal reports from livestock producers of no rainfall from May through July. The pastures that were located at the geographical center of the epizootic were dry and closely grazed relative to other non-affected pastures. Regarding environmental temperatures, the mean monthly temperatures were normal from January through June 1974.

Geological data about soil characteristics that had been recorded at the nearest official weather station were obtained from the Soil Conservation Service, U.S. Department of Agriculture. The soil characteristics (e.g., texture, surface soil pH, subsoil pH, surface water drainage, water permeability of the soil, and herbage production during years in which the climate was unfavorable) of affected premises and control premises were compared. The significant findings from the descriptive analysis of the ecological investigation were:

- The mean precipitation during January through June of the year of the outbreak was only 52 percent of the mean (i.e., 48 cm) for this same six-month period during the preceding 77-years.

- Records from the county seat in Falls County, Texas showed that there was no rainfall during the 5 weeks preceding the outbreak.

- There were anecdotal reports of no rainfall at the geographical epicenter of the outbreak, neither during the five weeks preceding the outbreak, nor during the duration of the outbreak itself.

- Loamy soil was predominant on the anthrax-affected premises. Clay soil was predominant on the non-affected premises.

- The pH of the top soil on the anthrax-affected premises was acidic. Alkaline top soils were predominant on the non-affected premises. The pH of subsoils was alkaline, on anthrax-affected premises and non-affected premises.

- The ability of soils on the anthrax-affected premises to store moisture was lower. Absence of stored moisture may promote growth of *B. anthracis* and inhibit growth of saprophytic competitors of *B. anthracis*.

Although there were some differences in the frequencies of these geological characteristics between anthrax-affected premises and non-affected premises, it was not evident that these differences were due to anything but chance differences between the two types of premises, thus the weak statistical association between each factor and the frequency of anthrax outbreaks **(Table 03)**.

The Anthrax Epizootic in Victoria, Australia, 1997

The Epizootic

Prior to 1997, anthrax epizootics were uncommon in Victoria. Only three small outbreaks were reported between 1983 and 1996. An epizootic of anthrax in an intensive dairy production region in the Goulburn Valley of Victoria occurred in 1997 (Turner et al., 1999A). This dairy region is comprised of small and large irrigated premises with very high

Figure 11. Week of onset of anthrax on 83 premises, Victoria Australia, 1997. *(Source:* Turner et al., 1999A. Revised.)

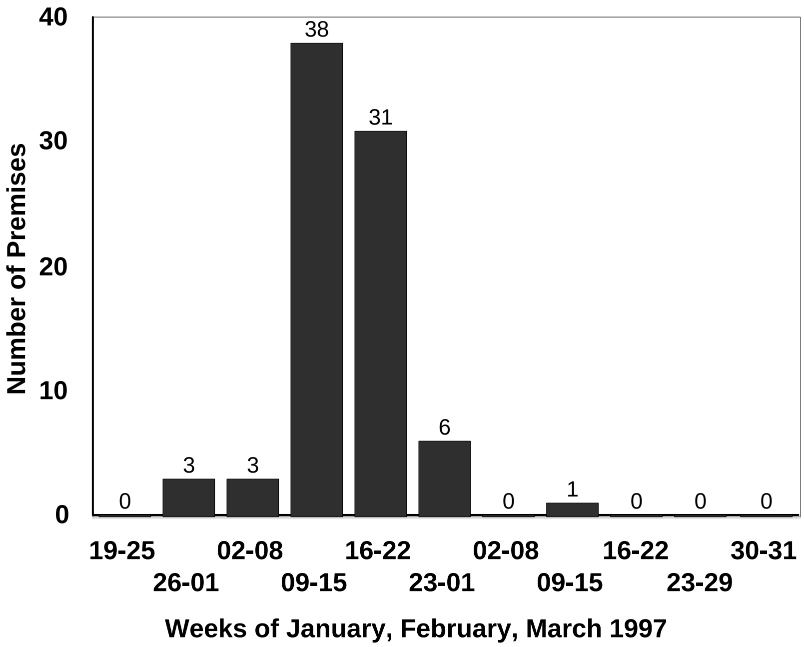
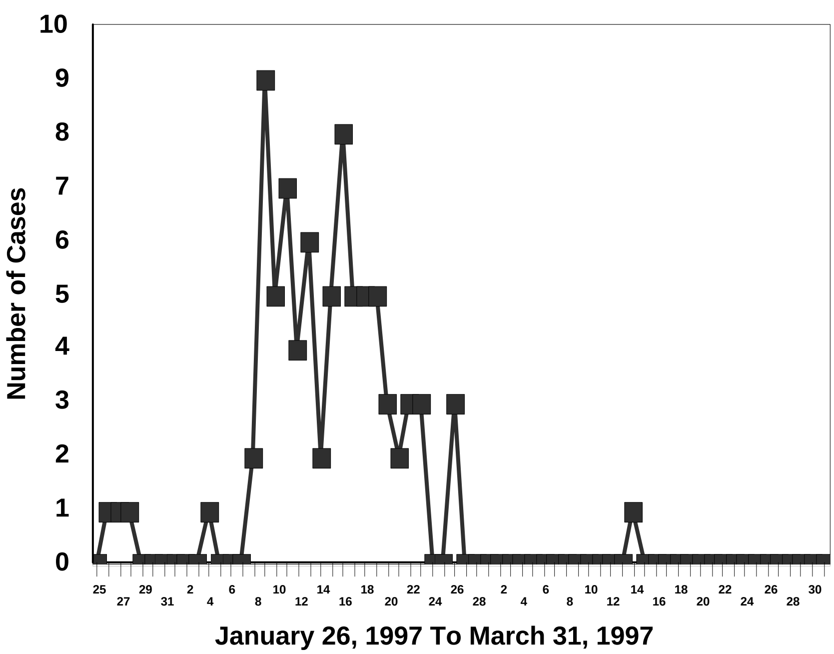


Figure 12. Date of onset of anthrax on 83 premises, Victoria Australia, 1997. *(Source:* Turner et al., 1999A. Revised.)



stock densities. Although the initial case of anthrax may have occurred on a premises on January 10, 1997, the first laboratory-confirmed case was diagnosed on an adjacent dairy premises on January 26, 1997. Anthrax had been diagnosed on 82 additional operations by March 31, 1997 **(Figures 11, 12)**.

During the peak week of the epizootic (i.e., week number three), new outbreaks on at least 30 premises were reported. The temporal distribution of individual cases was similar to the distribution of affected premises **(Figure 13)**. The peak number of cases was diagnosed during the third week of the epizootic. The total duration of the outbreak was approximately 10 weeks.

