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Anthrax – A Model of the Universality of Medicine

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With regards to education and work habits, we divide medicine into two major fields – human and animal. However, their paths are amazingly interwoven. Understanding how these paths are interconnected is becoming more important with the rise in human and livestock populations, the increase in global movement, and environmental changes. The universality of medicine is exemplified by anthrax, an ancient adversary of both man and beast. There has been heightened public concern recently due to the potential use of anthrax as a biological weapon. In the last three years, apprehension about this disease on the Canadian prairies has increased due to repeated livestock outbreaks in Manitoba, Saskatchewan, and Alberta. This issue of *Large Animal Veterinary Rounds* reviews the history of this disease, the biological conditions necessary for its transmission and infection – both naturally and as a bio-weapon – its clinical signs and symptoms and strategies for treatment, prevention, and control.

History

We do not know when anthrax first emerged, but it has probably caused sickness for thousands of years. The fifth and sixth plagues in the bible (Book of Exodus, approximately 1300 BC) may have been anthrax, since the fifth plague affected livestock and the sixth caused skin problems in humans. In 300 BC, Hippocrates described an anthrax-like disease. More substantiated timelines are:

25 BC – Virgil's *Georgics* depicts a disease in livestock and wild animals that authorities today would classify as anthrax.

1100-1500 AD – Medieval Europe has a continuing problem with anthrax, called the "Black Bane."

1700 – One of the first reports of an incidence of anthrax in North America, involving deer in the Mississippi delta.

1800s – First probable incidence of occupational respiratory infectious disease was described in English wool sorters, due to the aerosolization of anthrax spores from industrial processing of goat hair and alpaca wool.

1876 – Robert Koch firmly establishes that disease is caused by a microbe; he uses anthrax to establish his postulates.

1881 – First vaccine developed for a bacterial disease by Louis Pasteur.

1903 – McFadyen develops a polychrome methylene blue stain for the anthrax bacterial capsule; this is still the most reliable and simple stain to use.

1911 – Ascoli develops a precipitin test (thermostable antigen test) to assess if animal by-products are anthrax-free and safe to use. This test detects anthrax capsular remnants in tissues and is still used today.

1914-1918 – Use of anthrax as a weapon probably by the Germans during World War I and used to contaminate animal feed and infect livestock.



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1932-1945 – Japanese in occupied China use civilians as human “guinea pigs” to test anthrax as a biological weapon (Unit 731). The researchers were granted immunity after World War II on the condition that they disclose their information.

1937 – Sterne develops an effective veterinary vaccine using a non-encapsulated live anthrax strain. This is the basis for the vaccine most commonly used today.

1939-1945 – The USA and Britain make anthrax a weapon and Britain explodes a test bomb on Gruinard Island off Scotland. In 1979, Britain starts the decontamination of Gruinard Island, since the spores are still viable. The process takes 8 years, using 280 tons of formaldehyde and 2000 tons of seawater to complete.

1945 – Anthrax kills over one million sheep in Iran.

1954 – Smith and Keppie demonstrate a factor in the serum of anthrax-infected animals that is lethal when injected into animals. Previously, death was attributed to the “log-jam” effect of massive numbers of anthrax bacilli in blood.

1957 – Largest outbreak of inhalation anthrax recorded in the USA, due to processing goat hair from Pakistan; 4 of the 9 infected workers die.

1969 – Nixon terminates USA involvement in anthrax research as a biological weapon.

1972 – Biological Weapons and Toxins Convention signed by most countries to prohibit research and production of biological weapons. Russia and Iraq, who co-signed the convention, subsequently continue bio-weapon programs. There is concern that other countries may be doing the same.

1979 – The largest epidemic of inhalation anthrax in the 20th century occurs, due to the accidental release of anthrax spores (several kilograms) from a military research facility in Sverdlovsk, Russia; 68 human deaths reported from 96 infections, but possibly up to 600 deaths occurred. The Russians initially state that the outbreak was from contaminated meat, but in 1992, they confirm that the epidemic was from a military accident. Most affected people are within a few hundred meters of the release site, but animals are affected more than 50 km from the site.

