HEALTH RISKS TO WILDLIFE PERSONNEL: HAZARDS FROM DISEASE-CAUSING AGENTS

Presented by:

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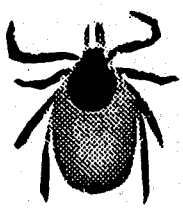

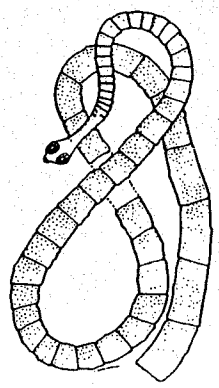

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RABIES

Name of the Disease: Rabies

Rabies is an infectious disease which is due to inflammation of the brain (encephalitis) caused by a virus, usually transmitted from an infected animal by bite inoculation or by contamination of a skin wound by infected saliva. Rabies is also rarely transmitted by contact exposure of the oral, ocular or nasal mucous membranes.

Causative Agent: Rabies viruses belong to the genus ***Lyssavirus***, in the family Rhabdoviridae. They are bullet-shaped viruses, averaging about 180nm long and 75nm in diameter, with a coiled single-stranded RNA nucleocapsid contained in a phospholipid envelope. Studded over the surface of the envelope are spikes of G protein, which is the antigen against which the immune response is directed. Rabies is *Lyssavirus* Serotype 1, and is responsible for classical rabies in terrestrial mammals and New World insectivorous and haematophagous bats; Serotypes 2, 3 and 4 have been isolated from old world bats, shrews, and from occasional cases of rabies-like disease in pets and people in Europe and Africa.

Distribution: Rabies virus infects all types of mammals, and may infect birds, though the latter are not significant in the epidemiology of the disease. Rabies occurs on all continents except Australia and Oceania, though some areas elsewhere, especially islands such as Great Britain and Ireland, are completely free of rabies. In some regions, rabies may not occur in terrestrial animals, though bats are infected. Although rabies may occur in any species of mammal, the epidemiology of the disease in a particular area tends to be associated with a single species of mammal, which acts as a reservoir of infection, from which rabies "spills over" into other species. For instance, arctic foxes (*Alopex lagopus*) are central to the epidemiology in arctic regions, while raccoons (*Procyon lotor*) are important in eastern United States, vampire bats (especially *Desmodus rotundus*) in Central and South America, and dogs (*Canis familiaris*) in Africa and parts of Asia.

Ecology/Epidemiology: The epidemiologic association of rabies with particular reservoir species in various geographic regions is related to genetic variation in the virus, which is presumably the result of evolutionary adaptation of particular rabies virus strains or variants to specific host species. This adaptation is likely favourable to maintenance of viral infection in a mammal population, and may explain the greater "efficiency" of rabies virus infections in hosts to which they are adapted. For instance, the arctic fox strain of rabies seems to require a lower dose of virus to infect foxes than some other species, and has a shorter incubation period and may result in a higher concentration of virus in saliva in foxes, in comparison with other species. The adaptation of rabies variants to particular hosts may explain the variations in susceptibility to infection evident in some mammal species. Rabies variants are identified by molecular analysis of genetic homology, or by comparison of minor variations in the G antigen, detectable by monoclonal antibodies.

In North America, 5 major variants of rabies virus are established in terrestrial mammals.

An arctic fox variant infects arctic and red foxes (*Vulpes vulpes*) in arctic and subarctic regions. Following an incursion from the arctic in the mid-1950's, this variant established an endemic focus in red foxes in southern Ontario, from which it periodically spills over into adjacent areas of Quebec, New England and New York. Periodically, this variant has also reached Newfoundland from the arctic via Labrador, across sea ice in the winter.

A raccoon variant emerged in Florida in the late 1940's. It has spread north into contiguous areas of Georgia, Alabama and South Carolina. In the late 1970's an outbreak of this variant occurred in Virginia, probably as the result of coon hunters who translocated raccoons incubating rabies from an endemic area. Raccoon rabies has now spread from this focus over a broad front, to encompass large parts of the states of Virginia, West Virginia, Pennsylvania, Delaware, Maryland, New York, Connecticut, and Massachusetts. The front has moved north and west through New York at about 50km/yr since 1990, and is now on the Niagara frontier with Ontario at Buffalo.

Two variants of rabies virus infect skunks, one in the south central United States west of the Mississippi, and one in the northern midwest and Great Plains region. The latter variant has expanded into Manitoba and Saskatchewan over the past several decades, and is controlled within the southern and eastern borders of Alberta by an active surveillance programme. The northern skunk variant also occurs in California, perhaps due to animal translocation, and rabies in gray foxes (*Urocyon cinereoargentus*) in Texas is also caused by a virus in this variant. Dog and coyote (*Canis latrans*) rabies in Texas, which has invaded from Mexico in recent years, is also caused by a virus within the northern skunk genetic variant type.

The 5th variant is found only in gray foxes in the southeast part of Arizona.

In addition to the variants of rabies recognized in terrestrial animals, at least 6 distinct variants are associated with particular species of insectivorous bats distributed widely in North America. Though occasional individual cases of rabies caused by bat variants are recognized in other species, bat rabies variants do not establish endemic infections in populations of terrestrial animals. For instance, though bat rabies occurs in British Columbia, and a bat variant has been associated with rabies in a horse in B.C., endemic terrestrial rabies does not occur in that province. In North America, rabies may be maintained endemically especially in the big brown bat (*Eptesicus fuscus*), the hoary bat (*Lasiurus cinereus*), the red bat (*L. borealis*), the Mexican free-tailed bat (*Choeronycteris mexicana*) and perhaps the silver-haired bat (*Lasionycteris noctivagans*) and the Florida yellow bat (*L. intermedius*). Rabies in other species of bats, especially *Myotis spp.* may result from "spillover" from these endemically infected species.

The big brown bat is the species most commonly diagnosed rabid in western Canada and in Ontario. The silver-haired and hoary bats are the second and third most-frequently diagnosed as rabid, and the prevalence of positives is highest in hoary bats. In all areas, other species of bats, including the common little brown bat (*Myotis lucifugus*), are less frequently diagnosed as rabid, and rabid bats are relatively rarely reported from the Maritime provinces. Of the last 5 cases of human rabies diagnosed in Canada, 3 are known to be bat-related (Saskatchewan, 1970; Nova Scotia, 1978; Alberta, 1985).

Signs and Symptoms: Rabies virus inoculated into the tissues eventually enters peripheral nerves, and is transported along them (centripetally) to the spinal cord and brain. In the brain, the virus replicates in, and spreads among, nerve cells (neurons), infecting many parts of the brain and causing encephalitis. Abnormal behaviour results from the effects of viral infection in neurons. Virus then moves away from the brain (centrifugally) in the nerve processes (axons) that radiate from neurons. It is disseminated via the nervous system (not in the bloodstream) to many peripheral parts of the body. Many organs become secondarily infected with rabies virus, but it is in the salivary glands that high concentrations of virus replicate, so that the saliva of an infected animal may contain huge amounts of virus, capable of infecting any other animal bitten. Virus generally is present in saliva by the time that an animal shows signs of rabies.