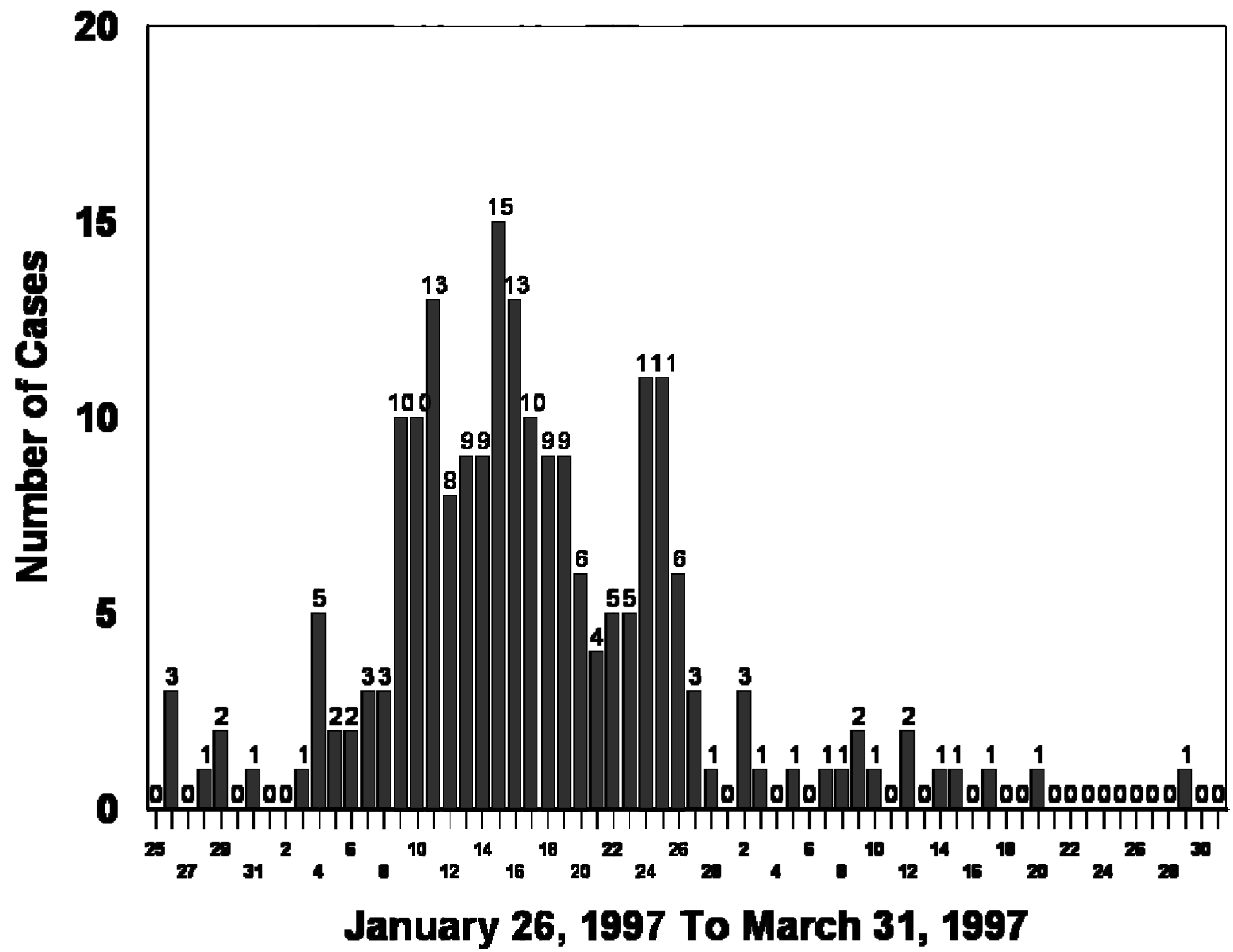
Epizootiological Observations

- Epizootiologically speaking, the disease pattern was described as a common source epizootic with multiple outbreaks.
- The geographical location of the epizootic was historically a major route for movement of cattle and sheep from southern Victoria to northern Victoria.
- There were no previous recordings of anthrax in grazing livestock in the area. It was presumed that anthrax occurred originally in the region during the preceding century, in association with: (1) movement of livestock along the route, (2) knackeries, and (3) “boiling-down works”.
- Spread of anthrax among farm operations was thought to be rare. Spread among the operations was **not** thought to be a significant propagator of the epizootic.
- Some outbreaks of anthrax occurred on farm operations that were physically remote from currently-infected operations. The occurrences of these outbreaks were beyond the incubation period for anthrax, thus disfavoring a point source epizootic, and favoring a common source, multiple-outbreaks epizootic.
- Poorly drained dairy operations on which there were **no** outbreaks of anthrax were operations that practiced vaccination against anthrax. Apparently, vaccination protected these operations, even though the environment may have been conducive for an anthrax outbreak.
- There was no epizootiological evidence that flies, biting insects, carrion scavengers, manufactured feed, milk factory tanker routes, veterinary visits, medical therapies, movement of personnel among farms, and burning of carcasses contributed to the spread of anthrax.

Ecological Observations

- Poorly drained swamps containing alluvial soils were common among anthrax-affected farm operations. These soils may support the accumulation and multiplication of anthrax spores.
- Historically speaking, the swamps on anthrax-affected farm operations were reported to accumulate water routinely after moderate rainfalls.
- There were no outbreaks of anthrax on operations located immediately north of the geographic region in which there were outbreaks, presumably because community drainage systems had been constructed in the more northerly regions.
- The soil in the anthrax-affected region had been disturbed recently due to improvements in the irrigation system and reconstruction of channels and drainage systems.
- The weather conditions preceding and during the epizootic approximated that of a drought. They were described as being dry, hot, and humid.
- The 30-year daily maximum temperature of 29^{o}C was exceeded on 26 (86.6 %) of the 30 days between January 21, 1997 (i.e., five days preceding the identification of the index case premises) and February 20, 1997 (i.e., when few, newly affected premises were identified).

Figure 13. Epizootic curve of anthrax, Victoria Australia, 1997. *(Source:* Turner et al., 1999A. Revised.)



- The relative humidity was either normal, or it was higher than normal, on 23 (76.6 %) of the 30 days between January 21, 1997 and February 20, 1997.

- The environmental temperature decreased in mid-March. The lower temperature and widespread vaccination against anthrax were two factors that were associated with a dramatic decrease in the number of newly affected premises.

The number of laboratory-confirmed cases per premises was as few as one to more than 10. There was one confirmed case on 49 percent of affected premises, two to four confirmed cases on 25 percent of affected premises, five to nine cases on three percent of the affected premises, and 10 or more cases on five percent of affected premises **(Figure 14)**.

A vaccination program was used to decrease the impact of the outbreak. There were 457 cattle herds and 78,649 cattle located within the 30 km by 20 km vaccination buffer zone. The cattle on the affected premises, as well as cattle on those premises that were located adjacent to the incident case-premises, were vaccinated. The cattle were vaccinated during a three-week period from January 26 to February 15, 1997, thereabout. A sufficiently protective immune response to anthrax vaccine occurs 10 to 14 days after the initial vaccination. During the first 10 days after vaccination, there were approximately 146 incident cases of anthrax **(Figure 15)**. After the first 10 days of vaccination, 28 additional incident cases were diagnosed. Only one incident case was diagnosed beyond 41 days after the vaccination program had begun. The sharp decrease in incident cases 14 days after the initial vaccinations is evidence that vaccination was highly efficacious, given that the population-at-risk in the vaccination buffer zone was more than 78,000 animals.

A disease prevention strategy using vaccination was employed to reduce the incidence of anthrax during years 1998 and 1999. All cattle on the affected properties were vaccinated between May 01 and October 31 of each year. The costs of the vaccination program was shared equally among the affected producers, the Australian government, and cooperating industries.

Figure 14. Confirmed cases of anthrax per affected premises during an epizootic, Victoria Australia, 1997. *(Source:* Turner et al., 1999B.)

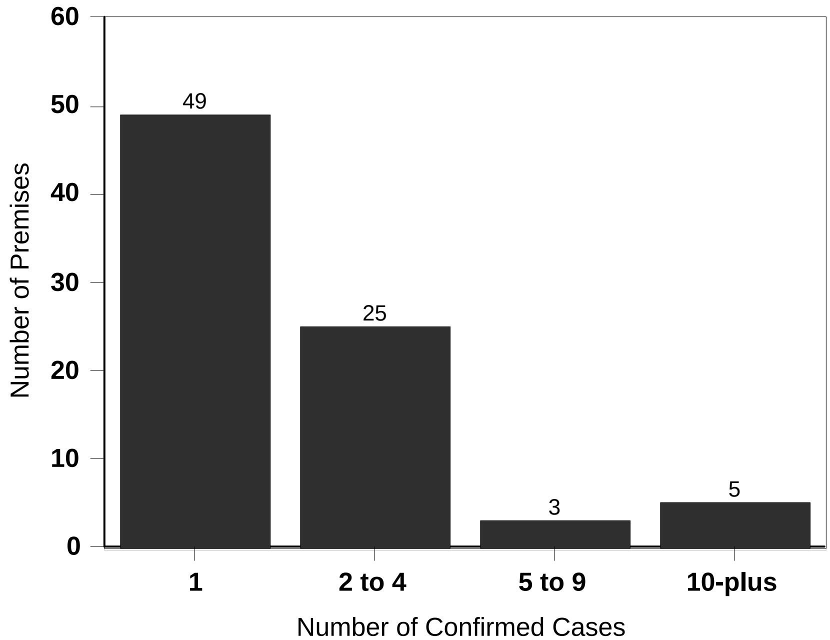
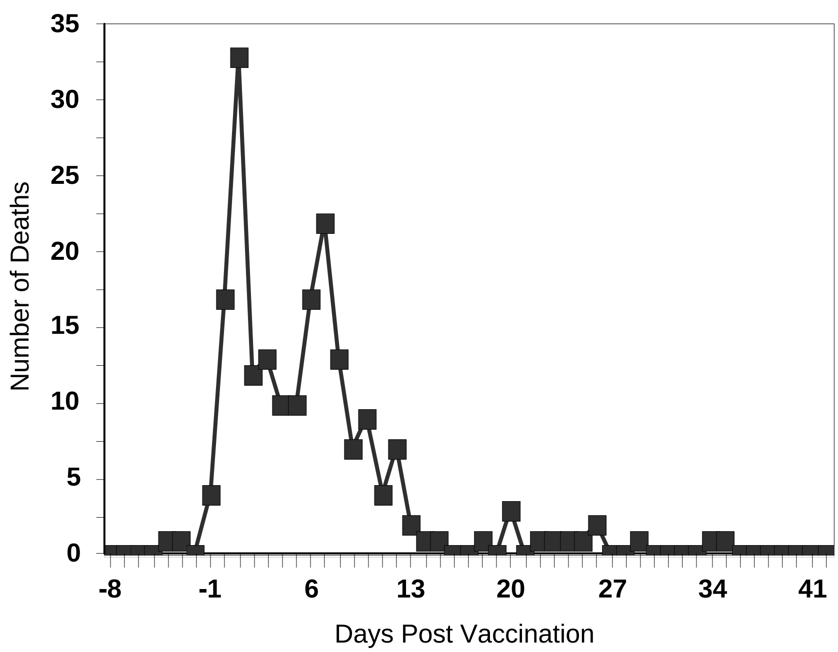