1978-1985 – The largest reported epidemic of human anthrax occurs in Zimbabwe, with 10,000 cases. The cases are primarily the cutaneous form, with rare gastrointestinal and inhalation forms.

1987 and 1988 – Large outbreak in Zambia kills over 4000 hippopotami.

1991 – Iraq deploys anthrax-laden Scud missiles and rockets during the Gulf War; they are reported to have over 6000 L of anthrax slurry in storage.

1993 – Aum Shinrikyo, a doomsday cult in Japan, becomes the first known terrorist group to culture and use anthrax. The

group releases anthrax at least twice from the roof of their 8-story headquarters; it was not effective.

1997 – Russian scientist develops an anthrax strain resistant to the Russian vaccine.

1997 – An animal outbreak in Ghana causes 185 human cases and 26 deaths.

1997-present – Many anthrax hoaxes reported in the USA; sometimes up to two or three a day.

1998 – L. Harris caught with a vaccine anthrax strain in the USA. Harris, a microbiologist with neo-Nazi anti-government affiliations, has published information on how to obtain anthrax and use it as a bio-weapon.

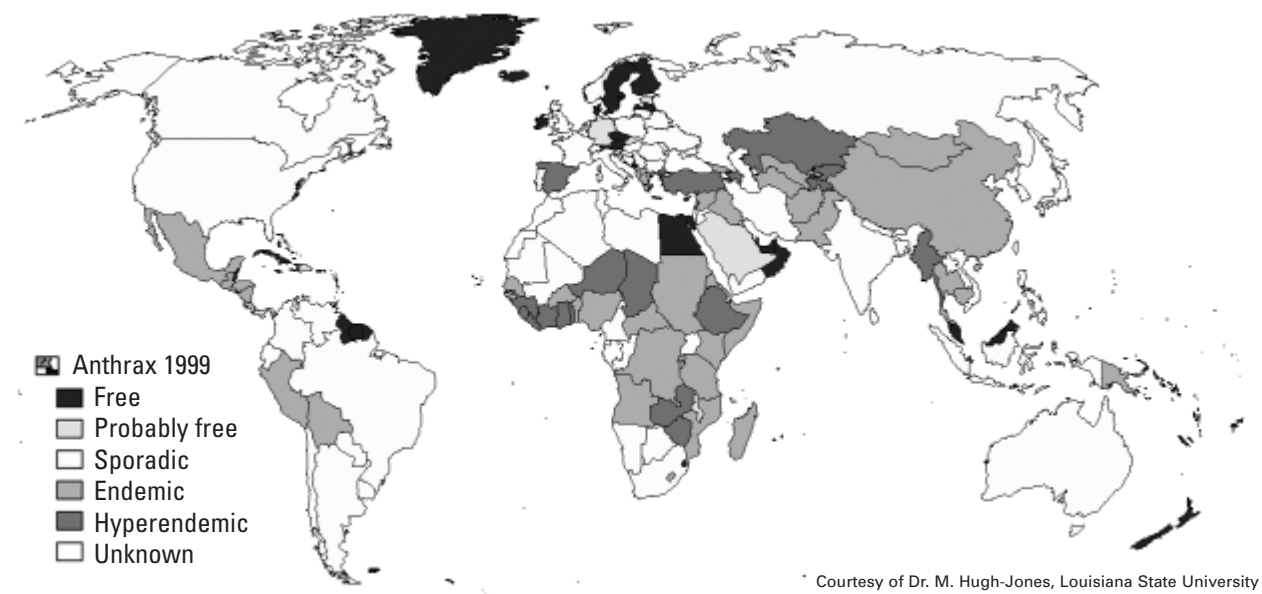
2001 – First reports in the USA of confirmed cases of anthrax due to intentional exposure. The contact is through anthrax spores in mailed letters or packages; perpetrators are unknown, but suspected to be domestic terrorists.