Rabies should be suspected in any wild animal showing any behaviour considered abnormal, including: loss of fear, or unusual friendliness; excitation or aggression; daylight wandering in nocturnal species; depression; incoordination, ataxia or paralysis; twitching, convulsions or seizures; abnormal vocalization; inability or reluctance to swallow, or signs of choking; drooling of saliva or frothing at the mouth; and, in carnivores, evidence of having attacked porcupines.

In people, signs may include a prodromal phase of anxiety, perhaps with abnormal sensation in the vicinity of sites of exposure. Subsequently, non-specific signs such as depression, headache, vertigo, etc. may occur in the early clinical period, followed by excitability, stiff neck, hyperreflexia, hydrophobia (inability to drink), spasms, paralysis, and ultimate death due to the effects of cerebral edema (swelling of the brain) or complicating aspiration pneumonia. Clinical rabies is almost always fatal.

Diagnosis: In animals, rabies is diagnosed by demonstration of virus in infected cells in the nervous system using a fluorescent antibody technique (FAT). The brain is removed from the animal and a small sample placed on a slide to be examined in this way. Hence, it is important that the brains from animals suspected of being rabid not be destroyed, by gunshot or other major trauma.

In cases in which human exposure has occurred, if the FAT on the animal's brain is negative, suspect brain is inoculated into mouse neuroblastoma cells in tissue culture, and they are examined by FAT for the presence of rabies virus over the subsequent week. This test is more sensitive, and detects FAT "false negative" reactions. All rabies diagnosis in Canada is carried out by the Health of Animals laboratories of Agriculture and Agrifood Canada. Brains of infected animals may be examined histologically as well, but the microscopic lesions of encephalitis are not specific for rabies, and Negri bodies, which may occur in rabies-infected neurons, are often not present in rabid animals.

Prevention and Control: Prevention of rabies hinges on 4 basic principles. (1) Vaccinate domestic animals which may be exposed to wildlife reservoirs of rabies and subsequently come into close contact with people. (2) Avoid contact with potentially infected animals. (3) If there is a high risk of occupational exposure, consider prophylactic rabies vaccination. (4) If possible exposure occurs, immediately wash the wound or affected area with soap and water, disinfect with a quaternary ammonium compound or 40-70% alcohol, and apply other treatment

appropriate to the type of wound. Present to a physician with a history of possible rabies exposure. If possible, kill the animal involved and submit it immediately for rabies diagnosis. Based on an evaluation of the risk, medical treatment including infusion of the wound with rabies antiglobulin, and post-exposure vaccination, may be carried out.

Rabies control in wildlife populations is based on vaccination of a sufficient proportion of the population to halt epidemic transmission, and to suppress the development of endemic foci. This is accomplished by trap/vaccinate/release programmes using conventional injectable vaccines in small populations at high risk (eg. raccoons in the Niagara frontier of Ontario); use of modified live rabies virus vaccine baits placed by hand or by aerial application over wide areas (eg. fox rabies in western Europe, Ontario); or by use of genetically engineered recombinant vaccines, in which the gene for the G protein of rabies virus is inserted into the genetic material of another (vector) virus, which is disseminated in oral baits (eg. rabies-vaccinia recombinant vaccine for raccoons in NE USA and Canada).

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HANTAVIRUSES

Name of the disease: 1) Hantavirus pulmonary syndrome (HPS)
2) Hemorrhagic fever with renal syndrome (HFRS)

Causative agent(s): 1) Most HPS cases have been caused by Sin nombre (Muerto Canyon) virus but cases of HPS have also been contracted by hantaviruses tentatively named Black Creek Canal, Bayou and Shelter Island-1 viruses.
2) Hantaviruses causing HFRS include Hantaan, Puumala, Dobrava and Seoul viruses.

Other hantavirus strains have been identified but are not currently known to be etiological agents of human disease.

Distribution:

Geographic: As of January 11, 1995 a total of 102 cases of HPS have been recognized in 21 American states and seven cases have been seen in Canada including three in British Columbia and four in Alberta. An additional possible case has been reported from Brazil, South America.

Cases of HFRS due to Hantaan virus have been seen in Asia and the Balkans. Seoul virus has been found worldwide, Puumala virus in Scandinavia, northern and eastern Europe and the Balkans and Dobrava virus in the Balkans.

Host Species: The major HPS host species include for Sin nombre virus the deer mouse *Peromyscus maniculatus*, for Black Creek Canal virus the cotton rat *Sigmodon hispidus* and for Shelter Island-1 virus the white-footed mouse *Peromyscus leucopus.* The host species of Bayou virus is currently unknown.

The major HFRS host species include for Hantaan virus the striped field mouse *Apodemus agrarius,* for Puumala virus the bank vole *Clehtrionomys glariolus*, for Dobrava virus the yellow-necked field mouse *Apodemus flavicollis* and for Seoul virus the brown rat.

Epidemiology of the disease: Infected rodents are believed to shed virus in saliva, urine, and feces. Human infection is mainly believed to occur by inhalation of infective saliva or excreta produced directly from the animal, or when dried materials contaminated by rodent excreta are disturbed. Other possible sources of infection include direct introduction into broken skin or onto the conjunctivae or, possibly, ingestion in contaminated food or water. Persons have also become infected after being bitten by rodents. Cases have been epidemiologically associated with planting or harvesting field crops, occupying previously vacant cabins or other dwellings, cleaning barns and other outbuildings, disturbing rodent-infested areas while hiking or camping, inhabiting dwellings with indoor rodent populations, and residing or visiting areas in which the rodent population has shown an increase in density.

Signs and symptoms of the disease:

Animals: No disease reported.

Humans: 1) HPS begins with a **prodromal phase** that generally lasts for 3-6 days. Fever and myalgia are almost universal features during the prodrome with gastrointestinal symptoms, headache and dizziness also observed in some cases. The prodrome is followed by a **cardiopulmonary phase** generally heralded by progressive cough, shortness of breath, rapid heart rate and fluid buildup in lung. Fatalities often occur at this stage. The **convalescent phase** of HPS is marked by improved oxygenation and hemodynamic function. Progression to this phase can be remarkably rapid. Although data on the long-term sequelae of HPS are lacking, recovery has apparently been complete among patients who survive the cardiopulmonary phase.

2) HFRS caused by Hantaan virus is abrupt in onset and is classically described as progressing through five distinct clinical phases. A **febrile phase** is manifested by headache, abdominal and lumbar pain, facial flush, and widespread petechiae. After 3-5 days, the febrile phase is followed by a **hypotensive phase** during which shock occurs. The next clinical event is the **oliguric phase** during which hypertension may develop. Hemorrhagic complications and disseminated intravascular coagulation may occur during these first three phases. As renal failure and hemorrhagic manifestations resolve, there is a **diuretic phase** during which electrolyte imbalances may occur. The final phase, a **convalescent phase**, may persist for weeks. Infection with Seoul virus results in a mild form of HFRS. Puumala virus is associated with a usually-benign form of HFRS, often referred to by its original name, nephropathia epidemica. Severe HFRS associated with Dobrava virus infection in the Balkans has been described.