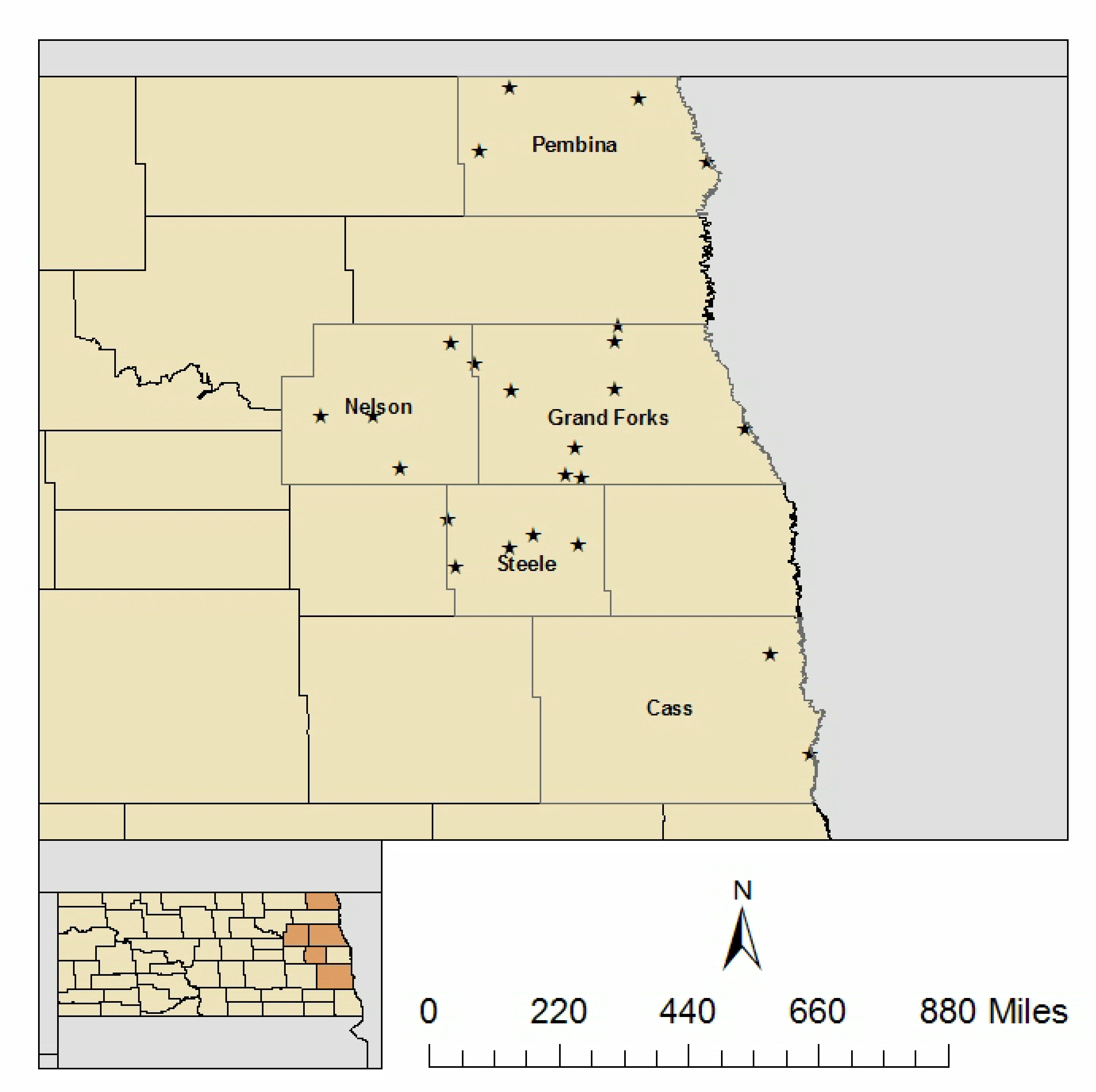
Figure 15. Confirmed clinical cases of anthrax after vaccination during an epizootic, Victoria Australia, 1997. Day 0 equals day of first vaccination. *(Source:* Turner et al., 1999B.)



Anthrax Epizootic in North Dakota, 2000

An anthrax epizootic occurred in North Dakota on 31 premises during the summer of 2000. All affected premises were confined to five, mostly-contiguous counties located in east-central and northeastern North Dakota. The affected counties were Pembina, Grand Forks, Nelson, Traill, and Steele **(Figure 16)**.

Figure 16. Geographical distribution by county of 24 of the 31 livestock premises affected by anthrax, North Dakota, 2000.



The index case premises was reported during the first week of May. Twenty-nine (94%) of the 31 case premises were diagnosed during July and August **(Figure 17)**. The last reported case occurred during the last week of September. The primary species on the premises were beef cattle, bison, sheep, and horses. Approximately 161 animal deaths were reported by the 31 case premises. The species-specific mortality was highest among horses at 12.16%, but the proportional mortality was highest among cattle at 85.7% **(Figures 18, 19, 20)**. No deaths due to anthrax in sheep were reported. Detailed data about other factors were not available for analysis of the North Dakota 2000 epizootic.31 case premises were diagnosed during July and August **(Figure 17)**. The last reported case occurred during the last week of September. The primary species on the premises were beef cattle, bison, sheep, and horses. Approximately 161 animal deaths were reported by the 31 case premises. The species-specific mortality was highest among horses at 12.16%, but the proportional mortality was highest among cattle at 85.7% **(Figures 18, 19, 20)**. No deaths due to anthrax in sheep were reported. Detailed data about other factors were not available for analysis of the North Dakota 2000 epizootic.

Figure 17. Date of onset of anthrax on 31 livestock premises, North Dakota, 2000.

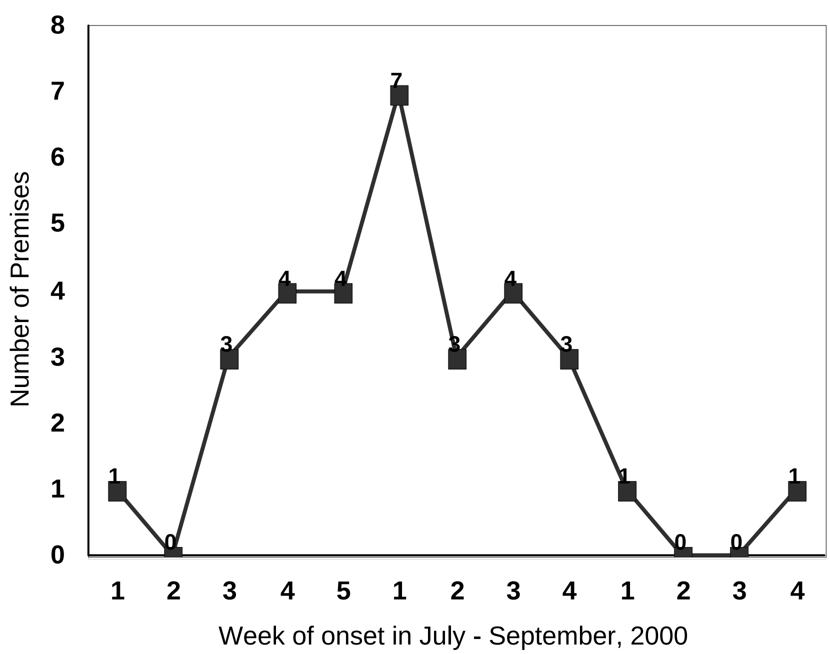


Figure 18. Population census of livestock on 31 premises affected by anthrax, North Dakota, 2000.

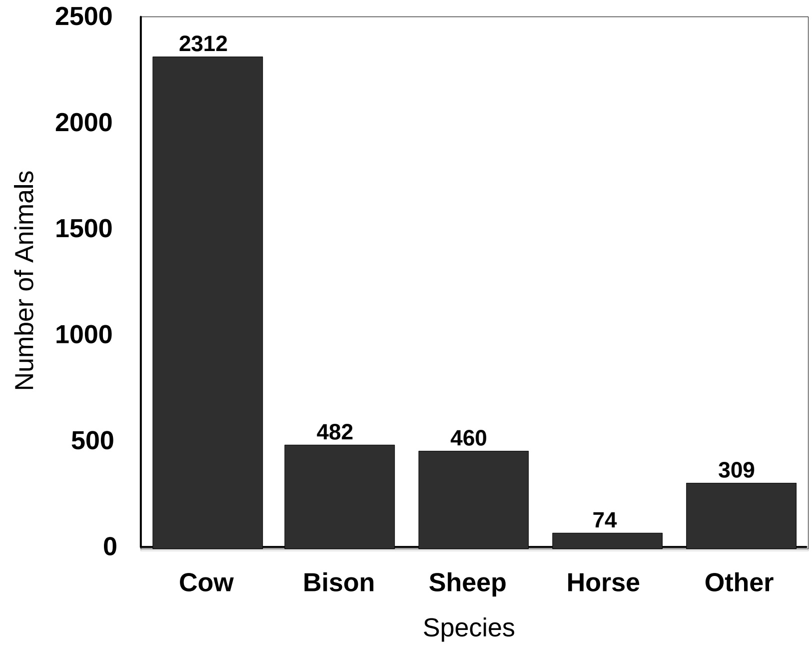


Figure 19. Species-specific mortality in 3,637 livestock during an anthrax epizootic, North Dakota, 2000.