The cause of anthrax

The disease anthrax is caused by *Bacillus anthracis*. The name is derived from the Greek word *anthrakis* for coal, due to the colour of skin lesions and the almost black appearance of blood in infections. The bacterium occurs in vegetative and spore forms. The vegetative form is a large, fragile, facultative anaerobic, non-motile, encapsulated, Gram-positive rod, about 1 to 1.5 μm by 3 to 10 μm in size. When nutrients are exhausted and the rod is exposed to oxygen, spores of about 1 μm in diameter form within the cell. A very thick spore wall makes it able to survive extremes of heat, cold, pH, desiccation, chemicals, and irradiation. Spores may last for 50 years or more. In Russia, viable anthrax spores were found in an archeological dig of a village likely abandoned due to anthrax in 700 AD. The stability of the spores varies with the conditions in which they are formed. Temperature, pH, oxygen availability, the presence of cations such as magnesium and calcium affect their hardness and the rate and extent of spore formation. Spores germinate into the vegetative form at 8° to 45°C, pH 5 to 9, humidity greater than 95%, and the presence of adequate levels of certain amino acids, nucleosides, and glucose. Research suggests that the nutrients required are unlikely to be present in the environment, making the organism virtually an obligate pathogen.

The restricted and limited time for bacterial growth within animals helps explain why *B anthracis* is remarkably monomorphic with few natural strains. Human manipulation, however, has produced more variation and more virulent forms. Virulence is primarily due to an antiphagocytic capsule and an exotoxin consisting of three proteins. Transcription of the genes for virulence factors only occurs when the bacterium is in the log phase of growth, with increased carbon dioxide, bicarbonate, and temperature in the immediate environment. The capsule is encoded in plasmid pX02. The capsule is not

Figure 1: Worldwide distribution of anthrax



toxic, but it protects the bacterium from phagocytosis. Without the capsule, *B anthracis* is nearly avirulent. Another plasmid, pX01 encodes for the toxin, which has three factors: Edema factor, causes edema; Lethal factor, essential for lethal effects although the mode is not clearly understood; and Protective antigen, induces protective antibodies. The Edema factor and the Lethal factor, alone or in combination with each other, are inactive. They require the Protective antigen to bind them to the host cell. The maximum effect on the host cell is seen when all three factors are present.

Political, geographic, climatic and ecological factors

Anthrax is a global problem and on the List B of reportable disease with the OIE (Office International des Epizooties). List B diseases are transmissible diseases of social-economic or public health importance and they affect the international trade of animals and animal products. List B diseases are reported annually. List A diseases are reported immediately eg, foot-and-mouth disease.

Over the last 30 years, control programs have reduced the incidence of anthrax. In the year 2000, however, 48 countries still reported cases. The disease remains common in many countries in Africa and Asia, several European countries, multiple areas on the American continent, and in certain regions of Australia (Figure 1).

It is unknown how or when anthrax first came to North America. It is suspected that migrating buffalo were of major importance in the spread of anthrax on the prairies. This is supported by livestock outbreaks in Alberta that are often found to

be associated with old bison remains. Livestock movement along old cattle trails in the 1800s also probably played a role in the dissemination. DNA fingerprinting shows that a few recent livestock anthrax cases, within non-traditional areas of the North American continent, originated from imported African bone meal. This is a common problem in the United Kingdom where half of anthrax cases are due to imported feed, primarily bone meal. A human Canadian case in 1991 was linked to an imported sweater. In Canada, there has been a continual anthrax problem in bison of the McKenzie Bison Range, as well as in the North West Territories and in Wood Buffalo National Park of northern Alberta. It is sporadic in southern Alberta, Saskatchewan, and Manitoba, with the rare outbreak elsewhere.

