Hippocrates was one of the first to recognize that there may be multiple determinants of disease when he pointed out the role of various environmental factors in the causation of disease in his *Airs, Waters and Places*. In 1930 Georg Francke and Viktor Gorttler wrote:

"Epidemics never arise from a single cause, but from the interaction of several, at times numerous causes; their strength depending on various influences. These causal forces can strengthen or weaken or cancel each other. The end effects of these forces determine the course of the epidemic"

- quoted in Schwabe, 1984

Anthrax and its expression in wild animal populations is a complex interaction between the infective organism, the host species and the environment. There is a certain global uniformity to anthrax epidemics in the way that they are initiated from spores in the environment and the general manner that wild animals are thought to be exposed to the causative organism. The factors that perpetuate an outbreak tend to be geographically specific.

Globally, the spores of *Bacillus anthracis* are associated with alkaline, calciferous soils that are rich in organic matter (Van Ness, 1971). Outbreaks of disease usually occur in circumstances of hot, dry weather, frequently preceded by a marked ecologic or climatic change such as flooding or rainfall followed by drought. This ultimately favours the exposure of susceptible animals to the anthrax organisms (Choquette & Broughton, 1981). The mechanical vectors of anthrax like insects and carnivorous scavengers tend to be unique to the ecosystem involved (Pyper and Willoughby, 1964; Kellogg and Prestwood, 1970; Braack and de Vos, 1990; Gainer, 1987). The level of resistance to *B. anthracis* within and between species of animals, and the strain and virulence of the organism are more variables in the causal web surrounding anthrax epidemics (Titball and Manchee, 1987; Sterne, 1959; Choquette and Broughton, 1981)

**Outbreak initiation**

Outbreaks in domestic and zoo animals have been traced to a variety of sources like animal origin feed, and contaminated effluent water from tanneries or other animal product processing, but naturally contaminated soil is the primary source of infection for free ranging wildlife (Kaufman 1989). The primary route of infection is thought to be ingestion of anthrax spores. Spores are resistant to gastric fluid and when ingested and absorbed into the body, cause systemic disease from the germination and multiplication of the vegetative stage of the anthrax bacillus. The vegetative form elaborates a rapidly fatal exotoxin.

Soil contamination arises when the vegetative form of *B. anthracis* is exposed to environmental conditions favouring spore formation. This event usually occurs after the death of the host and subsequent leaking of contaminated fluid from body orifices. It was once believed that spore formation proceeds after exposure of the organism to aerobic conditions and is optimal at a temperature of 22°C (Titball and Manchee, 1987). However, sporulation is essentially a starvation response and it is not specifically the presence of atmospheric oxygen that stimulates spore formation but the depletion of nutrients in the bacterial microenvironment caused by drying of tissues and aerosolisation of body fluids caused when the carcass is opened (Dragon and Rennie, 1995). In intact carcasses, anthrax bacilli are destroyed by putrefaction within 4 days except when ambient temperatures are 5 to 10 °C where they can survive up to a few weeks (Choquette and Broughton, 1981). Spores are highly resistant to normal environmental temperatures and the effects of sunlight and drying. They can maintain their viability for years in dry soil where microbial activity is minimal (Sterne 1959; Choquette and Broughton 1981).
Ideas of how spores persist in soil have changed somewhat in recent years. The knowledge that spores can persist in dry soil for long periods may have led to the assumption that the population of spores in contaminated soil is static, and that once an area is contaminated with spores it stays that way indefinitely. (Pyper, 1964; Wilson and Russell, 1964; Stein, 1945). There is evidence that germination of spores is dependent on factors such as soil temperature and moisture content. Contamination by anthrax organisms can disappear in a few months because they cannot multiply and compete with microbes capable of more complex syntheses and production of antibiotic substances (Minnett, 1950; Sterne, 1959). This has caused some researchers to believe that an organism-spore-organism cycle occurs in soil where spores vegetate under favourable conditions of moisture and temperature and then sporulate when conditions become satisfactory. It is possible that in areas where the pH of the soil is higher than 6.0 and in an ambient temperature above 15.5°C, there is propagation of the organism in the environment creating a situation of increased risk for animals inhabiting the ecosystem. (Van Ness 1971). Conversely, soil that is unsuitable for this cycle because of acidity or biological competition causes elimination of the organism from the environment and infection of animals at that location is no longer possible (Minnett 1950).