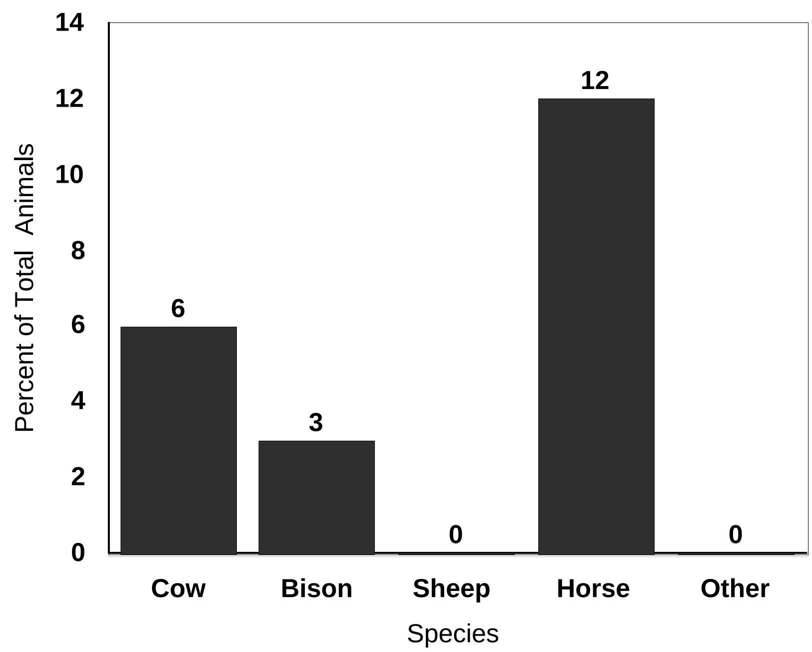
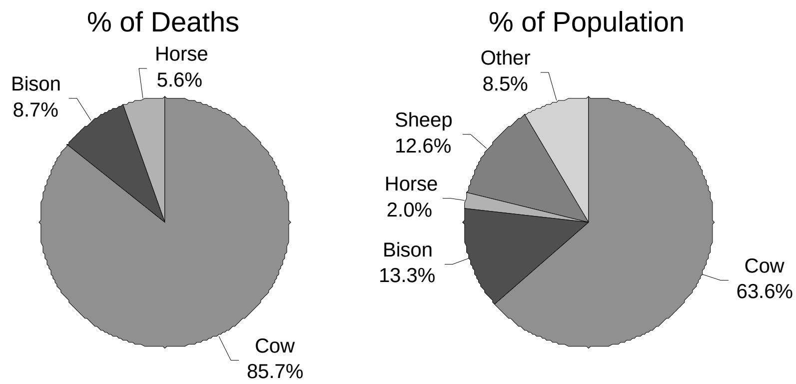


Figure 20. Proportional mortality in 161 animals from 31 premises during an anthrax epizootic, North Dakota, 2000.



Anthrax Epizootic in Texas, 2001

Anthrax is enzootic in several counties in southwestern Texas near the border of Mexico**.** The most consistently affected counties are Kinney, Val Verde, Uvalde, Edwards, and Real. A total of 25 culture-confirmed cases of anthrax in animals were reported in Texas during years 1993 to 2001 **(Figure 21)**. Fourteen of the 25 cases were reported during the seven-year interval from1993 through 2000. The remaining 11 cases (details discussed below) were reported during year 2001 **(Table 04)**. Regarding the seasonal distribution of cases, twenty-one (84%) of the 25 cases were reported during the months of June, July, August, and September **(Figure 22)**. The geographical distribution of approximately 190 confirmed cases diagnosed between 1974 and 2001 (n = 179 cases prior to 2001, and n = 11 cases during 2001) was concentrated in the same five counties **(Figure 23).** Data that showed the seasonal distribution of those cases that occurred from 1974 to 1992 were unavailable.

A major epizootic occurred during the summer of 2001, and the great magnitude of this epizootic lead to a more thorough investigation of the affected premises. The epizootic of 2001 was confined to Kinney, Val Verde, Uvalde, Edwards, and Real counties. Thus, the geographical distribution of the culture-confirmed cases of anthrax in animals in Texas in 2001 was parallel to the geographical distribution of cases from years 1974 through 2000 **(Figure 24)**. A clinical diagnosis of anthrax was made on animals from 71 premises, of which 11(15%) were confirmed via laboratory culture between June 07 and September 20. In addition to laboratory diagnosis, the ranch personnel were responsible for 56% of the diagnoses, veterinary practitioners were responsible for 24%, and an investigation team was responsible for the remaining 5%. The outbreak during 2001 was not restricted to a narrow host-range. Cervids, bovids, equids and camelids were represented among the 11 culture-confirmed cases, as well as the cases that were not confirmed via culture. More specifically, the affected species included white tailed deer, red deer, fallow deer, elk, antelope, gazelle, water buffalo, beef cow, goat, sheep, bison, horse, mule, donkey, and llama (**Table 05)**.

Figure 21. Distribution by year of 25 culture-confirmed cases of anthrax, Texas, 1993 to 2001. (*Source:* Personal communication, Terry H. Conger, Texas Animal Health Commission, 2001.)

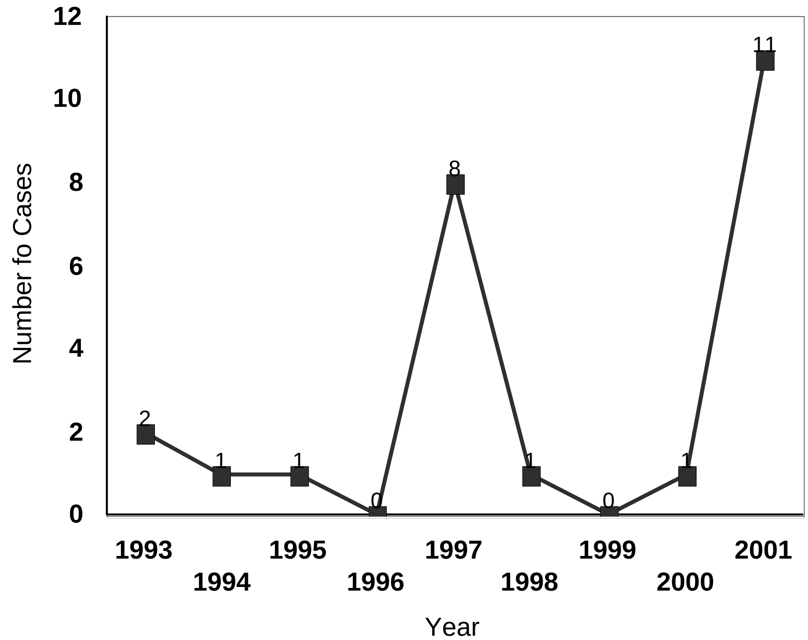
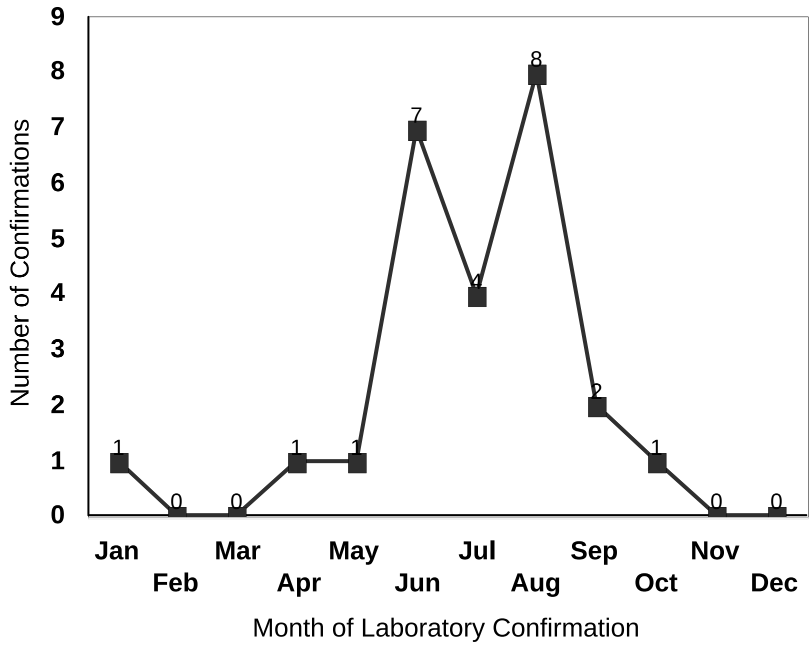


Table 04. Date of laboratory confirmation, county-of-origin, and affected species for 11 culture-confirmed cases of anthrax in Texas, year 2001.			
Date Confirmed	**County**	**Taxonomic Group**	**Common Name**
June 07, 2001	Val Verde	Cervid (exotic)	Fallow deer
June 08, 2001	Uvalde	Cervid	White tail deer
June 21, 2001	Val Verde	Bovid (domestic)	Beef cow
June 21, 2001	Val Verde	Cervid	White tail deer
June 26, 2001	Uvalde	Cervid	White tail deer
June 26, 2001	Val Verde	Equid	Horse
June 26, 2001	Edwards	Equid	Horse
July 14, 2001	Uvalde	Cervid (exotic)	Red deer
August 01, 2001	Uvalde	Camelid	Llama
August 07, 2001	Real	Bovid (domestic)	Beef cow
September 20, 2001	Kinney	Bovid (domestic)	Goat
(*Source:* Personal communication, T. H. Conger, Texas Animal Health Commission, 2001.)			