Outbreaks usually occur in areas where the soil is heavy in organic matter, high in calcium or magnesium, and with neutral to alkaline pH. There are two theories why certain areas become “hot spots” for anthrax. The “incubator” theory is based on the soil type that allows the build-up of anthrax spores due to their periodic germination and vegetative growth in the environment. The second theory purports that hot spots are formed because the soil favours the formation of numerous, hardy, spores when anthrax does occur. Undetected “silent” cases of anthrax in these areas are suspected to play a role. Although scavengers may not open up the carcasses resulting from the outbreak, the large number of bacilli present in bloody discharges still contaminates the area. Spores may be further concentrated by drainage patterns with periodic flooding and drying out of wet areas. Although the issue remains unsettled, the incubator theory has lost favour based

on the fastidious requirements for vegetative growth by anthrax bacilli.

Outbreaks often occur at multiple sites and within a short period, particularly when certain weather conditions arise, thus making it possible to predict anthrax years. On the prairies, this pattern consists of unusually wet weather in the early summer followed by a dry, hot, late summer and early fall. The first cases are usually seen in late summer and early fall when dried out wetland areas can be finally grazed. The occasional winter outbreak of anthrax is usually due to hay harvested from fields flooded in the spring.

Other factors involved in outbreaks include overgrazing, abrasions, pasture drainage changes or disturbances, use of standing water for drinking, depressed immune systems, and increased numbers of large biting insects such as horse-flies. For example, in Alberta, anthrax is more common in areas with selenium deficiencies and in rutting young bison.

Anthrax bacilli generally enter the body through inhalation, a break in the skin or a break in a mucosal surface. Lesions in the gastrointestinal mucosa are thought to be the most common site of entry. Insect bites also may play a major role in certain areas and in some species (eg, blow-flies in Kudu anthrax in Kruger National Park, Africa, and Whitetail Deer anthrax in Texas).

Once spores have penetrated the skin or mucosal barrier, they are ingested by macrophages and germinate to vegetative bacilli with a capsule and exotoxin production. No underlying lesion is necessary for entry with inhalation anthrax. Spores are taken up by macrophages within alveoli and transported to regional lymph nodes where germination occurs.

Use as a biological weapon

Research into anthrax as a biological weapon started over 80 years ago. Currently it is estimated that 7 to 17 countries may be using the pathogen as part of their military weaponry because:

- Spores can be cheaply produced and stored for long periods of time.
- Spores are very hardy and can withstand many delivery mechanisms, including bombs.
- Virulent strains have high potency with very little mass; theoretically, 1 gram of spores can kill 100 million people. This is 100,000 times deadlier than the deadliest chemical.
- Strains can be produced that are resistant to common antibiotics such as penicillin and tetracycline derivatives.
- Spores may not be easy to detect, as they are odourless and very small; in large quantities they form a brown fine powder similar to cinnamon or cocoa.

Table 1: Clinical signs for anthrax in animals

Cattle, sheep and deer

- Sudden death most common
- May see hematuria, bloody diarrhea, blood tinged milk, staggering, difficult breathing, trembling, collapse, death
- May be ill for 1 to 2 days, sometimes 5 days
- May be initially excited and may charge, this is usually followed by depression

Horses, dogs and pigs

- Localized involvement of throat with marked swelling
- Intestines often involved, causing colic and diarrhea
- Sudden death sometimes seen in horses (eg, Alberta), sometimes with nervous signs and brisket edema
- Death often occurs in 2 to 4 days

When anthrax is used as a bio-weapon, aerosolization is the most effective delivery. The length of time anthrax can remain as an aerosol is unknown, but it is unlikely that the spores can remain aloft for more than one day. Once the anthrax is deposited onto a surface, experience from Sverdlovsk suggests that re-aerosolization is not of major concern since no case could be attributed to that phenomena.

Clinical signs of disease

All warm-blooded animals are susceptible to anthrax, but there are species differences in resistance. In general, cattle and sheep are most susceptible, followed closely by goats and horses. Humans are of intermediate resistance. Swine and carnivores are relatively resistant and birds and reptiles are very resistant. The clinical signs divided by species are described in Table 1 and Table 2.