It has been suggested that the typical pre-outbreak weather disturbance of rain creates suitable "incubator areas" for propagation of anthrax bacillus (Van Ness, 1971). These might be depressions in topography or flood plains where water has stood long enough to kill the vegetation that was underwater. This satisfies the ecologic requirement of alkaline soil enriched by vegetable matter, and creates an environment suitable for the cycle of anthrax organisms (Van Ness, 1971). In Europe outbreaks are confined to low lying marshland areas, rich in organic material, but in South Africa the occurrence of anthrax is just as common on dry areas, independent of moisture content in the soil (Choquette and Broughton 1981). However, it appears usual for an outbreak to occur after the marshes or flood plains have undergone considerable drying. The soil ecology at the time of infection is similar regardless of its original condition (Choquette and Broughton, 1981).

Dragon and Rennie (1995) have referred to these low lying geographic areas as “storage areas”, the idea being that rains and wet weather collect the spores from the surrounding environment and deposit the spores in the low-lying depressions. Rather than an incubator cycle of vegetative and sporulated bacteria mentioned above successive cycles of runoff and evaporation merely serve to concentrate the spores in these areas and as the water recedes the spores are deposited on the surface vegetation where they are readily available for consumption by susceptible herbivores.

**Epidemic factors**

The potential for the transmission of anthrax to other animals follows the death of an animal and the spread of the organism to other environmentally suitable areas. The biological requirements of the anthrax bacillus are well established and conditions that favour survival of the spores are similar on all continents (Sterne, 1959; Gainer and Saunders, 1989; Minett, 1950; Titball and Manchee, 1987; Van Ness, 1971). However, the factors that cause transmission and dispersion of anthrax can be as diverse as the ecosystems where anthrax is found.

The formation of an epidemic depends on further exposure of a population to the initial outbreak. This exposure can be brought about by dying or dead animals locally concentrating infective material in the form of discharges and excretions, or an opened carcass (Pyper and Willoughby, 1964; Sterne, 1959; Choquette and Broughton, 1981). These reservoirs of infective material increase the chance of further exposure in a population through direct contact, spread through wind and water movements or from vectors from the reservoir, like insects or scavengers and predators (Choquette and Broughton, 1981). Spores can be spread by adherence of contaminated dust and mud to the hair of wallowing bison or in dried blood and tissue on the fur and feathers of scavengers feeding on infected carcasses (Ebedes, 1975; Dragon and Rennie, 1995). Most vectors are the indirect variety, causing contamination of other geographic locations but some insects are thought to directly transmit the organism to susceptible hosts.
through biting (Gainer, 1987; Van Ness, 1971; Turell and Knudson, 1987). Not surprisingly, the species of vector involved is unique to the location of the epidemic, as is the role that each has in the spread of infection. Epidemic characteristics tend to be ecosystem dependent, making geographic features like water holes more significant to the African pattern of disease (Prins and Weyerhaeuser, 1987) and similarly, bison wallows are important to transmission cycles in Northern Canada (Dragon and Rennie, 1995).

Risk factors associated with anthrax outbreaks in domestic cattle herds have been identified using case-control studies of recent outbreaks in 2005 and 2006 (Mongoh, 2008; Epp, 2010). There were very few surprises in these studies. Vaccination was demonstrated to be protective from outbreaks and further, prompt vaccination in the face of an outbreak (as opposed to vaccination after more than a week from the first reported anthrax case) was shown to be related to decreased mortality. Pasture management factors such as shorter pasture grass length and high animal densities were positively correlated to the occurrence of anthrax on case farms as were the usual environmental factors such as wet pastures followed by a period of dryness.

Anthrax in North America

Anthrax in North America is thought to have been introduced during the 1700's by French settlers along the Mississippi delta and was first seen in deer in the marshes at the mouth of the Mississippi River (Fox et al., 1973; Kellogg and Prestwood, 1970). The disease subsequently appeared in bison (Bison bison) on the western plains and it is thought that this original contamination of the plains soil was responsible for later outbreaks in livestock (Stein, 1948). It has been suggested that extermination of mountain sheep in a region of Montana resulted from anthrax through the introduction of domestic sheep (Grinnell, 1928). Anthrax has been reported in deer in Florida, Louisiana, California and Texas and in moose (Alces alces) in Wyoming (Choquette and Broughton, 1981).

Anthrax was first diagnosed in Canadian wildlife in 1962 when it occurred in bison (Bison bison) in the Northwest Territories. Between 1962 and 1978 more than 1000 bison died in this area and Wood Buffalo National Park (WBNP) (Choquette and others, 1972). In 1963 and 1964 the disease was diagnosed in a few of the local moose population (Choquette, 1981). Every year between 1962 and 1978 except 1965 and 1966 anthrax was reported in the region near Hook Lake and Grand Detour, and in 1964 and 1967 the outbreaks included WBNP (Choquette et al., 1972).