Figure 22. Distribution by month of 25 culture-confirmed cases of anthrax, Texas, 1993 to 2001. (*Source:* Personal communication, Terry H. Conger, Texas Animal Health Commission, 2001.)



diagnosis of anthrax was made on animals from 71 premises, of which 11(15%) were confirmed via laboratory culture between June 07 and September 20. In addition to laboratory diagnosis, the ranch personnel were responsible for 56% of the diagnoses, veterinary practitioners were responsible for 24%, and an investigation team was responsible for the remaining 5%. The outbreak during 2001 was not restricted to a narrow host-range. Cervids, bovids, equids and camelids were represented among the 11 culture-confirmed cases, as well as the cases that were not confirmed via culture. More specifically, the affected species included white tailed deer, red deer, fallow deer, elk, antelope, gazelle, water buffalo, beef cow, goat, sheep, bison, horse, mule, donkey, and llama (**Table 05)**.

The total number of animals that died on the 71 premises was 1,637. The proportional mortality suggests that the less-traditional species were affected by anthrax more than were the traditional domestic livestock species **(Figure 25)**. Only 200 (12.2%) of the 1,637 recorded deaths occurred in traditional domestic livestock species (i.e., beef cow, horse, mule, goat, sheep, donkey). The remaining 1,437 deaths occurred in deer and other less-traditional species. The number of deaths for a given species was as low as one (e.g., bison, donkey, llama) and as high as 1,216 (e.g., white tail deer). The average number of deaths for a given species was as low as one for equids and as high as 35 for cervids **(Table 05)**.

A quarantine was imposed on 20 of the 71 premises to control transmission of anthrax. A premises became eligible for removal of the quarantine after all livestock on the premises had been vaccinated, and 14 days after the last animal died of anthrax. Non-pregnant domestic livestock were vaccinated with two doses of a non-encapsulated, anthrax spore vaccine, there being a three-week interval between the first and second injections.

While anthrax in humans is not the emphasis of this paper, the number of reported cases of anthrax in humans in Texas decreased significantly between 1935 and 1960, and has remain very low since 1960 **(Figure 26)**.

Figure 23. Geographical distribution by county of 11 culture-confirmed cases of anthrax in animals, Texas, 2001. (*Source:* Personal communication, Terry H. Conger, Texas Animal Health Commission, 2001.)

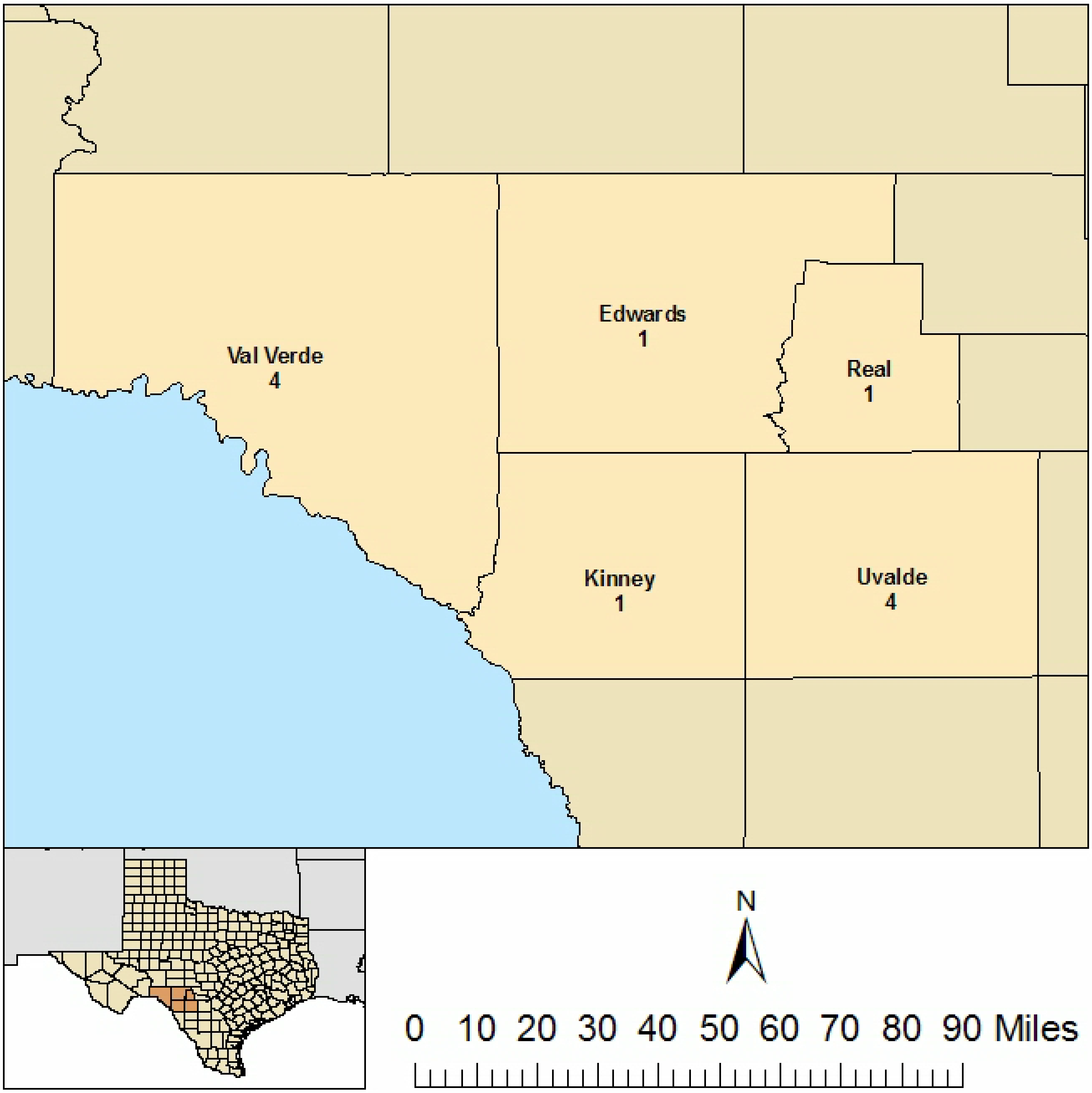


Figure 24. Geographical distribution by county of 179 culture-confirmed cases of anthrax, Texas, 1974-2000. (*Source:* Personal communication, Terry H. Conger, Texas Animal Health Commission, 2001.)