Human health implications

There are about 2000 human cases of anthrax each year that are usually the cutaneous form contracted from animals or animal products. Anthrax does not spread between people. The highest numbers occur in Africa, the Middle East, and central and southern Asia. Canada had one case in 1991 and another in 2000. The anthrax source for humans is often categorized as:

- Non-industrial; usually from infected animals or carcasses. It affects farmers, veterinarians, butchers, knackers, and is almost always the cutaneous form.
- Industrial; generally from handling contaminated hair, hides, wool, or bone meal. Individuals are at risk for inhalation and cutaneous anthrax.

Table 2: Clinical signs of the three forms of anthrax in humans

Cutaneous

- 95% of anthrax cases, 1 human affected for every 10 livestock cases
- Incubation period 1 to 5 days, possibly up to 12 days – no latency
- Needs a cut to get in – often on face as touched with gloves
- First sign, a small papule (like an insect bite), often itchy; 1-2 days later vesicles develops (up to ~1-2 cm in diameter) with serosanguinous fluid containing many bacteria and few leukocytes
- Vesicle ruptures leaving a necrotic ulcer, which develops a black eschar; the latter separates after 2-3 weeks, often leaving a scar; usually painless but edematous and edema may be marked
- May see satellite vesicles that may have pus and/or pain if there is a secondary infection
- May see fever, malaise, headache, localized lymphadenitis
- Mortality – unusual

Inhalation anthrax

- Incubation period of 1 to 6 days, sometimes up to 60 days, due to spores in the mediastinal lymph nodes that do not germinate (Reason for delayed germination not apparent)
- Initially flu-like symptoms with malaise, fatigue, fever; may see non-productive cough, mild chest discomfort; no pneumonia
- Persists 2 to 3 days and may see short period of improvement
- Sudden onset of increasing respiratory distress; may have edema of chest and neck
- Characteristic widening of mediastinum; anthrax is almost the only cause of acute mediastinal lymphadenitis.
- There are often pleural effusions
- After onset of respiratory distress, patients usually die from shock 24 to 36 hours later.
- Underlying pulmonary disease may increase risk
- Meningitis in 50% of cases, may present with seizures
- Mortality near 100%

Oropharyngeal and gastrointestinal anthrax

- From ingestion of insufficiently cooked, infected meat; one human case per 30 to 60 infected animals eaten.
- Incubation period of 2-5 days; patients may have severe sore throat or oral ulcer often with fever, toxicity and swelling
- In gastrointestinal anthrax, nonspecific vomiting, fever, nausea, followed usually by severe abdominal pain
- May present with severe acute abdomen, hematemesis, massive ascites, and diarrhea
- 50% mortality

A third category for bio-terrorism may need to be added.

How anthrax is diagnosed

In Canada, anthrax is a “reportable disease.” Federal legislation requires all suspected anthrax cases to be immediately reported to the nearest Canadian Food Inspections Agency

(CFIA) Animal Health District Office. The telephone number for the local District Office can be found in the blue pages of the telephone book under the federal government. If the local District Office staff cannot be reached, there is a weekend anthrax emergency number available during the summer months (May 1 to September 30; Tel: 403-308-1131).

In most veterinary cases, death without being seen as sick (sudden death), will be the presenting problem. There are many more common causes of sudden death, such as clostridial infections, bloating, lightning or electrocution, acute interstitial pneumonia, poisonings (eg, lead, high salinity water), and deficiencies (eg, hypocalcaemia or hypomagnesaemia). However, due to the serious consequences of anthrax, it is still an important differential diagnosis to rule in or out whenever an animal dies after a short period of illness.

Factors that increase the likelihood that an animal may have died from anthrax include:

- The presence of dark, unclotted blood leaking from orifices, occurring shortly before or at the time of death with rapid bloating and decomposition of the body
- Failure of rigor mortis
- History of anthrax in area

In addition, it is useful to consider clostridial vaccination status (unvaccinated animals are more likely to die from clostridial infections); the environment and weather (lightning, recent pasture change, flooding or wet conditions followed by dry hot weather, soil disruption), and the numbers and ages of affected animals.