Methods of diagnosis: Diagnosis is made by serological tests that detect hantavirus IgM antibodies in serum or a fourfold or greater rise in serum IgG antibodies; by detection of hantavirus antigen in tissue using immunohistochemistry; and/or by amplification of hantavirus nucleotide sequences by the polymerase chain reaction test.

Prevention and control: The primary target for prevention is avoidance of rodent contact by taking specific steps to reduce rodent infestation in areas where people live and work. Rodent control around households is undertaken by use of traps, rodenticides and by modification of rodent habitats. Food and garbage should be placed in rodent-proof containers, openings that allow rodent entry into homes covered and all potential rodent nesting sites, e.g. woodpiles, dense shrubbery, removed from close proximity to human dwellings.

Treatment: Treatment of patients showing early symptoms of HPS i.e. respiratory difficulties is of paramount importance and may save lives. An antiviral drug, ribavirin, has reduced mortality in patients with HFRS but has not shown any clear effectiveness to date when treating patients with HPS.

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CONTAGIOUS ECTHYMA

Name of the disease: Contagious Ecthyma or Orf. Orf was originally the name used for human infections but is now frequently used for animal infections as well. Other names given to this disease are contagious pustular dermatitis, soremouth or scabby mouth.

Causative agent: Contagious ecthyma (CE) is caused by a virus which is a member of the poxvirus family.

Distribution: Infection of domestic sheep and goats by CE virus is common worldwide. A similar wide distribution is seen in wild species of sheep and goats. The disease is reported infrequently in reindeer, caribou, muskox, moose, dog, cat and humans.

Ecology/Epidemiology: Contagious ecthyma is most common in lambs and kids. Infections can be numerous in some flocks; however, deaths are usually infrequent but have been reported in bighorn sheep, mountain goats and muskoxen. Contact with contaminated objects such as rubbed trees and rocks are thought to be the most common means of transmission. Virus in shed scabs is extremely resistant and can survive in the environment for more than 15 years. Damage to skin is required for transmission of disease and, at least in domestic animals, prevalence is higher during periods of drought when animals are eating coarse feed. Chronically infected, reinfected or, possibly latently infected animals may allow virus to persist in a flock for several years. Human infections are most common in abattoir workers handling wool and skins.

Signs and symptoms of the disease: Contagious ecthyma is characterized by tan, grey proliferative scabby areas on the muzzle and lips. Occasionally lesions are seen around the eyes, on distal aspects of the limbs and on the udder. In severe cases, lesions are present in the oral cavity, esophagus and rarely extend into the rumen. Lesion development is typical of pox virus infections but more proliferative. The infection begins as a small (1-3 mm) red area which enlarges, becomes raised, blisters and then fills with pus. Within 7 to 19 days scabs begin to form and, if the area does not become secondarily infected by bacteria, the lesions spontaneously regress in about 4 weeks. Animals lose condition because they are reluctant to eat or nurse. Transmission between females and their young are common. Females with infected udders are reluctant to nurse resulting in loss of condition and sometimes death of young. Bighorn sheep infected with CE are extremely itchy and can be observed rubbing affected areas on trees, rocks and nearby sheep. Death is usually a result of starvation or secondary infection of sores by bacteria and sometimes fly larvae.

Human infections typically occur on the hands at the site of a skin abrasion and are similar to those described in animals. The area is often pruritic.

Methods of diagnosis: Diagnosis is based on gross appearance of the lesion, and characteristic histological features. Infection can also be detected by identifying antibodies to CE virus in the serum.

Prevention, treatment and control: Human infection is prevented by wearing gloves and coveralls when handling infected animals. The virus is killed by phenol disinfectants and steam. Animals may be vaccinated by inoculating a scarified region of the skin on the flank with live virus. The lesion that develops is of little consequence to the host and immunity lasts for approximately 2 years. Immunization is impractical in the wild.

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ANTHRAX

Name of the disease: Anthrax

Causative agent(s): The bacterium *Bacillus anthracis*

Distribution: This bacterium occurs in many countries but, within the general distribution, there are local areas where the disease occurs more frequently. An example in Canada is the Wood Buffalo National Park area where the disease occurs sporadically in bison. Anthrax is usually a disease of ungulates (cattle, bison, sheep, goats, deer) as well as horses but may also occur in more resistant species such as pigs and dogs.

Ecology/epidemiology of the disease: *Bacillus anthracis* occurs in two forms: a vegetative or growing state and a highly resistant spore form. Animals become infected by ingesting spores from soil or vegetation. The bacteria grows in the vegetative state within the animal's body and if the carcass is opened after the animal dies the bacteria sporulate when exposed to oxygen and contaminate the soil, vegetation and water in the area. Spores may persist for many years in the soil. Outbreaks often occur when heavy rains wash spores from the soil and concentrate spores in low areas that are subsequently grazed by animals. Animals may also be infected by eating contaminated tissues or bones. It appears that under suitable circumstances the bacterium may grow vegetatively and multiply in the soil. This has been suggested to occur in areas with alkaline soil with abundant decaying organic matter, alternating periods of wet and dry soil, and warm soil temperatures (>15.5 C). (It is not known if this occurs in Canada). Humans become infected through contact with infected animal carcasses, contaminated animal products, or spores from the vicinity of infected carcasses. Biting flies may transmit the infection locally between infected animals and uninfected animals or people.

Signs and symptoms of the disease: In highly susceptible animals including cattle, bison, sheep, goats and deer, the disease is usually very acute with fever, depression, difficulty in breathing, convulsions and death. Most animals are found dead without any signs having been observed. There is often a bloody discharge from the orifices.

More chronic disease occurs in resistant species, such as pigs and dogs, in which there is often local infection of the pharyngeal area leading to severe edema of the throat that may cause asphyxiation.

The most common form in humans is cutaneous infection in which the skin is infected through contact with contaminated material during handling, skinning or butchering infected animals. Within 2 to 5 days, the skin in the area of contact , e.g., on the forearm above gloves, becomes itchy, then a small, raised, red area appears that may become a vesicle (blister) in the skin, this area then becomes dark and sunken. The skin lesion may not be very painful but if not treated the infection may become generalized and fatal.

Humans may also become infected by inhaling or ingesting spores, resulting in severe pneumonia or gastroenteritis with a high fatality rate.

Methods of diagnosis: A preliminary diagnosis of anthrax can be made by examination of blood smears from animals that died of the disease or of fluid from skin vesicles in humans. This should be confirmed by isolation of the bacterium.

Animals that are suspected to have died of anthrax should not be necropsied. To make a diagnosis, a small volume of blood can be withdrawn from a vessel in the unopened carcass using a syringe and the blood sample submitted to a laboratory. Animals that die of the acute disease have bloody exudate at the orifices, the blood is unclotted, there may be hemorrhages in organs and edema fluid under the skin, particularly of the neck region, and the spleen is usually greatly enlarged, red-black and semi-fluid.