Reports of disease occurrence in these areas do not discuss the existing environmental conditions at the time of, or immediately preceding the outbreaks of anthrax. One report mentions that local conditions favoured the persistence of soil contamination and the subsequent occurrence of outbreaks, but does not state what the local conditions were (Choquette et al., 1972). We might assume that having been declared an endemic area, the ecology of the outbreak zones typifies the alkaline bottomland that the anthrax bacillus has been shown to favour. There has been no reference in the reports of other factors predisposing the bison to anthrax (Choquette et al., 1972; Pyper and Willoughby 1964; Choquette and Broughton, 1981). These factors might be overpopulation, pre-existing disease other than anthrax, low food supply, low water supply or the hot, dry weather conditions preceded by rain or flooding that are described in outbreaks elsewhere (Van Ness, 1971; Kellogg and Prestwood, 1970). In 1989, Gainer and Saunders suggested, in an effort to explain the low numbers of B. anthracis spores recovered from the WBNP environment, that high ambient temperatures, rutting activity, high levels of insect activity and concentration of bison during the breeding season may have contributed to lower immune function and increased susceptibility to disease. They hypothesized that this may have led to fatal infections subsequent to relatively low-dose exposures to anthrax organisms. It was thought that contaminated grass or water was the primary source of infection for these epidemics (Choquette et al., 1972).
The perpetuation of the outbreaks was thought to be aided by the bison herd characteristic of roaming so that the organism was disseminated by diseased bison over large areas (Choquette et al., 1972). It was suggested that the means of spread to other areas and to Wood Buffalo Park included predation or scavenging on sick or dead bison by wolves, coyotes, foxes or bears. Anthrax spores have been found in the cloaca of gulls (Larus argentatus) that had fed on bison carcasses (Choquette et al., 1972).

Biting flies (Tabanus striatus, Hematobia irritans) have been incriminated in the transmission of anthrax in these, and other epidemics in North American animals (Pyper and Willoughby, 1964; Stein, 1945; Choquette and Broughton, 1981; Fox et al., 1973; Kellogg and Prestwood, 1970). A laboratory study using mosquitoes to feed on anthrax affected guinea pigs showed that at least 2 species of mosquitoes are capable of transmitting anthrax through biting and feeding (Turell and Knudson, 1987). These observations indicate that insects can act both as indirect and direct vectors in the spread of anthrax.

Dragon and Rennie (1995) convincingly demonstrate a gender bias in anthrax related bison deaths. Adult males appear to be much more susceptible to anthrax than females or sub adults of either sex. They suggest that breeding activity may be responsible for this higher mortality rate. Not only are the bulls under considerable physical and psychological stress during rut, decreasing their resistance to disease, but aggressive rutting behaviours such as stamping and wallowing greatly increase their exposure to anthrax spores. During hot dry weather this type of behaviour in incubator or storage areas would lead to large clouds of spore-containing dust which would then be inhaled by the bulls participating in the rutting behaviour.

After 1978 no deaths were observed in the park and surrounding areas until the summer of 1991 when anthrax was again found in bison from the Salt Plains area of Wood Buffalo Park. Thirty-four bison died in the outbreak and remarkably, deaths were seen in yearlings and calves (Broughton, 1992). It was reported that the park received heavy rainfall in the spring of 1991, and that the resulting flooding may have caused the spread and concentration of spores, or promoted vegetation in areas that had not been productive in recent years (Broughton, 1992). This epidemic ceased in mid August when the weather changed to colder temperatures. The colder temperatures would lead to increased humidity and less dust during wallowing and rutting behaviour, decreasing the risk of aerosol infection. Coincidentally, there was also an abrupt reduction in the population of biting flies in the area at this time (Broughton, 1992). In 1993 there was an outbreak involving approximately 172 deaths in the Mackenzie Bison sanctuary but this was followed by seven years of relative inactivity until 2000 when WBNP was again struck with an outbreak involving 100 cases in the Lake One (48) and Davidson Tower (52) areas. In 2001 there were 92 more deaths from anthrax in the Lake One area of WBNP as well as an additional 12 cases around Hook Lake in the Slave River Lowlands. In 2006 there were 26 additional cases at Hook Lake. In 2007 WBNP was struck again, this time in the Park Central area, which suffered the loss of 64 bison from anthrax. In 2010 all three major regions, Slave River Lowlands, WBNP and the McKenzie Bison Sanctuary, sustained anthrax losses of 45, 6, and 10 bison respectively.