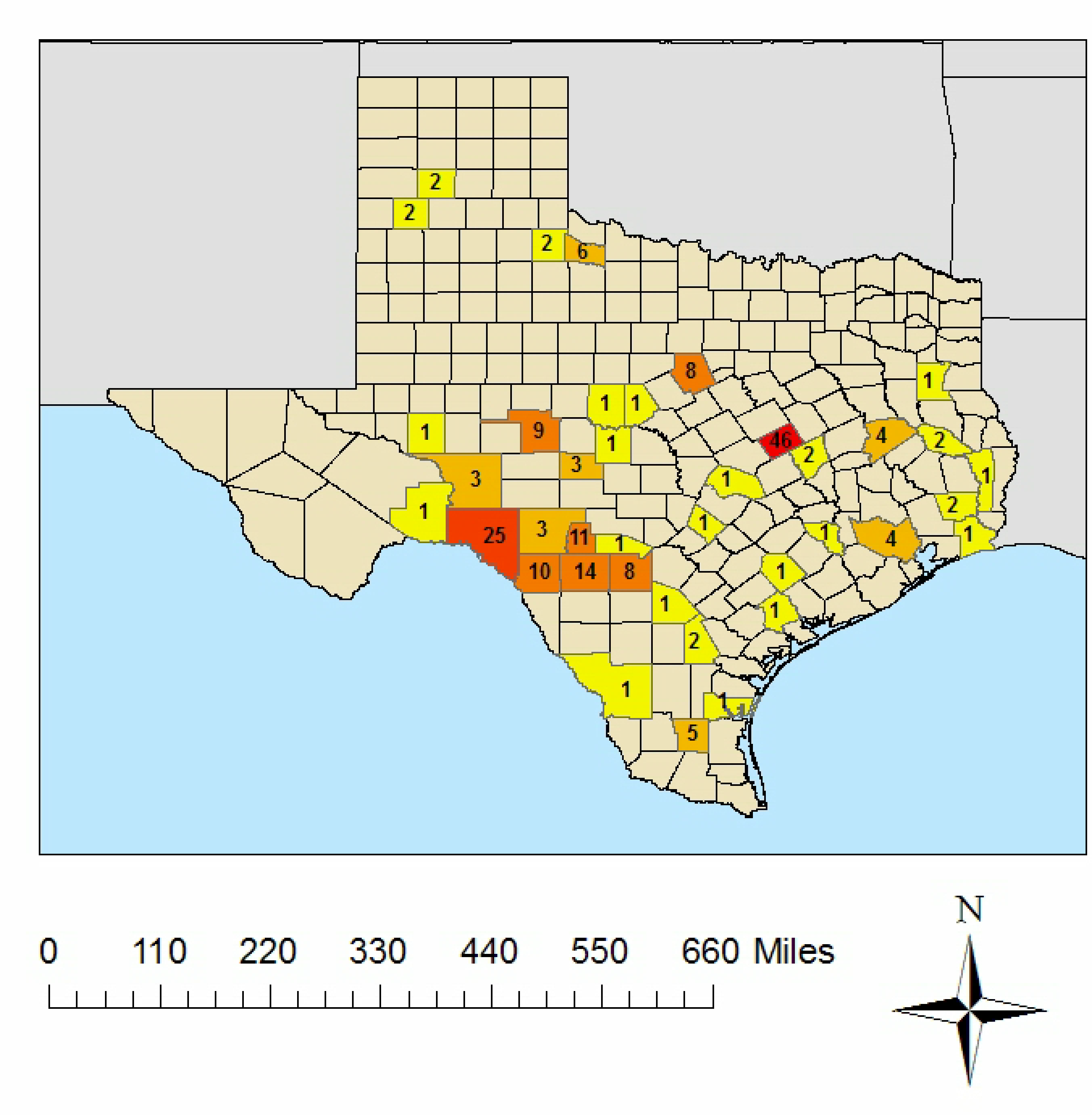


Figure 25. Mortality due to anthrax among 1,637 animals from 71 premises, Texas, 2001. (*Source:* Personal communication, Terry H. Conger, Texas Animal Health Commission, 2001.)

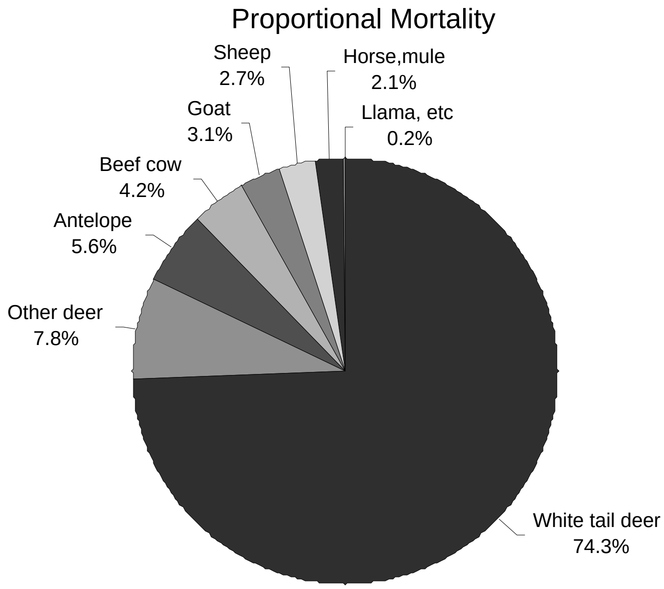
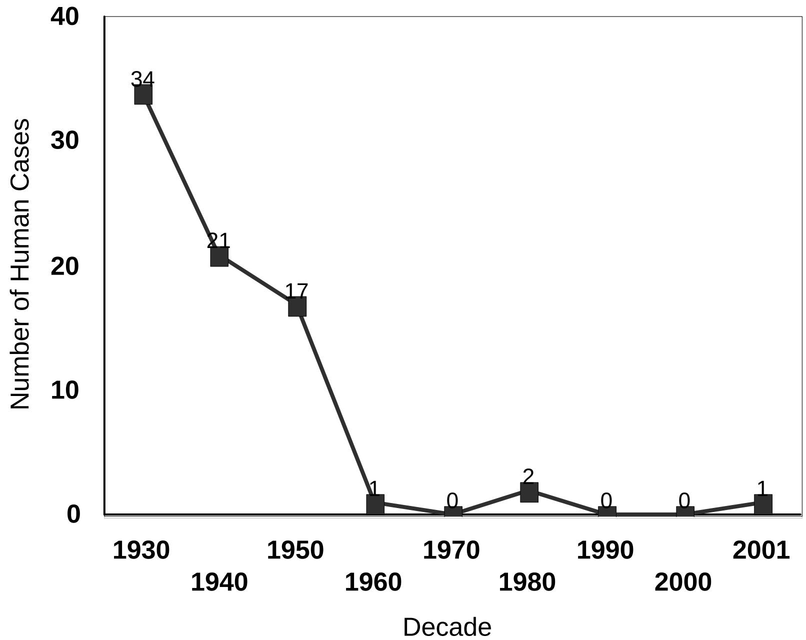


Table 05. The species affected, number of premises, total number of dead animals, and mean number of dead animals per premises for 11 premises with culture-confirmed anthrax and 60 premises with non-culture-confirmed anthrax in Texas, year 2001.

Taxonomic Group	**Common Name**	**Premises (#)**	**Deaths per Species (#)**	**Mean Deaths (#)**
Bovid (domestic)	Beef cattle	37	69	2
Cervid	White tail deer	34	1,216	35
Equid	Horse, mule	33	35	1
Bovid (exotic)	Antelope, gazelle, water buffalo	30	91	3
Bovid (domestic)	Goat	26	50	2
Cervid (exotic)	Red deer, elk, fallow deer	20	128	6
Bovid (domestic)	Sheep	14	45	3
Bovid	Bison	1	1	1
Equid	Donkey	1	1	1
Camelid	Llama	1	1	1
Totals			**1,637**	

Number of deaths equals producer-reported deaths. A given premises may have been inhabited by more than one species. *(Source:* Personal communication, T. H. Conger, Texas Animal Health Commission, 2001.)

Figure 26. Distribution by decade of 75 cases of anthrax in humans in Texas, 1935 to 2001. (*Source:* Personal communication, Terry H. Conger, Texas Animal Health Commission, 2001.)



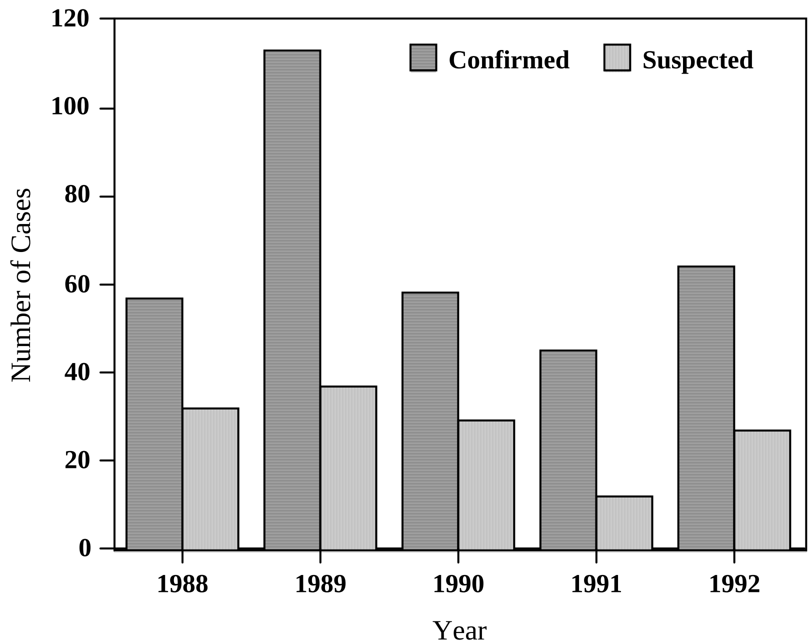
Anthrax Epizootics in Ungulates and Elephants in Namibia, 1964 to 1992

Epizootics

The first reported diagnoses of anthrax in Etosha National Park (hereafter referred to as Etosha NP) in Namibia were in 1964. The early outbreaks were attributed to the excavation of gravel pits during construction of tourist routes throughout the park (Ebedes, 1976). However, the initial attempts to isolate *B. anthracis* from these gravel pits failed (Turnbull et al., 1986), and water from the pits would not readily support the persistent growth of *B. anthracis*. While it is clear that some areas in the park are enzootic for anthrax, the definitive source(s) of infection of animals in the enzootic areas has not been identified.