Prevention, treatment and control: Animals that are suspected to have died of anthrax should not be necropsied. If the carcass is left unopened it will decompose destroying the vegetative stage of *B. anthracis*. However, if the carcass is opened and tissues are exposed to the air, spores will form and contaminate the area for many years. The unopened carcass should be destroyed at the site by burning completely or by burying deeply and covering thoroughly with quicklime (calcium oxide). If a carcass is inadvertently opened and lesions suggestive of anthrax are found (see above), a blood sample should be taken for confirmation and the carcass destroyed as rapidly as possible. Care must be taken to prevent human infection during handling carcasses through use of protective clothing and respirators. People working with known cases of anthrax can be vaccinated. Contaminated clothing should be destroyed by burning as the spores are very difficult to kill with disinfectants. Any illness following potential exposure should be reported immediately to a physician. Infections respond well to antibiotics if therapy is administered early in the diseases.

Key references: (two examples of recent Canadian outbreaks are listed).

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TUBERCULOSIS

Name of the disease: Tuberculosis

Causative agent(s): The bacterium *Mycobacterium bovis*

Distribution: *Mycobacterium bovis* was at one time widespread in cattle in Canada but the infection has been eliminated from cattle. Bison in and near Wood Buffalo National Park are infected with *M. bovis*. An outbreak of tuberculosis was recognized among game ranch elk in Alberta and Saskatchewan in 1990 and the infected herds were depopulated. Tuberculosis was present in ungulates in many zoos and animal collections, testing and control of these facilities is not complete at present. Tuberculosis has been diagnosed very rarely in free-ranging wild animals.

Ecology/epidemiology of the disease: Tuberculosis caused by *M. bovis* is a chronic disease that spreads primarily via inhalation of organisms expelled from the lungs as droplets by infected animals. Infection can also occur by ingestion of material contaminated by secretions or excretions from infected animals. Infection of the female reproductive tract or mammary glands may result in infection of the offspring. The organism is very resistant and will survive for an extended period in the environment. Infection among animals is enhanced by close contact. Human infection can occur through inhalation of infected droplets during handling infected animals or their carcasses, or orally through ingestion of infected material. (Infection via milk from infected cows occurred prior to pasteurization of milk and elimination of the disease from cattle).

Signs and symptoms of the disease: In cattle and bison, the disease most commonly causes chronic pneumonia; infected animals may remain clinically normal for an extended period although they are infectious. In advanced cases there is severe respiratory distress, weight loss and debilitation. In some animals the infection may spread to other organs causing a variety of signs of debilitation, such as chronic arthritis. In elk and other deer, lesions are often confined to lymph nodes of the head and neck, and these are sometimes noticeable externally as swellings, with less common involvement of the lungs. Animals with extensive lesions may appear clinically normal.

Infection of humans with *Mycobacterium bovis* cannot be distinguished clinically or radiologically from infection with *M. tuberculosis* (the human tuberculosis agent).

Methods of diagnosis: Clinical examination can not be used to detect infected animals, as many appear clinically normal. Tuberculin tests of various types are used to detect exposed animals; however, some infected animals may be negative on these tests. Infections in humans must be distinguished from infection with *M. tuberculosis* by isolating and typing the bacterium.

Prevention, treatment and control: The risk to the general public has been eliminated by eradication of the disease in cattle. Infection of humans with *M. bovis* is now a risk only for those in close contact with infected animals during handling, necropsy or butchering. At present, this only applies to bison in the Wood Buffalo National Park area. Use of protective clothing and respirators and sound hygiene is required.

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BRUCELLOSIS

Name of the disease: Brucellosis, sometimes called "undulant fever" in humans.

Causative agent(s): The bacteria *Brucella abortus* and *Brucella suis* biovar 4.

Distribution: There are two forms of the disease associated with wild animals in Canada: *Brucella abortus* was associated with cattle but is now only known to occur in association with bison in the Wood Buffalo National Park area. Wolves, a fox and a moose have also been found to be infected in that area. This infection also occurs in bison and elk in the Greater Yellowstone area of the USA.

Brucella suis biovar 4 is associated with barren-ground caribou and with reindeer and may spill over into carnivores (wolves, dogs, bears).

Ecology/epidemiology of the disease: Bison and elk are the only wild species in which a self-sustaining infection of a population with *Brucella abortus* has been documented. Many other animals are susceptible to experimental infection and occasional individuals have been found infected in association with infected cattle, bison or elk. For example, wolves have been found infected in Wood Buffalo National Park and coyotes have been found infected in association with infected cattle in Texas. It is thought that carnivores became infected by consuming infected animals, placentas or aborted fetuses and there is no evidence that the disease will become established and spread within these species. Moose seem to be unusually susceptible to infection and develop severe and likely fatal disease when infected. Transmission within bison and elk occurs through close contact with infected animals, and particularly with aborted fetuses or vaginal discharges. Venereal transmission may occur. Mechanical transmission by biting flies from infected to uninfected animals is possible.

The ecology of *B. suis* biovar 4 is very similar, except that the primary hosts are reindeer and caribou. Transmission occurs by the same methods as for *B. abortus*. Naturally infected muskoxen and a moose have also been identified.

Humans become infected through exposure to infected animals or products from these animals during butchering, or through handling fetuses, afterbirth or contact with secretions and excretions from the genital system of infected animals. Infection can also occur through ingestion of raw meat from infected animals.

Signs and symptoms of the disease: *Brucella* infections in animals affect primarily the reproductive tract of both males and females and, less commonly, cause arthritis, abscessation of lymph nodes, inflammation of the mammary tissues and generalized disease. Common lesions include abortion during late pregnancy ; inflammation of the uterus; destructive inflammation of the testicles in males (orchitis); fluid-filled swellings on the forelegs (carpal bursitis); abscessed lymph nodes; and arthritis.

In humans, *Brucella* spp. may cause disease as early as 1 week or as late as several months after exposure. The clinical disease is a septicemia accompanied by fever that may be continuous or intermittent, (the intermittent fever has resulted in the name "undulant fever"), together with chills, profuse sweating, pronounced weakness and fatigue, joint pain and marked neurological effects including irritation, nervousness and depression. *Brucella* localize in lymph nodes and spleen (causing swelling of these organs) as well as in liver and bone marrow. If untreated the disease may last from weeks to months, and may cause a variety of serious effects because of infection of brain, joints, bones or heart. *Brucella suis* is considered to be more pathogenic and invasive than *B. abortus* for humans.

Methods of diagnosis: Exposure to *Brucella* spp. can be detected by serologic tests. Isolation of the bacterium from tissues is required to confirm infection in animals.

Prevention, treatment and control:

Infection of humans occurs when bacteria enter through skin abrasions, mucus membranes or conjunctiva of the eyes while individuals are handling tissues from infected animals. The most highly contaminated tissues are the reproductive organs, affected joints and bursae, and lymph nodes. Tissues from animals suspected to be infected should always be handled with protective clothing ; meat from such animals should be cooked thoroughly.

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TULAREMIA

Name of the Disease: Tularemia

Causative Agent: The bacterium *Francisella tularensis*.
(There are two subtypes or subspecies: *F.t. tularensis*, also called Type A and *F. t. palaearctica*, also called Type B. Either one can cause the disease called tularemia).