Anthrax control measures in the bison population of Canada have been directed at limiting the spread of infection by hygienic clean-up of carcass sites and, until about 1974, annual round-up and vaccination of susceptible animals. Prior to about 1990, carcasses were burned, where possible, and buried with quicklime treatment (Choquette and Broughton, 1981). Current methods of carcass disposal involve an initial treatment of the carcass with a 10% formaldehyde solution (20-40 litres per carcass) which discourages scavengers such as bears or wolves and provides some superficial disinfection at the carcass site. This is followed by labour intensive and resource consuming incineration of carcasses. Each carcass consumes approximately 440 kg of stoker's coal, 220 kg of green wood, and 1400 kg of dried wood. A coal and green wood bed is prepared and the carcass is hand winched onto the bed. The dried wood is stacked on top of the carcass and the pyre is doused with 20 litres of diesel fuel and lit. Secondary burning is often performed to incinerate any remaining hair and bone material observed after the initial burn has been performed (Nishi, 2007). Early detection of dead animals and subsequent incineration are key to the control of anthrax in Canada's north (Nishi, 2002). Vector control has not been mentioned in any of the studies on anthrax in northern bison to date.
In 1963 an anthrax epidemic was diagnosed in White-tailed deer (*Odocoileus virginianus*) in Arkansas (Kellogg and Prestwood, 1970). The outbreak occurred on Beulah Island in the Mississippi River and by description and inference the ecology of the soil favoured the existence of anthrax spores and a spore-organism-spore cycle. For two months prior to deer mortality, drought conditions prevailed throughout the area (Kellogg and Prestwood, 1970). These factors are thought to greatly predispose an ecosystem to an outbreak of anthrax (Van Ness, 1971; Choquette and Broughton, 1981). Observations of range conditions indicated to researchers that the deer herd on the island exceeded carrying capacity and deer appeared to be eating from the ground. Water was available from three stagnant lakes and the Mississippi river. Coincidentally with the outbreak heavy concentrations of biting flies were observed (Kellogg and Prestwood, 1970). The authors of this report concluded that the conditions favouring the outbreak and formation of the epidemic were: previous occurrence of anthrax in the area, overpopulation in deer, low food supply, bottomland soil, high ambient temperatures and drought conditions, utilization of stagnant lakes for water supply and high numbers of biting insects. Carrion feeders or predators were not considered factors in the epidemiology of this outbreak (Kellogg and Prestwood, 1970).

Although anthrax has been reported in domestic species from virtually every state in the continental United States, and in deer from California, Florida, Louisiana, and Texas (Stein, 1952) there appear to be no detailed studies of the epidemiology or ecology of the disease in wild species other than those discussed here.

**Anthrax in Africa**

Anthrax has been well controlled over the last half-century by the vaccination of livestock, improvements in hygiene, animal husbandry and public health measures and it has become almost a forgotten disease in the western world. In African wildlife, which cannot be easily vaccinated and in which other aspects of control are not relevant, the disease remains a major cause of uncontrolled mortality in herbivores (Turnbull et al., 1991). The interplay between feeding behaviour of the host, condition of the habitat, ecology of the disease and physiological immunity of the host to the disease are all important in establishing disease. In Africa, ecological conditions favoring anthrax transmission from the environment tend to occur at the end of the dry season (Prins and Weyerhaeuser, 1987). Studies show that oral infection via spores is dose dependent. \(1 \times 10^7\) spores were required in domestic cattle to cause the peracute and sudden death type of infection while smaller doses usually cause subacute, nonlethal or inapparent infection. Spores in the environment must be concentrated in such a manner that potential hosts will be exposed orally to large doses. These situations may prevail in natural epidemics in open seasonally arid areas because moderate numbers of spores have been recovered from watering holes where potential hosts and scavengers concentrate (Schlingman et al., 1956).

If an animal encounters a high risk area, an increased chance of infection occurs from abrasions in the oral mucosa or perhaps in the intestinal tract (Blood et al., 1983). Close grazing of rough forage in dry environments can result in such abrasions (Van Ness, 1971; Blood et al., 1983). Postmortem findings in buffalo (*Syncerus caffer*) suggest that the mouth and/or the pharyngeal area is the main portal of entry of infection. In this study the lymph glands of the pharyngeal region on post mortem examination of anthrax diseased animals had the most chronic and severe lesions. This strongly supports the hypothesis that a primary pharyngitis precedes the systemic disease. Gastrointestinal lesions were the next most chronic lesions (McConnell et al., 1972). Ecological conditions determine the presence of rough, closely cropped stubble in the habitat. The utilization of the habitat by other herbivores, the population density, the rainfall pattern and water storage capacity of the soil are determinants in the growth of plants and the occurrence of rough forage (Prins and Weyerhaeuser, 1987).