Confirmed and suspected cases of anthrax in Etosha National Park have been recorded since 1964. Anthrax occurred mainly as sporadic cases with outbreaks interspersed between the sporadic cases. A total of 2,935 cases of suspected **and** confirmed anthrax were reported between 1964 and 1992. Eleven different species of large herbivores were affected, as well as one species of elephants and ten species of plains ungulates. The Burchell's zebra, blue wildebeest, springbok, and elephant accounted for 97 percent of the cases, but only 55 percent of all large herbivores. During the period 1964 to1987, 2,461 cases (mean = 107 cases per year) of anthrax were reported. The data for this 23-year period were not reported as annual data; however, the data for the five-year period 1988 to 1992 were reported as annual data **(Figure 27)**. The number of confirmed cases reported from 1988 to 1992 was 337 (interval = 45 to 113; mean = 67 per year). The number of suspected cases reported between 1988 and 1992 was 137 (interval 12 to 37; mean = 27 per year). Thus, the number of confirmed cases was 71 percent of 474 total cases. Three large epizootics occurred during years 1976, 1981 and 1984 (detailed data not available). The hosts affected during these outbreaks were elephants, several species of ungulates, and elephants again, respectively. The species of ungulates that were affected by anthrax during the three outbreaks were springbok, blue wildebeest, and Burchell's zebra.

Figure 27. Confirmed and suspected cases of anthrax in wildlife, Etosha National Park, Namibia, Africa, 1988 to 1992 (*Source:* Lindeque and Turnbull, 1994.)



Epidemiological Observations

- Males of each species, including elephants, were at greater risk of anthrax than females. (One possible exception was the zebra; however, it is more challenging to accurately distinguish a male zebra carcass from that of a female zebra, in comparison to male and female carcasses from other species in Etosha NP).

The peak incidence of anthrax in plains ungulates occurred in March, at the end of the rainy season **(Figure 28)**.

- The peak incidence of anthrax in elephants occurred in November, near the end of the dry season and the beginning of the wet season **(Figure 29)**.

B. anthracis was recovered from approximately 50 percent of the fecal specimens from scavengers, when those specimens were located at “close” proximity to anthrax-affected carcasses. *B. anthracis* was not recovered from the fecal specimens that were located at “distant” proximity to anthrax carcasses **(Table 06)**.

Figure 28. Anthrax incidence by month in ungulates, Etosha National Park, Namibia, Africa, 1976 to 1992. (*Source:* Lindeque and Turnbull, 1994.)

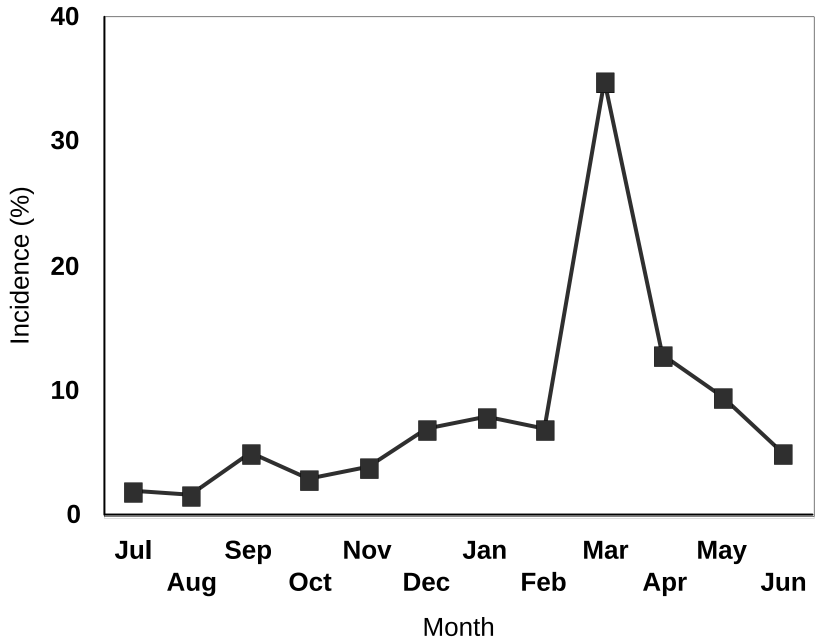


Figure 29. Anthrax incidence by month in elephants, Etosha National Park, Namibia, Africa, 1976 to 1992. (*Source:* Lindeque and Turnbull, 1994.)

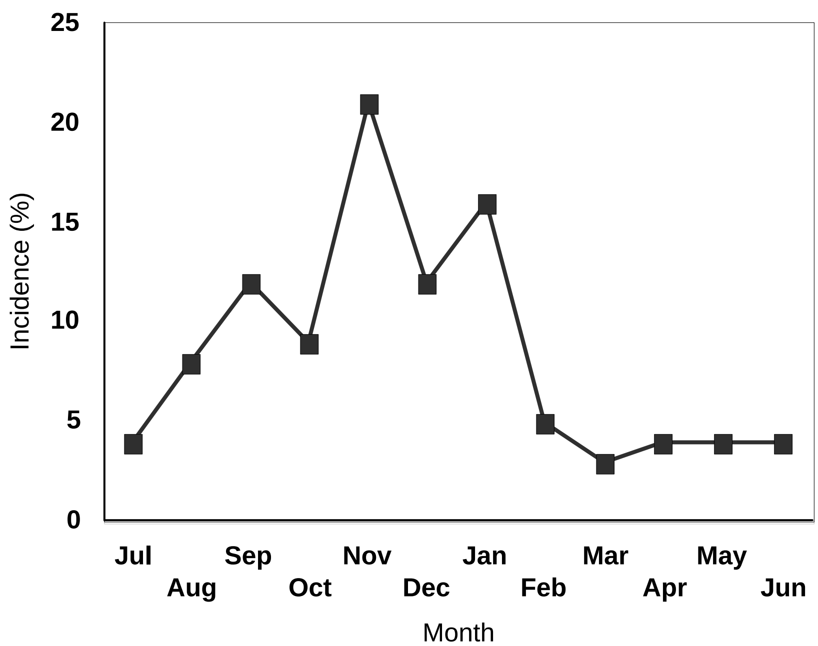


Table 06. Prevalence of anthrax spores in fecal specimens of scavengers relative to the location of anthrax carcasses in Etosha National Park, Namibia, Africa.			
Species	**Location of Specimen Relative to Anthrax Carcasses**	**Specimens Tested (Number)**	**Specimens Positive (Percent)**
Jackal	Near	25	72
	Far	9	0
Vulture	Near	18	50
	Far	4	0
Hyaena	Near	5	66
	Far	1	0
(Source: Lindeque and Turnbull, 1994. Adapted with permission.)			

Ecological Observations

- *B. anthracis* was detected in only 3.3 percent of 92 water samples that were collected from enzootic areas in the park.

- *B. anthracis* was detected in only 3.0 percent of 230 soil samples that were collected from enzootic areas in the park.

- *B. anthracis* was detected in 1.4 percent of soil and water samples that were collected from the enzootic areas during the hot, wet season (January to April).

- *B. anthracis* was detected in 5.7 percent of soil and water samples that were collected from the enzootic areas during the cold, dry season (May to August).

- *B. anthracis* was detected in 3.5 percent of soil and water samples that were collected from the enzootic areas during the hot, dry season (September to December).

- The concentration of spores in the soil adjacent to the carcasses (5.0×10^4 maximum), relative to the concentration of spores in blood specimens from the carcasses (7.0×10^9 maximum), indicated that the survival rate of spores that contaminated the soil was extremely low, even during the first 4 weeks of contamination.

- *B. anthracis* was able to multiply and sporulate in springwater and water from gravel-pits during laboratory experiments, if the waters were contaminated with low dilutions (i.e., high concentrations) of blood.

- *B. anthracis* was highly susceptible to ultraviolet radiation and to solar radiation after as few as four hours of exposure during laboratory experiments.

Summary

Anthrax deaths have been reported in ten of the 13 large herbivorous mammals in Etosha NP. Rarely has the disease been reported in carnivores. Excessively grazed vegetation was an unlikely contributing factor to the outbreaks, because the incident cases were most frequent during optimum grazing conditions. Generally, any single anthrax epizootic in Etosha NP involved a single host species, although the environment in which there were outbreaks was occupied simultaneously by other susceptible host species. This suggests that host factors, rather than environmental factors, may be the predominant category of causal factors of anthrax in Etosha NP. Contrary to what has been described in bison bulls in Canada, there is no explanation to account for the apparent gender differences in the incidence of anthrax in Etosha NP.

During the environmental survey of enzootic areas, no specific sites were identified as a source of *B. anthracis* in Etosha NP. Specimens were collected from gravel pits, natural pans, springs, and boreholes. There was little evidence to support the hypothesized association between exposure to gravel pits and outbreaks of anthrax, which was consistent with the previous findings of other investigators (Turnbull, 1986). Although *B. anthracis* was isolated from gravel pits, the isolations were rare. *B. anthracis* was present in low numbers in waterholes in the environment, and only for abbreviated periods of time. One explanation for the transient contamination of waterholes is that the waterholes, and gravel pits in particular, become the last sources of water as the dry season progresses. Thus, they become a point of concentration for animals that have anthrax already, as well as for those animals that may develop anthrax. Another explanation is the translocation of spores from the carcasses to the waterholes by vultures and other scavengers.