Distribution: Tularemia occurs **throughout the northern hemisphere** with the exceptions of Great Britain and the Iberian Peninsula. Of the two subspecies that can cause the disease, only *F.t. palaearctica* (Type B) has this wide distribution; *F. t. tularensis* (Type A) occurs only in North America.

Ecology and Epidemiology: **In North America**, the preferred habitats of the two subspecies of the bacterium appear to differ. *F. tularensis tularensis* (Type A), typically occurs in terrestrial habitats where it infects rodents and lagomorphs and is transmitted particularly by ticks, but also by mites and biting insects such as mosquitos, fleas, lice and biting flies (Tabanids). *F.t. palaearctica* (Type B) occurs most often in aquatic habitat where it infects aquatic rodents and is transmitted via contamination of the water itself, in which it can remain infectious for weeks to months. Contact with the feces, urine or body parts of infected animals are additional ways by which infection with either subspecies of *F. tularensis* can occur. In both habitats, the bacterium appears to persist by infection of rodent or lagomorph species that do not suffer severe disease but remain infected for long periods and act as sources of infection for other animals and for biting arthropods. Both subspecies of *F. tularensis* can cause disease in a very wide range of species of mammals. The bacterium appears to persist in various local environments through a complex ecological association with particular rodent or lagomorph species and arthropod vectors. Ticks can act not only as vectors of transmission but also as reservoirs of the bacterium which can live in certain tick species for months. The mammal and arthropod associations in which the bacterium persists differ from location to location. In North America, tularemia occurs commonly in cottontail rabbits (*Sylvilagus* sp.), and in beavers and muskrats. Antibodies to the bacterium are rarely found in these species, which suggests that the disease for them is usually fatal. By contrast, antibodies are commonly found in the snowshoe hare, a species that does not appear to suffer much from the disease.

Human infections in North America have been associated overwhelmingly with exposure to cottontail rabbits (*Sylvilagus* sp.) infected with *F.t. tularensis* (Type A). In Canada, this was true prior to 1950. Since then, the major risk factor has been exposure to muskrats, which are usually infected with *F.t. palaearctica (Type B)*. Between 1929 and 1979, 290 cases of tularemia in humans were officially recorded in Canada; it is likely that many more cases occurred but were either not recognized or were not reported.

Signs and Symptoms of the Disease: **In Animals**, tularemia is most often recognized through examination of dead animals. Signs of tularemia in diseased wildlife are not different from those of other acute illnesses. Lesions present in dead animals are similar to those of plague, yersiniosis and several other infections. In all cases, tiny pale spots on the liver, spleen and/or lung, and enlargement of the spleen are typical. A thin layer of pale, easily-removed material (fibrin) may overly the liver and abdominal organs. Inflammation of the intestine is common in aquatic rodents infected by ingestion of the bacterium. **In Humans**, the initial symptoms are those of generalized febrile illness (fever with chills, headache, vomition, etc in various combinations) which begin 1-10 days after infection. The course of the disease depends in part on the route of infection. Infection by arthropod bite is usually followed by development of an ulcer at the bite location and swelling of the lymph node(s) that filters body fluid from the bite wound area. Infection via inhalation of infected material such as fecal particles results in pneumonia, although secondary pneumonia is common after infection by any route. Oral infection leads to inflammation of the pharynx and intestines. The disease caused by *F.t. tularensis* (Type A) is often more serious than that caused by *F. t. palaearctica* (Type B); 40-60% of untreated cases of pneumonia or enteritis and 7% of all forms of untreated infection with this subspecies have been fatal, compared with overall fatality of 1% in untreated infections due to *F.t. palaearctica* (Type B). Human to human transmission of tularemia is very rare, even in cases of pneumonia.

Diagnosis of the Disease: Since the signs and symptoms of the disease in humans are not themselves diagnostic, clinical diagnosis is usually based on the presence of symptoms and a history of exposure to wild animals, biting arthropods or untreated water. Diagnosis is confirmed by identifying the bacterium from lesions or sputum by culture or by immunological techniques. The sudden appearance of antibodies in the patient's blood 2-3 weeks after the onset of illness also confirms the disease. Patients with brucellosis sometimes test positive in tests for tularemia that are based on finding antibodies in the blood. This is due to some similarities between the bacteria that cause these two diseases. Thus, it is important to use appropriate controls for this cross-reaction in tests for tularemia.

Treatment, Prevention, Control: Tularemia is readily treated with antibiotics if treatment is begun early in the course of the disease. Thus, wildlife personnel should not ignore the onset of symptoms of fever and general malaise but should seek immediate medical attention. They should advise the attending medical personnel that they have had contact with wildlife and wild areas, and thus might be at risk with respect to various zoonotic diseases. There is no means to control tularemia in wild animal populations. Prevention of the disease in humans involves common-sense precautions: use of repellents and protective clothing to avoid arthropod bites, inspection for and removal of ticks, use of gloves when handling or dissecting wild animals, particularly rodents and lagomorphs. For persons at regular high risk, vaccination against tularemia may be justified. An attenuated live vaccine exists that has been very effective in reducing human cases of the disease in high-risk areas, such as portions of Russia.

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PLAGUE

Name of the Disease: Plague (also Bubonic Plague, Sylvatic Plague)

Causative Agent: The bacterium *Yersinia pestis*
(more recently named *Yersinia pseudotuberculosis* var. *pestis)*

Distribution: **In North America**: Plague occurs among wild rodents and their fleas over a broad area of the Western United States, including all states west of the longitude of the North Dakota-Montana border and western portions of some states east of that line. Areas of regular occurrence are centred in the southwest in northern New Mexico and in the area of the California-Oregon border. Counties bordering Canada in Washington, Montana and North Dakota have had occurrences. **In Canada**, plague was reported in Richardson's Ground Squirrels (*Spermophilus richardsoni*) in south central Alberta in the 1940's and in Bushy-tailed Woodrats (*Neotoma cinerea*) in British Columbia in the upper Fraser Valley in 1988 and 1989. **World-wide**, plague occurs or has occurred on all continents except Australia, and is present among wild rodents in North and South America, Asia and Africa. *Y. pestis* can infect a wide range of mammalian hosts and many species of flea. Severe disease occurs in some species, including humans, while in others, infection is virtually inapparent.