Etosha National Park today, consists of 22,270 km\(^2\) surrounded by wire and electric fencing. Etosha has a saltpan which is approximately 6,133 km\(^2\). Most of the time the saltpan is dry, but periodic flooding from the Ekuma and Oshingambo rivers causes springs to form where limestone beds contact impervious clay. The springs and flooding of the Pan provide numerous drinking places for massive congregations of animals (Cloudsley-Thompson, 1990). The vegetation around the edge consists of grasses, shrubs with bushveld
nearby and is classified as a saline desert with dwarf shrub savanna (Cloudsley-Thompson, 1990; Ebedes, 1975). These areas of water enriched with vegetable matter provide the ideal environment for anthrax spores. Samples of water, mud and soil from low lying areas and shallow seasonal rivers used as watering holes in Etosha were found to be highly contaminated with anthrax spores. Water was considered to be the main source of infection in Etosha (Ebedes, 1975).

Between 1967 and 1974 anthrax was responsible for 54.5% of the total mortalities in Etosha National Park. The mortalities occurred in: plains zebra (Equus burchelli), blue wildebeest (Connochaetes taurinus), springbok (Antidorcas marsupialis), elephant (Loxodonta africana), gemsbok (Oryx gazella), kudu (Tragelaphus strepsiceros), giraffe (Giraffa camelopardalis), ostrich (Struthio camelus), eland (Taurotragus oryx) and cheetah (Acinonyx jubatus) (Ebedes, 1975). Anthrax was responsible for 54% of the mortality of plains zebra and 39% of blue wildebeest. From 1976 to 1978, 43% of zebra and 62% of blue wildebeest died. The zebra population dropped from 18,000 to 9166 by 1978 and the wildebeest suffered a similar decline. The estimated population of wildebeest was 30,000 in 1965 and from 1976 to 1978 the population dropped from 3300 to 2500. Anthrax, as well as, uncontrolled veld burning and habitat deterioration and bush encroachment caused overgrazing which effectively eliminated wildebeest (Ebedes, 1981).

Adult zebras and wildebeest of both sexes appeared to be more susceptible than immature animals, but indirect fluorescent antibody tests on zebra from enzootic and anthrax free areas indicated that immunity occurs at greater than three months of age (Ebedes, 1975). The discrepancy between adult verses immature animal mortality was more likely due to feeding habits (Mbise et al., 1991). The deaths recorded amongst gemsbok and eland were the first record of anthrax in these species. It is suggested that stress factors, including nutritional deficiencies, are responsible for causing breakdowns in immunity (Ebedes, 1975).

The anthrax problem within Etosha National Park resulted from the repercussions of management policy and manmade environmental changes. Initiatives that were designed to protect and encourage game led to the demise of several populations. Increased tourism to Etosha necessitated the provision of additional watering holes allowing tourists to view game. They also reduced animal movement. The erection of a fence along the 850 km boundary was aimed at preventing the spread of foot and mouth disease and keeping animals within the park and poachers out. This prevented normal migration of nomadic herds such as the blue wildebeest in times of drought (Cloudsley-Thompson, 1990; Ebedes, 1975; 1981). Anthrax was not evident and no epizootics were previously recorded in Etosha prior to the disturbance of the natural conditions and environment (Ebedes, 1975).

Overconcentration of animals in Etosha in the traditional wet season dispersal areas resulted in range deterioration. The restriction of normal migration, high densities of animals and subsequent overgrazing, forced animals to consume plant material closer to the ground. The change in grazing pattern predisposed animals to the ingestion of spores. Habitat deterioration in areas when climatic and soil conditions were suitable for the survival of anthrax spores also played a significant role in the development of disease in susceptible animals (Ebedes, 1975; 1981). This situation demonstrated a definite relationship between the overconcentration of animals in Etosha causing overutilization of habitat and anthrax epizootics. Anthrax occurs in a cyclical nature with periodic build-up of populations and over utilized habitats that may precipitate outbreaks (Ebedes, 1975; Piennaar, 1967).