If the carcass is suspicious for anthrax based on history and external lesions, a necropsy is discouraged since the animal will have large numbers of bacilli in the blood (about 10⁹ bacilli/ml for cattle). A postmortem will expose these bacilli to oxygen, causing sporulation, and escalating environmental contamination. In an unopened carcass, *B anthracis* will die out in approximately two days.

To decrease exposure of internal tissues in suspected anthrax cases, the CFIA recommends the following samples in order of preference, be taken as soon as possible after the death of the animal. Early collection will ease detection due to the presence of more viable *B anthracis* and fewer after-death bacterial invaders.

1. Whole blood taken by syringe and needle from the jugular
2. Blood-soaked swab taken carefully through a small incision into the jugular. Cover the opening to capture any leakage
3. Swabs taken from blood-tinged fluids exuding from anus, vulva, nostrils or mouth
4. Sample exudate-contaminated soil. Examine the ground near the nostrils/mouth and anus/vulva for exudate-stained soil.
5. As a last resort, submit a swab soaked with fluid from the spleen if predators have eaten the animal or if a necropsy was performed.

In all cases the sample should be placed in a sterile tube and left “dry”, (ie, do not immerse it in a transport medium since this will inhibit sporulation). The submission of solid tissues from organs is discouraged, unless no other sample is available. Do not submit ears, tongue or hide.

The attending veterinarian should first evaluate smears of the samples, when possible, and the District veterinarian contacted with suspicions. A diagnostic laboratory may also be useful to evaluate smears depending on location and time of postmortem. The carcass should be tarped and limed to prevent scavenging while waiting for the results.

The suggested laboratory stain to identify *B anthracis* morphology is Loeffler’s polychrome methylene blue stain (McFadyen’s reaction). Polychrome methylene blue is a complex mixture of methylene blue and other homologs, primarily azure A and azure B, that are produced by oxidation or ripening. Natural ripening takes one year, but can be hastened by adding 1% K₂CO₃ to Loeffler’s alkaline methylene blue. Anthrax capsules will be tinted pink with this stain. The protocol for using this stain is listed in Table 3.

In veterinary clinics, Loeffler’s polychrome methylene blue is not usually practical. Fortunately, Wrights and Giemsa stains also contain polychrome methylene blue. The combination of Wrights–Giemsa used in commercial quick stains for hematological evaluation can be used. Although the literature suggests results may be more variable with these stains, they work better than Loeffler’s in some circumstances. With the Wrights and Giemsa stain, the capsule will stain mauve. Gram stain will not detect the anthrax capsule.

Anthrax bacilli, on stained smears from fresh specimens, can be seen singly or in chains of 2 to 3 bacilli with square ends, when opposed, and a faint pink to mauve capsule. In living tissue, there will be no spores. Spores will form on exposure to air in about 2 hours. Spores are ovoid and central to paracentral, causing no swelling of the bacilli. Unfortunately, the capsule is

Table 3: CFIA recommended staining protocol using Loeffler’s polychrome methylene blue stain.

1. Make a thin smear of blood or tissue fluid.
2. Discard spreaders (slide or coverslip) into bleach solution.
3. Air dry and fix in absolute methanol or ethanol for 60 seconds and allow it to dry.
4. Place a drop of stain onto the smear and spread with a loop to cover the entire smear.
5. Allow it to stand for 60 seconds, and then wash it with water into bleach solution.
6. Blot dry and discard blotter into bleach solution.
7. Examine under oil immersion (100X)

Table 4: Additional methods for anthrax diagnosis

- Animal inoculation of guinea pigs or mice
- Fluorescent antibody test of smears to detect capsular antigen
- Immunochromatographic assay for protective antigen (PA) excreted by *B anthracis*
- Ascoli thermostable antigen precipitin test can be used to detect residual capsular antigens in tissue when the bacilli can no longer be detected microscopically or on a culture. The test will react with other bacilli species, but since these only rarely cause death, this is not considered important. Care must be taken, however, not to contaminate specimens with soil and sand that often contain saprophytes of the *Bacillus* genus.
- Polymerase chain reaction to detect the genes coding for the virulence factors of *B anthracis*
- Serology (ELISA) for antibodies to PA; in human cutaneous anthrax it will detect about 68% to 93% of cases.

not always easily detectable after staining. This could be due to putrefaction, problems with the stain, or for unknown reasons.