Ecology and Epidemiology: Plague occurs regularly in two different ecological settings. **Urban Plague** occurs among rats (*Rattus norvegicus*) and their fleas, and is transmitted to humans by bites of the rat fleas. *Y. pestis* is pathogenic for the rats and the fleas as well as for humans in this setting. Thus, urban plague tends to occur as epidemics that may be small and local or very extensive. Three world-wide epidemics have occurred, in AD 524, 1346-1650 and 1884 to the 1930's. The natural home of *Y. pestis* is among wild rodents and their fleas. Plague in this setting is often referred to as **Sylvatic Plague** and urban plague probably is initiated by periodic spread of the bacterium from this natural home to urban rats. *Y. pestis* appears to persist best in arid, open dry forest and grassland habitats. The bacterium persists in rodent species that become infected but suffer little harmful effect, species such as voles (*Microtus* sp) and deer mice (*Peromyscus* sp.) in North America, and in the fleas of these rodents. The bacterium is most often transmitted among mammalian hosts by flea bites. Fleas are very important in the maintenance and transmission of the bacterium. Some species of flea are excellent vectors of the *Y. pestis* while others are very poor vectors. Maintenance of the bacterium in nature involves complex interactions among mammalian hosts, their fleas and *Y. pestis*. From time to time, epidemics of plague emerge from these preferred habitats of the bacterium and spread great distances. Such epidemics may take the form of long-distance spread of the bacterium among rodents that suffer little actual disease or of large-scale mortality among more susceptible species such as prairie dogs (*Cynomys* sp.) and ground squirrels (*Spermophilus* sp.). Humans most

commonly become infected with *Y. pestis* from bites of infected fleas or from unprotected handling of the tissues of animals that died of plague. Human to human transmission of plague does not usually occur unless an infected person develops pneumonia, in which case aerosol transmission of the bacterium among people can and does occur.

Signs and Symptoms of the Disease: **In Animals**, plague is most often either inapparent or fatal. Thus, affected animals either show no signs of disease or are found dead. Recovered animals may have internal abscesses. Susceptible species may suffer very high rates of mortality; for example, mortality rates in prairie dogs have approached 100%. Cat species are susceptible to plague and often die of the infection. Most carnivores appear to be resistant to the disease. The lesions seen in animals that die of plague are similar to those seen in tularemia, other forms of yersiniosis and a variety of other generalized infections. Enlarged spleen, one or more enlarged lymph nodes, tiny pale spots on the surface of the liver, spleen and/or lung all are typical. Abscesses in lymph nodes also may develop. **In Humans**, three different clinical forms of plague are recognized, but each begins with similar symptoms 2-6 days after infection: fever, chills, headaches, nausea. None of these symptoms is unique to plague. The three clinical forms that may follow are bubonic, septicemic and pneumonic. A bubo is an enlarged, painful lymph node (gland-like structures that filter tissue fluid as it is drained back to the blood stream.) In most situations, bubonic plague is the most common form in humans and is characterized by one or more buboes that develop most commonly in the lymph node that drains the point of infection, usually a as flea bite. Since legs and arms are common points of entry for the bacterium, painful, swollen lymph nodes in the arm pit or groin are typical. In untreated cases of bubonic plague, death occurs in 25-60% of cases. In septicemic plague, the bacterium infects and multiplies in the blood stream (septicemia). Death is the usual outcome after 1-3 days. Pneumonic plague develops either from spread of the infection in lymph node or blood to the lung, or from infection by inhalation of bacteria aerosolized from another person with pneumonic plague. The result is severe pneumonia and pneumonic plague is nearly always fatal.

Methods of Diagnosis: Diagnosis may be suspected on the basis of symptoms and the possiblity of exposure or, in animals, from lesions observed at autopsy. Diagnosis is confirmed by culture of the organism or a rise in antibody titre during illness and recovery.

Treatment, Prevention, Control: Fortunately, plague can be treated effectively with antibiotic drugs if treatment is initiated fairly soon after symptoms are first recognized. Prompt diagnosis and initiation of therapy are key. Wildlife personnel should not ignore the onset of any illness and should quickly seek medical attention and inform the physician that they have regular contact with wild animals. There is no effective vaccine for lasting protection against plague. Thus, vaccination is not an option as a preventive strategy except for persons at regular high risk, for whom vaccination with a currently-available vaccine that may provide immunity for up to 6 months may be justified.

Prevention is effected by taking precautions against infection, such as use of protective clothing and gloves when handling wild rodents in areas affected by plague, caution against contact with fleas, etc. Measures to control plague by reducing its prevalence in wild rodents and

the liklihood of transmission to people have been undertaken in the United States in areas such a public parks. Control has been aimed at destruction of fleas that transmit the disease. Treatment of burrow entrances with insecticide and use of rodent bait stations that dust rodents with insecticide have proved effective.

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YERSINIOSIS

Name of the Disease: Yersiniosis

Causative Agents: The bacteria *Yersinia pseudotuberculosis* var. *pseudotuberculosis* or *Yersinia enterocolitica*

Distribution: Both species of bacteria appear to occur world-wide.

Ecology and Epidemiology: These two bacteria are considered together for convenience because they can produce similar diseases and lesions in all affected species. They are not the same bacteria, however, and their ecology and epidemiology may be different.

Y. p. pseudotuberculosis (referred to henceforth as "*Y. pseudotb.*") infects a huge number of species of mammals, birds and reptiles. It can cause disease in these animals but also may infect individuals that develop no illness but do maintain the bacterium and shed it in their feces, regularly or periodically. Infection is by ingestion of material contaminated by feces or by infected tissues of diseases animals. There are six different serotypes of this bacterium, most of them further subdivided into subtypes. The ecology and epidemiology of these serotypes and subtypes may differ, but this is not yet known. With a few exceptions, the occurrence of disease due to *Y. pseudotb* in animals and humans is sporadic. Human and animal disease due to this bacterium appears to occur much more commonly in Europe and eastern Russia than in North America. For example, 26% of 1,129 dead brown hares (*Lepus europaeus*) examined in France in 1993 had died of this infection. By contrast, the disease in North America is recognized relatively infrequently and as sporadic cases. One large epidemic among Muskoxen (*Ovibos moschatus*) in the Canadian arctic is known to have occurred. In Saskatchewan, disease due to this bacterium is seen with some regularity in beavers and muskrats.

Y. enterocolitica has been subdivided into 5 biotypes and 34 serotypes. As a species, the bacterium has been found in a wide range of mammals, birds and reptiles. In general, the serotypes found in animals are not the same as those that usually cause disease in humans. Thus, most human disease is not due to transmission of the bacterium from wild animals. This does not mean, however, that the biotypes and serotypes of the bacterium that occur in wild animals do not pose a risk of infection to humans. They do. *Y. enterocolitica* causes disease sporadically in wild species. Transmission is by ingestion of material contaminated with feces from infected animals. In Saskatchewan, muskrats dead of this infection have been seen occasionally.

Signs and Symptoms of the Disease: **In Animals**, disease caused by either species of bacterium can take several forms. Acute disease with rapid death due to wide-spread and overwhelming infection (septicemia) has been described in wild muskoxen and also in captive red deer (*Cervus elaphus*) on farms in New Zealand. In these cases, there is also a severe inflammation on the intestines (enteritis). More often, the infection results in a more prolonged disease and typical lesions in animals found dead of the disease are abscesses in lymph nodes along the intestinal tract, numerous tiny pale spots on the surface of the liver and spleen and enlargement of the

spleen. With *Y. pseudotb* and especially with *Y. enterocolitica*, there is usually enteritis. **In Humans**, the diseases produced by these bacteria are similar to those in animals. Most often, children, adolescents and young adults are affected. Inflammation of the lymph nodes along the intestine is the most common form of disease from *Y. pseudotb*, and can be associated with abdominal pain similar to that of appendicitis. Diarrhea occurs in about 20% of patients. Illness may be prolonged for up to six months. Infection with *Y. enterocolitica* more often causes severe diarrhea lasting for a few days to up to two weeks, particularly in young children. An appendicitis-like disease also can occur.