The construction of gravel roads played a crucial role in the spread of anthrax. Large gravel pits were dug for road fill, which subsequently filled with water that became alkaline. This environment provided ideal environmental conditions for anthrax spores (Cloudsley-Thompson, 1990; Ebedes, 1981). In addition, the gravel pits were bigger and deeper than the pans and retained water for longer periods which increased the amount of time migrating animal would normally spend in the wet season areas (Ebedes, 1975). Studies showed that there was a geographical association between the regions of highest incidence of anthrax and artificial water holes or gravel pits. Experiments, however, have provided no evidence that B. anthracis can multiply in water from either type of water hole without added nutrients. Vegetative forms appear to die off rapidly while the number of spores remains constant (Turnbull et al., 1991).
Anthrax epizootics in Tanzania wildlife have been recorded in Tanangire and Arusha National Parks in 1974 and in Lake Manyara National Park in 1962, 1974 and 1984. The 1974 outbreak in Arusha National Park and the 1962 and 1974 outbreaks in Manyara National Park affected mostly cape buffalo, while the epizootics in Tanangire and Lake Manyara National Parks in 1974 and 1984 affected mostly impalas (Aepyceros melampus). The Tanangire National Park covering about 26000 square kilometres in the dry season has the second highest density of wildlife in Africa (Mbise et al., 1991). The close association of livestock and game animals may have facilitated the disease transfer. Other possible sources of the infection include: migrating animals form Manyara National Park to the northwest of Tanangire National Park and livestock passing between Tanangire and Lake Manyara National Parks on their way to northern and eastern Tanzania (Mbise et al. 1991).

During the wet season, wildebeest and several other species were concentrated in short grass regions and during the dry season most of the animals dispersed into the woodlands. Anthrax mortality coincided with the movement of wildebeest onto the short grasses (Gainer, 1987). Field observations indicate similarities in the most favorable conditions for spore survival and the actual ecology of Tanangire Park.

Conditions favouring incubation and perpetuation of anthrax spores in Tanangire National Park included: adequate moisture and temperature, the alkaline soils and water pH of 6 to 10.8 and the organic materials in the water. Soils in the alkaline parts of the park were popular as licks for animals and good incubator foci for B. anthracis (Mbise et al., 1991).

Another contributing factor to the causal web of anthrax in Tanzania is drought. During the dry periods of three consecutive years, swamps, pools, marshes and bottom lands dried out and became available for grazing. This situation attracted an overabundance of animals and subsequent over utilization of vegetation, close grazing and ingestion of infected soil. The Tanangire River became the only available source of water, concentrating large numbers of animals in a relatively small area. Scavengers such as hyenas, jackals, foxes, vultures, some predators, blood sucking flies and non-biting Diptera sp., all present within the park, may have played a major role in disseminating the organism. Spread of the disease was aided by the contamination of soil waterholes, grass and forage with excreta and discharges of infected animals (Mbise et al., 1991).

The apparently high susceptibility of impalas at Lake Manyara relative to other species may be due to behavioral differences. It was suggested that periodic build up of the impala population in already over utilized habitats could precipitate outbreaks and that stress factors such as nutritional deficiencies and overcrowding could also be responsible for precipitating the breakdown of immunity (Ebedes, 1981). Adult impalas of both sexes appear to be more susceptible to anthrax than immature animals, possibly because of behavioral differences (Ebedes, 1981). Within days after the onset of rains, attack rates diminished dramatically. Rain probably helps to wash contamination from the vegetation and to dilute the salinity. In addition, through regenerative growth of vegetation, there is a reduction in overconcentration of animals and an end to grazing close to the soil (Mbise et al., 1991).

Periodic outbreaks of anthrax in Kruger National Park have resulted in the death of many hundreds of game animals and pose a major threat to endangered species such as the roan antelope (Hippotragus equinus). Studies carried out in the park suggest that there are differences in immunity between species. In outbreaks of anthrax, the eland is not as susceptible as the bushbuck (Tragelaphus scriptus) and the sable antelope (Hippotragus niger) not as susceptible as the closely related roan antelope (Pinaar 1961; 1967). There may be genetic difference between the impala populations of Lake Manyara National Park and Kruger National Park since impala were once nearly eliminated at Lake Manyara but hardly affected at Kruger (Pinaar, 1967). Effectiveness of immunity can be modified by stress such as overcrowding or overgrazing, lack of micronutrients or loss of physical condition (Pinaar, 1967; Prins and Weyerhaeuser, 1987). These factors and the likelihood of grazing on more alkaline soil tend to occur at the end of the dry season (Prins and Weyerhaeuser, 1987). Environmental conditions within the Kruger National Park often approach the optimal conditions for B. anthracis survival.
Blow-flies play an important integral part in the maintenance and spread of anthrax during epizootics in northern Kruger National Park. *Chrysomyia albiceps* and *C. marginalis* are the most abundant carrion blow-flies in northern Kruger National Park. Flies arrive in large numbers soon after the death of an animal to feed on blood at hemorrhaging body orifices (Braack and Retief, 1986; Braak and de Vos, 1990). They imbibe bacteria contaminated blood and other fluids at the carcass site, then fly to vegetation in the immediate area and rest for lengthy periods in the shade (Braak and Retief, 1986). Reject food is voided via the anus, and partial regurgitation of "vomit drops" occurs. In addition, like many other hematophagous insects, these flies engage in hemoconcentration, a process where many but not all erythrocytes and other blood constituents are selectively retained in the body while a clear fluid is ejected from the anus. These are "discard droplets" and are different then the darker and pastier excreta which is passed several hours later and called fecal droplets. Because the process of hemoconcentration is very rapid, the major portion of the meal and infective droplets are discarded in the immediate vicinity of the carcass ingestion (Braak and de Vos, 1990). In this manner anthrax spores pass unharmed through the flies and are deposited in large numbers on leaves. The process increases the probability of herbivorous mammals ingesting the pathogen in fatal doses (Braack and Retief, 1986). This is an ideal source and major route of infection for browsing herbivores such as the kudu (Braak and de Vos, 1990).