The level of anthrax spores in the feces of scavengers is a function of the degree of anthrax sporulation in carcasses consumed by the scavengers. The fragility of vegetative *B. anthracis* predisposes it to destruction during intestinal transit in many scavengers. Vultures may contaminate the waterholes after having consumed infected carcasses that are at distant proximities to waterholes. However, vultures consume only carcasses that are relatively fresh, and they consume only the parts of carcasses that are laden with fewer spores. Because these parts of fresh carcasses are devoid of spores, the importance of vultures in dissemination of anthrax spores and contamination of waterholes may not be significant. Carnivores (e.g., hyenas), on the other hand, may consume the parts of carcasses that are heavily laden with spores.

The bacterial count in the blood of animals that die from anthrax can be high. Thus, another source of anthrax spores in soil simply may have been from the carcasses of animals that died in close proximity to their last available source of water. The soil adjacent to these carcasses was examined to study the extent of, and the persistence of, freshly shed *B. anthracis*. Although extensive contamination of soil with blood that drained from the carcasses could have occurred, the concentration of spores in the soil was relatively low, probably because sporulation of the released bacilli was very low. The soil from a fraction of these sites that had been contaminated with hemorrhage from anthrax carcasses was examined for as long as 60 consecutive months to determine the persistence of anthrax spores, and persistence was much abbreviated. Regarding soil type, the persistence of spores in karstveld soils was higher than in other soil types.

The fate of *B. anthracis* that had been isolated from water fountains and gravel pits was examined experimentally using contaminated blood within the dilution range of 1:5 to 1:1640. Multiplication of *B. anthracis* occurred at lower dilutions (1:5 to 1:320). However, the vegetative cells died prior to sporulation at the higher dilutions (1:1640), maybe due to a reduction of the nutritional benefits of the blood. The susceptibility of *B. anthracis* to ultraviolet radiation and to solar radiation as a function of time was examined also. After four hours of exposure, the growth of *B. anthracis* was detectable only poorly, or not at all.

Conclusions

The goal of this paper was to compile published and un-published information about the epizootiology and ecology of anthrax. The epizootics reviewed herein included a period of at least 30 years (1971 to 2001), three continents (North America, Australia, and Africa), four countries/provinces (the United States, Canada, Namibia, Victoria) and two broad classifications of animals (domesticated livestock and wildlife).

Anthrax epizootics in livestock and wildlife are restricted to specific geographical regions, regardless of continent, country, or geopolitical unit within a given country. Epizootics in livestock in the US are restricted to states west of the Mississippi River. Albeit somewhat speculative, the location of epizootics within these western states parallels the movement of cattle that may have become infected with anthrax during cattle drives and during the westward migration of pioneers and their livestock. Similarly, epizootics of anthrax in livestock and bison in Canada have been restricted to the western provinces for decades. The epizootics in bison in Wood Buffalo National Park have been rewarding scientifically in that they have provided several opportunities to explore hypotheses about the ecology of anthrax, the results of which may be applicable to anthrax in domesticated species.

Observations of the role of climatic factors such as season-of-year, ambient temperature, and drought in promoting anthrax epizootics have been made for several decades. There also have been attempts to support these observations via quantitative methodology. The commonality of summer months, high ambient temperatures, drought and anthrax are non-contentious. The roles of environmental factors such as soil types and soil disturbances via excavation are poorly defined despite attempts to evaluate these potential factors.

The most extensive analysis of risk factors for anthrax epizootics in the US were undertaken during investigation of the 1971 epizootic in Louisiana, the 1974 epizootic in Texas, and the 2001 epizootic in Texas. Unfortunately, there were no great revelations from the analyses, but the hypothesized risk factors that were examined should not be discounted because anthrax investigations in livestock seem to lag behind the epizootic curves; thus, the null effects from these analyses may be inflated by problems of recall bias, small sample size, non-participation by livestock producers, etc. Quantitative analysis of risk factors for epizootics is one aspect of anthrax that can be pursued more aggressively.

A novel and potentially significant face of anthrax in the US is the host species. Mortality in exotic species was not evident in reports of the anthrax epizootic in Texas in 1971, nor other epizootics discussed here. To the contrary, white tail deer and other exotic species were severely affected during the epizootic in Texas in 2001. This change in affected host species appears to be a social factor, not a biological factor. The change can be attributed to the explosive popularity of game ranches in Texas, and not necessarily to increased host susceptibility of exotic species. The greater proportional mortality in exotic species is due to the inability to capture and immunize exotic species during epizootics. Traditional domestic livestock species can be vaccinated more successfully during an epizootic; thus, domestic livestock have received the benefits of vaccination, whereas the exotic species have not benefitted.

Bibliography

Anonymous. 1998. Anthrax. In: Aiello, S. E., Editor. The Merck Veterinary Manual. 8th ed. New Jersey: Merck & Co., Inc. 432–435.

Broughton, E. 1992. Anthrax in bison in Wood Buffalo National Park. Canadian Veterinary Journal. 33: 134–135.

Centers for Disease Control and Prevention. 2005. Bioterrorism agents/disease. Accessed 2005. Web Page. Available at: http://www.bt.cdc.gov/agent/agentlist.asp.

Conger, T.H. 2001. Anthrax epizootic Texas, Summer of 2001. In: Proceedings of the One Hundred and Fifth Annual Meeting, United States Animal Health Association: Hershey, Pennsylvania. Virginia: United States Animal Health Association; 207.

Dragon, D. C.; Elkin, B. T.; Nishi, J. S.; Ellsworth, T. R. 1999. A review of anthrax in Canada and implications for research on the disease in northern bison. Journal of Medical Microbiology —3rd International Conference on Anthrax, University of Plymouth, 7–10 September, 1998. 87: 208–213.

Dragon, D. C.; Rennie, R. P. 1995. The ecology of anthrax spores: tough but not invincible. Canadian Veterinary Journal 36: 295–301.

Ebedes, H. 1977. Anthrax epizootics in Etosha National Park. Madoqua 10: 99–118.

Fox, M. D.; Boyce, J. M.; Kaufmann, A. F.; Young, J. B.; Whitford, H.W. 1977. An epizootiologic study of anthrax in Falls County, Texas. Journal of the American Veterinary Medical Association 170: 327–333.

Fox, M. D.; Kaufmann, A. F.; Zendel, S. A.; Kolb, R.C.; Songy, C. G. Jr.; Cangelosi, D. A.; Fuller, C. E. 1973. Anthrax in Louisiana, 1971: epizootiologic study. Journal of the American Veterinary Medical Association 163:446–451.

Gainer, R.S.; Saunders, J.R. 1989. Aspects of the epidemiology of anthrax in Wood Buffalo National Park environs. Canadian Veterinary Journal 30: 953–956.

Gates, C. C.; Elkin, B. T.; Dragon, D. C. 1995. Investigation, control and epizootiology of anthrax in a geographically isolated, free-roaming bison population in northern Canada. Canadian Journal of Veterinary Research 59: 256–264.

Inglesby, T. V.; Henderson, D. A.; Bartlett, J. G.; Ascher, M. S.; Eitzen, E.; Friedlander, A. M.; Hauer, J.; McDade, J.; Osterholm, M. T.; O'Toole, T.; Parker, G.; Perl, T. M.; Russel, P. K.; Tonat, K. 1999. Anthrax as a biological weapon—medical and public health management. JAMA. 281: 1735–1745.

Inglesby, T. V.; O'Toole, T.; Henderson, D. A.; Bartlett, J. G.; Ascher, M. S.; Eitzen, E.; Friedlander, A. M.; Gerberding, J.; Hauer, J.; Hughes, J.; McDade, J.; Osterholm, M. T.; Parker, G.; Perl, T. M.; Russell, P. K.; Tonat, K. 2002. Anthrax as a biological weapon, 2002—updated recommendations for management. JAMA 287:2236–2252.

Lindeque, P.M.; Turnbull, P. C. B. 1994. Ecology and epidemiology of anthrax in the Etosha National Park, Namibia. Onderstepoort Journal of Veterinary Research 61:71–83.

Noah, D. L.; Sobel, A. L.; Ostroff, S. M.; Kildew, J. A. 1998. Biological warfare training: infectious disease outbreak differentiation criteria. Military Medicine 163:198-201.

Turnbull, P. C. B.; Hofmeyr, J. M.; McGetrick, A. M. T.; Oppenheim, B. A. 1986. Isolation of *Bacillus anthracis* , the agent of anthrax, in the Etosha National Park. Madoqua 14:321-331.

Turner, A. J.; Galvin, J. W.; Rubira, R. J.; Condron, R. J.; Bradley, T. 1999b. Experiences with vaccination and epidemiological investigations on an anthrax outbreak in Australia in 1997. Journal of Applied Microbiology—3rd International Conference on Anthrax, University of Plymouth, 7-10 September 1998. 87:294-297.

Turner, A. J.; Galvin, J. W.; Rubira, R. J.; Miller, G. T. 1999a. Anthrax explodes in an Australian summer. Journal of Applied Microbiology—3rd International Conference on Anthrax, University of Plymouth, 7-10 September 1998. 87:196-199.

Van Ness, G. B. 1971. Ecology of anthrax. Science 172:1303-1307.