Diagnosis of the Disease: Identification of the organism in the feces of the affected animal or human patient, or in the tissues of dead animals is the primary method of diagnosis. The lymph nodes along the intestine are the best organs to culture. These bacteria can be difficult to isolate and use of a special culture medium (CIN medium) is recommended. In addition, repeated cultures made weekly for several weeks from tissues kept at 5C (refrigerator temperature) is often required in order to isolate these bacteria from among other bacteria that may be present in partially-decomposed tissues.

Treatment, Prevention, Control: Treatment of the disease in humans involves both antibiotic drugs and appropriate supportive care. Wildlife personnel should not ignore any signs of significant illness but should immediately seek medical attention and inform the attending medical personnel of the fact that the patient has regular contact with wildlife or wild habitats. There is no means to control the occurrence of these bacteria and the diseases they cause in wild animals. Contracting infection from wild animal sources can be prevented by common-sense sanitary procedures such as use of gloves and protective clothing when handling animals, their tissues or their feces and preventing rodents or other species from contact with food or drinking water.

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LYME BORRELIOSIS

Name of the disease: Lyme borreliosis

Causative Agent: Lyme borreliosis is a disease of people and animals caused by a spirochetal bacterium, *Borrelia burgdorferi*, which resembles in some ways the bacteria that cause relapsing fever, leptospirosis, and syphilis. It is maintained in nature in a "reservoir" of infected vertebrate hosts, and is transmitted among susceptible hosts when vector ticks take a blood meal. As a disease transmitted from animals to people, it is known as a "zoonosis".

Susceptible Species: Tick vectors usually are members of the genus *Ixodes*. In eastern and north-central North America, the most common cycle involves the deer tick, *I. scapularis* (formerly *I. dammini*) which uses white-tailed deer as host for the adult stage, and a variety of birds and small mammals, principally the white-footed mouse (*Peromyscus leucopus*) as the hosts for the immature stages. Similar cycles have been identified in other areas. In western North America (California, Oregon and Washington), the principal cycle involves the dusky-footed woodrat (*Neotoma fuscipes*) and a host-specific tick, *I. neotomae*. Bacteria from this cycle can spill over into other vertebrates if another species of tick (*I. pacificus*), which has a wider host range, feeds first on infected woodrats, then on other small mammals or on people. In the southwestern USA a cycle involving the Mexican woodrat and *I. spinipalpis* has recently been identified, and elsewhere cycles involving rabbits and their ticks have been recognized. However, since *I. spinipalpis* and rabbit ticks don't feed on people, these cycles pose little direct zoonotic hazard. In Eurasia, *I. ricinus* and *I. persulcatus*, which feed upon a wide variety of host species, are the primary vectors.

There is little evidence for a deleterious effect in wildlife due to infection in any of these cycles. However, human beings bitten by an infected tick vector are susceptible to *B. burgdorferi* infection, and may develop a condition known as Lyme disease or Lyme borreliosis. Laboratory animals, particularly certain strains of rats and mice, are used as experimental models of Lyme borreliosis, and there is evidence that *B. burgdorferi* causes illness in domestic animals, particularly dogs.

Geographic Distribution: In Canada, the distribution of populations of vector ticks capable of infecting people with *B. burgdorferi* is limited. At Long Point, on Lake Erie, *B. burgdorferi* cycles in white-footed mice and white-tailed deer via the vector tick *I. scapularis*. Elsewhere in Ontario, individual *I. scapularis* have been found sporadically on people or animals with no history of travel to Long Point, or to areas outside of the province where the vector tick occurs ("endemic" areas). Similarly, in Quebec, Manitoba and the Maritime provinces, *I. scapularis* is occasionally encountered. Established populations of *I. scapularis* have not been confirmed in any of these areas, despite repeated searches. These ticks probably have been transported into scattered areas as immatures, while feeding on birds migrating from endemic areas to the south. Unless such ticks find a mate, they will not establish populations, but if they are infected with *B.*

burgdorferi, they may transmit Lyme borreliosis. In British Columbia, *I. pacificus* infected with *B. burgdorferi* occurs in the lower Fraser River valley, on Vancouver Island, and on the Gulf Islands.

Ecology of Lyme Borreliosis: Lyme disease has only recently been defined as a clinical entity in people, and *B. burgdorferi* was not described until 1982, yet there is evidence that the bacterium and the disease have been present in some areas for many years. It seems likely that ecological circumstances critical to populations of the vector tick have changed in a manner which has increased the opportunity for human disease. Two significant changes in this regard are higher population densities of white-tailed deer in the northeastern and upper midwestern USA, and increased contact, through exurban development in these areas, of deer with susceptible host species, such as people and their domestic animals. Thus, although Lyme Disease has been recognized as an emerging disease, it is not new.

I. scapularis and the other species of Ixodid ticks which transmit the disease to people are capable of feeding upon a wide range of hosts. Ticks seeking a host "quest" on vegetation in the environment, waving burr-like hooks on their forelegs; these they use to cling to animals or people which they contact. The bacterium is introduced into reservoir hosts, usually ground-frequenting birds or small mammals, through the bite of an infected nymphal tick. These hosts then serve as reservoirs of infection which will subsequently infect the next generation of ticks. The adult stage of the tick is found mainly on deer, on which the females feed, in order to prepare for egg deposition. Deer are considered to be a dead-end host for the bacterium, since it is not transmitted to the next generation of ticks through infection of the eggs produced by female ticks, nor is it transmitted from deer directly to other hosts. Deer are, however, critical for reproduction and maintenance of tick populations. People and domestic animals are accidentally infected when fed upon by an infected nymphal or adult vector tick.

Effects Upon the Host Species: The small mammals and birds which act as reservoirs of infection seem to be little affected by *B. burgdorferi*. Early in infection, spirochetes are found in the bloodstream, but later they localize in tissue, including the bladder and ears. Usually, no disease is seen in conjunction with these infections. Similarly, deer show no ill effects of infection, apart from the irritation and inflammation associated with heavy tick infestations.

In contrast, in human beings who develop Lyme borreliosis, following inoculation of the bacterium there is usually local inflammation around the site of the tick bite, producing a round, spreading target-like rash from 3 to over 20 cm in diameter, known as erythema migrans. An acute flu-like illness which usually resolves after a short period of time may follow. In some people, subsequent recurrent episodes of illness, usually involving joints, heart or nervous system, may persist over a period of years, if not diagnosed and treated. Untreated Lyme borreliosis is a debilitating, but usually not fatal, illness. In dogs, recurrent shifting lameness is the principal clinical manifestation of Lyme borreliosis.