As the animal decomposes, the anthrax bacilli become fewer and harder to find on smears among the more numerous postmortem bacterial invaders. The latter are often larger clostridial bacilli and they are not square-ended. Smears are unreliable after about 24 hours.

Although postmortems will cause more spore formation, they may occur when a veterinarian is establishing the first index case. If it is possible in these circumstances, the unopened carcass should first be moved to the site where it will be disposed of, on an impermeable material such as a plastic tarp. Orifices should be plugged with a disinfectant soaked material to prevent leakage during movement. This practice should also be considered for all postmortems, done on location, in order to decrease environmental contamination by other pathogens, including clostridial infections such as blackleg. In addition to samples being taken to assess for anthrax, additional selections should be considered that could rule in or out other diagnostic differentials. If samples are shipped, it is very important to package them according to the Transportation of Dangerous Goods Act.

The hallmarks of anthrax on postmortem include failure of rigor mortis, unclotted blood, a very enlarged, soft, dark spleen (usually 2–4 times normal size with a black cherry jam-like parenchyma), and paintbrush hemorrhages subcutaneously and on visceral surfaces. Other features include serosanguinous fluid within body cavities, severe enteritis, and edema under the tongue, sternum, perineum, and flanks. In certain cases, possibly due to massive ingestion of anthrax spores or to different strains, the carcass will not have bloody discharges, it may

have rigor mortis, blood will clot and the spleen will not be noticeably enlarged. In these cases, there are still paintbrush hemorrhages and usually some degree of subcutaneous edematous swelling. Other methods for diagnosis are listed in Table 4.

Control, treatment and prevention

The most important control measures for anthrax are proper disposal of the carcass and sanitation. In Canada, when anthrax is reported, CFIA places the operation under quarantine and carcasses are disposed of properly, usually by deep burial with quick lime (CaO, a powerful desiccant). A proper disposal site is important to prevent further environmental spread by ground water or other factors. Mounding earth over carcasses is not recommended given the problems that occurred in Wood Buffalo National Park. Here the mounds became the preferred home sites for foxes and ant colonies whose digging brought up spores. Incineration of carcasses can be performed, either within pits on site or at laboratories. The minor aerosolization of spores by incineration is not reported to be significant.

The soil surrounding and underneath infected carcasses may have large numbers of spores from the bloody exudates, even if the carcass is not opened. This potentially contaminated soil is scraped off and buried in the pit. Surface layers may also be limed and flamed with a blowtorch to kill spores.

Livestock within the affected area are vaccinated with the Sterne vaccine. They are usually moved to another site and watched carefully for disease. Deaths can occur for 8 days before the vaccine builds up sufficient antibodies. Quarantine is lifted 30 days after the last case of anthrax occurs on the farm. Owners are paid an indemnity for animals lost due to anthrax (eg, up to \$500 per cow), but not for the costs of disposal and sanitation.

Since the vaccine is live, interim antibiotics are avoided because the effectiveness of the vaccine will decrease. They may be used, however, if the risk of more animals dying in the first week after vaccination requires it. If antibiotics are used, the vaccine will need to be repeated within about two weeks. Repeat vaccination may still be warranted at 10 to 14 days, even without antibiotics to ensure high protective antibody levels. Yearly vaccines should be done after that. The vaccine is relatively inexpensive, at less than \$2 per head. On a few occasions, a poorly prepared vaccine has reverted to virulence and caused death. Vaccination of goats and llamas with the Sterne strain is not recommended since it can cause death. After vaccination, there is a withdrawal time for milk and meat, since live bacilli may pass into the tissues and milk.