In outbreaks involving medium to large mammals such as in the 1960 epizootic which accounted for 79.2% of the browser species losses and 20.8 % of the grazing species, kudu appear to be the most vulnerable. Kudu losses were 15% of the population. Waterbuck (*Kobus ellipsiprymnus*) and buffalo sustained 7.12% and 5.50% losses respectively, illustrating the disproportionate loss of life between browsers and grazers. Evidence suggests that the exceptionally heavy kudu mortalities are correlated with their feeding habits. The highest concentration of flies on trees is found between a height of 1 and 3 m; the same height that kudu do most of their browsing. Like kudu, blow flies are significantly more abundant in wooded areas. (Braak and de Vos, 1990). Because of their feeding habits kudu had a greater chance of ingesting enough anthrax spores to cause disease than grazing animals.

In spite of differential interspecies immunity, it is probably the specific feeding habits of host animals that determine the differences in mortality rates. The differential mortality between individuals may be due to inherent resistance but the potential of blowflies to sufficiently increase the ingested dose of bacterial spores should not be underestimated. The fact that the blow-flies deposit the majority of discard droplets in the immediate vicinity of the carcass also conveniently facilitates disease control programs as the area can be burned (Braak and de Vos, 1990).

Blow flies can cover considerable distances in their search for carrion. In several studies *Chrysomyia* were reported to have travelled between 25 and 63.5 km. Despite these findings, the post-feeding habit of resting on nearby vegetation for lengthy periods after engorgement and depositing the bulk of their potentially infective droplets in the immediate vicinity of the carcass mitigates against their causing infection at locations well removed from the carcass. (Braak and de Vos, 1990).

It has been suggested that from 1959 to 1961 the spores of *B. anthracis* in the Kruger were disseminated chiefly by vultures scavenging from dead animals, feeding via watering places in order to bathe or drink (Pienaar, 1961). Studies suggest that vultures are able to rapidly rid themselves of contamination by visiting watering holes immediately after gorging themselves. Blood adhering to their feathers is washed off and they may also regurgitate ingested infected material into the water. The behaviour of vultures therefore contributes to the perpetuation of the disease in an outbreak area.

Vultures are capable of spreading anthrax bacilli spores over vast areas by virtue of their scavenging habits and flight distances and are likely the main agents for long-distance dispersal of anthrax, blowflies causing only local dissemination but with the potential for explosive local spread (Braak and de Vos, 1990). The isolation of *B. anthracis* from both carnivore feces and vulture droppings has been reported. Spores have been isolated from fecal material from vultures for up to two weeks after feeding on an infected carcass. This has implicated the involvement of scavengers and predators in the spread and perpetuation of anthrax in wildlife (Turnbull et al., 1991). Vultures that were fed the vegetative form did not shed anthrax spores.
Sporulation would have to take place sometime after defecation but once they have been seeded in the environment the spores have the potential to undergo the hypothesized spore-organism-spore cycle, reaching sufficient concentrations to infect susceptible species.

Vultures, on the other hand, may reduce the amount of infective material in the environment by rapid removal of a carcass since a carcass must be open and exposed to air for several hours for spores to form (Dillman, 1956; Mundy and Brand, 1978). The carrion eaters themselves appear have innate resistance to the disease (Cloudsley-Thompson, 1990; Turnbull, 1992).

Naturally acquired anthrax specific antibodies are rare in herbivores but common in carnivores. Titres appear to reflect the prevalence of anthrax in the carnivore’s ranges and the feeding habits of that particular species. Antibody titres in lions (Panthera leo) are frequently high in comparison to jackals (Canis mesomelas) which have relatively low titres. This can be explained by the fact that jackals have a more varied diet than lions and have a lower dependence on anthrax carcasses for their food and hence a lower exposure to anthrax (Turnbull et al., 1992) The titre to B. anthracis in cheetahs is similarly low because they seldom return to their kill and scavenge except in extreme situations (Jager et al., 1990). The protective action of antibodies in carnivores against developing the clinical disease has not been determined. It is possible that stress factors may play a similar role in the development of disease in carnivores as they do in herbivores (Ebedes, 1975; Turnbull et al., 1992).

**Immunity**

It has been suggested that outbreaks of anthrax are not a result of simple exposure of susceptible animals to a high risk environment but that there are variable levels of immunity within a population, and that environmental factors cause the expression of the disease in compromised individuals (Gainer 1987; Gainer and Saunders, 1989). It is argued that low level exposure occurs in endemic areas and given the variability in susceptibility to natural infection it is not the infective dose of spores presented to the wild animal that determines disease, but the level of resistance at the time of exposure (Gainer, 1987; Gainer and Saunders, 1989).

Gainer (1987) proposed that anthrax has a subclinical dormant stage that reverts to the peracute infection when the host resistance is modified by environmental and behavioural factors. A serological survey found that populations indigenous to anthrax areas reflected widespread exposure to low doses. Such populations had a high prevalence (sic) of antibodies to anthrax (Provost et al, 1974 as referenced in Gainer & Saunders, 1989). A more recent study using ELISA testing for detecting anthrax antibody in white-tailed deer found no difference in the antibody levels from animals in anthrax endemic and non-endemic areas (Peterson et al, 1993). There may be a species difference in the immunologic reaction of wildlife to anthrax exposure.

It was suggested that Tabanid flies are responsible for low levels of exposure to populations at risk causing subclinical disease or a carrier state in resistant animals. Furthermore, these same levels of exposure that would normally be below the threshold dose for this disease are thought to result in fulminating disease in compromised animals because of the environmental and behavioural stresses commonly associated with anthrax outbreaks (Gainer & Saunders, 1989). Other evidence for subclinical infection might be the finding of anthrax organisms in the retropharyngeal lymph nodes of clinically unaffected, but apparently exposed animals (Gainer, 1987; McConnell, 1972)

Interspecies differences in resistance to infection by B. anthracis are thought to be related to the ability of immune mechanisms to deal with the capsule associated with virulent bacilli. It is this capsule that provides the organism with the ability to resist leucocytic activity in the host. Animals that possess the means to destroy the capsule are more or less resistant to infection because normally functioning immune systems can easily render the bacilli non-invasive (Hedlund, 1992).
Vaccines

In its vegetative state *Bacillus anthracis* produces a three part toxin made up of protective antigen (PA), edema factor (EF), and lethal factor (LF). Protective antigen is critical to the activation of both LF and EF and without the combination of PA and EF there is no edema; without PA the LF is not lethal. These combinations give edema toxin (ET) and lethal toxin (LT). Production of factors is plasmid mediated, as is the production of the enzymes needed for the synthesis of the capsule of *B. anthracis* that inhibits phagocytosis (Hedlund 1992). Immunity to infection from anthrax bacillus depends on antibodies against protective antigen, because without PA, EF and LF are not pathogenic (Hedlund, 1992). Protection has generally been attributed to toxin neutralizing antibody to the toxin components, however, studies indicate that antibody to vegetative cellular antigens may also be important (Ezzell, 1986).

The Sterne strain vaccine was developed about 1930. It is a highly effective live spore vaccine which in the vegetative state is non encapsulated but toxigenic and therefore highly effective in inducing a protective immune response. Because it lacks a protective capsule it is non-pathogenic in all but a small group of animal species such as goats, llamas and some strains of mice (Hedlund, 1992). There have been no improvements on this vaccine since it has been so widely accepted. However, since the discovery of the gene sequencing for all three components of anthrax toxin in plasmids there has been renewed interest in synthesis of purified components for use in anthrax vaccines (Hedlund, 1992). Problems still exist with this approach to immunizing animals because other immunogens that remain uncharacterized are associated with live spore vaccines, and acellular, extracted vaccines give inferior immunity at this point in time (Hedlund, 1992).

**Conclusion**

The list of contributing factors in anthrax outbreaks shows the complexity of what is clinically a straightforward bacterial disease affecting mostly ruminants. The risk factors associated with infection involve the classically described interactions between host, infective organism, and environment. The correct alignment of these factors allows the expression of the disease. The exact mechanisms and their relative importance remain contentious even though anthrax is one of the oldest documented epidemic diseases known to man.

**References**