Antibiotics can be used to prevent infection and can save animals, if given in the early stages of anthrax infection. Antibiotics also dramatically decrease the bacterial load, and even if the animal dies, greatly reduce environmental contamination. Antibiotics that are usually effective include penicillin,

tetracycline (doxycycline is preferred in humans), chloramphenicol, erythromycin, clindamycin, extended spectrum penicillins, macrolides, aminoglycosides, vancomycin hydrochloride, ceftazidime and other first generation cephalosporins.

Public health authorities are now recommending ciprofloxacin for the initial treatment in post-exposure to a bio-weapon-source anthrax until sensitivity results are completed. Ciprofloxacin was chosen because there is no known resistance in bio-weapon strains. It is believed that other fluoroquinolones will also work, but testing is not yet complete. Antibiotics against anthrax are very successful if given within 24 hours of aerosol exposure. Anthrax is resistant to sulfamethoxazole, trimethoprim, cefuroxime, cefotaxime sodium, aztreonam, ceftazidime and other extended spectrum cephalosporins.

The human vaccine used in North America stimulates antibodies against protective antigen (PA). The vaccine is a filtrate of an attenuated, unencapsulated, nonproteolytic anthrax strain and contains mostly protective antigen. The vaccine may also have small amounts of lethal factor and edema factor. The vaccine is given subcutaneously at 0, 2, and 4 wks, with a booster administered at 6, 12 and 18 months, and then given once annually. There have been allegations that the human vaccine may cause severe health problems in certain individuals, but investigations have not yet substantiated this claim. Research is ongoing to find a more easily produced and standardized vaccine.

Before the recent anthrax cases in the USA, there was only one company, located in Michigan, making the vaccine. The lack of a human vaccine has raised the concern that anxious individuals may try to use the live-animal vaccine instead. Russia uses a live anthrax human vaccine that is similar to the livestock one. The livestock vaccine, however, is specifically made and tested on livestock and could cause marked problems in people. The USA federal government has warned veterinarians to make sure that the livestock vaccine is only used in animals.

Vaccination is not usually recommended for private citizens, including veterinarians. Vaccination is recommended for industrial workers exposed to potentially contaminated animal products (wool, goat hair, hides and bones), laboratory workers, and the military.

Disinfection

When handling a suspect anthrax animal, use boots, coveralls, and disposable gloves. Aerosolization is not usually a problem but, to be cautious, the use of a high efficiency particulate air (HEPA) filter mask is suggested; ordinary surgical masks are not adequate to stop the spores. If someone is exposed to anthrax, use copious amounts of water with chlorine (5000 ppm, 1 in 10 dilution of household 5.25% bleach), for 1 minute, to wash the affected skin site. Do not use bleach if a cut is present. Follow-up antibiotics may still be required.

Cleaning equipment, facilities, and clothes is relatively simple if anthrax is present in the vegetative form. Once spore formation has occurred, approximately two hours after exposure to oxygen, disinfection becomes very difficult.

Some recommended methods are:

- 1% lysol for 2 days
- Preliminary soak with 30% formalin or 45% glutaraldehyde at 1 to 1.5L/m² for two hours followed by a final disinfection using one of the previous chemicals, or 3% hydrogen peroxide, or 1% peracetic acid for two hours at 0.4L/m². If hydrogen peroxide or peracetic acid is used, the material will need to be treated once more at least one hour later.
- Strong solutions of sodium hypochlorite or sodium hydroxide (5%-10%) can also be used.

Shelagh Copeland, DVM, MVSc, is a veterinarian with the Food Safety and Animal Health Unit of Saskatchewan Agriculture and Food. Since joining the unit in December of 2000, she has been extensively involved in Saskatchewan's Cervid Health Surveillance Program for CWD. Dr. Copeland graduated from the Western College of Veterinary Medicine in Saskatoon in 1979. She spent the next 9 years in a mixed large animal veterinary practice at Kindersley, Saskatchewan, before obtaining a degree in pathology in 1991. Dr. Copeland has worked in veterinary diagnostic laboratories for approximately 9 years in Ontario and Saskatchewan.

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