Significance: Lyme borreliosis is a major public health concern in endemic areas, where the costs of prevention and treatment may be considerable. It is now the most common arthropod-borne disease diagnosed in the USA, at approximately 8,000 cases per year; at least 15

states have endemic vector tick populations. Persons most at risk are those whose occupation or recreation involves spending long periods outdoors in suitable tick habitat. Thus, forestry and recreation workers, and other people engaged in outdoor activities or living in vector tick-infested areas, are at risk. In Canada, a small number of cases (e.g. in Ontario, about 15-25) are reported annually, many of which involve a history of travel to endemic areas within or outside Canada, though some sporadic cases appear to have been acquired in unpredictable localities, probably from ticks carried in on birds. There are no significant effects of this infection upon wildlife populations.

Method of Diagnosis: The identification of endemic areas involves proof of established populations of competent vector ticks. This is best done by surveying suitable habitat for ticks, usually by dragging flannel cloth through the survey area to capture questing ticks, or by examining live-trapped small mammals or hunter-killed deer for the presence of vector ticks. If ticks are found, they can be examined for the presence of spirochetes. Organs from mice suspected of harbouring ticks can be collected and cultured for the presence of *B. burgdorferi*. This bacterium is difficult to grow, even from experimentally infected animals. Recently, the polymerase chain reaction (PCR) has been developed for the identification of infected ticks or hosts.

In suspected clinical cases of Lyme borreliosis, diagnosis rests upon a combination of compatible clinical signs, a history of known or possible exposure to vector ticks, and measurement of specific antibodies in blood serum.

In both people and animals, the confirmation of infection is problematical. The tests available have shortcomings in both their ability to identify infected animals and in their propensity for incorrectly identifying non-infected individuals as being infected, often due to cross reactions with other, similar organisms.

Management Implications: There are no practical and environmentally acceptable methods for controlling populations of vector ticks, other than on a local scale. Vegetation management and insecticide treatment of mouse habitat have been tried with varying degrees of success. While sustained severe reduction of selected deer populations which are isolated from immigration, and exclosure of deer from small areas, may lead to diminished populations of *I. scapularis*, in most areas deer management is not a practical means of reducing the risk of exposure to vector ticks.

Vector tick-endemic areas need to be identified. People considering access to such areas may avoid doing so, or they can adopt measures to prevent tick bites and possible infection. Light-coloured long-sleeved shirts and trousers, with gaiters or long socks into which pants are tucked, render ticks more visible for manual removal and help prevent them from reaching the skin. Insect repellents containing high concentrations of DEET, used on clothing or exposed body surfaces, deter vector ticks from attaching. After being in suitable habitat, people should check themselves thoroughly for the presence of ticks, paying particular attention to the groin, armpits, and above the hairline on the head. While adult females 5-10mm long are more readily detected, nymphs are only a few millimetres in size and may be missed in a casual inspection. Ticks should be removed by grasping the mouthparts at the skin surface with fine forceps (tweezers), and pulling them straight out. The bite site should be treated with a disinfectant to

prevent secondary infection. If a skin rash over 3-4 cm in diameter, or flu-like illness, develops within the next 3-4 weeks, the individual should report the signs and a history of tick bite to a physician. Antibiotic treatment is effective in curing Lyme borreliosis if it is diagnosed early. Chronic disease is more difficult to treat.

Vaccines are under development. A bacterin is available for use in dogs, and is apparently effective under conditions of experimental challenge. No vaccine is currently available for people.

The provision of public health information is a major challenge for agencies operating in endemic areas and in other locations where there is considerable public concern regarding health risks of Lyme borreliosis. Prudent measures for personal protection, and the relatively low probability of contracting Lyme borreliosis in most parts of Canada, should be emphasized, to keep public concern at a level appropriate to the risk, while providing effective means of reducing exposure to all tick-borne diseases.

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CHLAMYDIOSIS

Name of the disease: Chlamydiosis, psittacosis, ornithosis

Causative agent(s): The bacterium *Chlamydia psittaci* which is a fastidious intracellular organisms that only grows in living cells.

Distribution: *Chlamydia psittaci* has been described from a very large number of bird species, and likely any species should be considered as a potential host. The organism occurs world wide. Other strains of this organism that occur in mammals and also in reptiles will not be considered here.

Ecology/epidemiology of the disease: The causative organism occurs in two distinct forms: a resistant form called the *elementary body* that is shed in feces and secretions of infected birds and which is the infectious form of the disease. Elementary bodies are very resistant to conditions outside the body and will persist in dried feces for an extended period of time. Birds and humans become infected by inhaling elementary bodies, e.g. in dust, or less commonly by ingesting the organisms. Within the body, elementary bodies enter cells and transform to the second form, the *reticulate body*, which is found only within cells and is not infectious. The reticulate body undergoes multiple divisions within the cell and then elementary bodies form and are released when the cell dies. Infected birds shed these in their excreta and secretions. There are several strains of *C. psittaci* and these are associated with different host species and cause differing degrees of disease.

Signs and symptoms of the disease: Most infections in birds likely cause no detectable disease. However, some birds develop severe and fatal infections. Signs have not been described in wild birds; domestic poultry stop eating, become thin and depressed, and pass green gelatinous droppings. Infected ducks and pigeons may have crusting about the eyes because of conjunctivitis. At necropsy the lungs are congested, the heart and liver may be coated with fibrin and the air sacs contain exudate. The liver and spleen are enlarged.

The disease in humans is highly variable. The incubation period following exposure is 1 to 2 weeks. The infection may not cause noticeable illness or pass as a minor respiratory illness. In more severe cases, these is sudden onset of fever, chills, muscle pain, headache, and respiratory difficulty. 80% of hospitalized cases have radiographic evidence of pneumonia. Normally symptoms persist for 7-10 days; the disease may be prolonged in patients not treated appropriately. Relapses are relatively common.

Methods of diagnosis: Diagnosis in birds is by isolation and identification of the organism. Diagnosis in human patients is usually made on the basis of an increase in the level of serum antibodies at the time of illness.

Prevention, treatment and control: Human infection usually occurs as a result of inhalation of aerosols containing elementary bodies. This can occur through inhalation of dust containing fecal material or inhalation of aerosols from carcasses of birds at necropsy. A respirator could be worn when working with birds in dusty situations. When "high-risk" species, such as psittacine birds, are to be necropsied, the carcass should be wetted with disinfectant prior to necropsy to reduce dust and a face mask should be worn.

Chlamydiosis in humans responds well to appropriate antibiotic therapy. It is important to tell your physician that you have been working with birds and may have been exposed to this disease because the signs of chlamydial infection are similar to many other respiratory infections. Most physicians are unfamiliar with the disease and usually do not consider it as a possibility unless they are aware of a history of recent contact with birds.

Key References:

Crosse, B.A. 1990. Psittacosis: a clinical review. Journal of Infection 21: 251-259.

Wobeser, G. and C.J. Brand. 1982. Chlamydiosis in two biologists investigating disease occurrences in wild waterfowl. Wildlife Society Bulletin 10:170-172.

ROCKY MOUNTAIN SPOTTED FEVER

Name of the Disease: Rocky Mountain Spotted Fever

Causative Agent: The rickettsial bacterium - *Rickettsia rickettsii*

Distribution: *Rickettsia rickettsii* is present throughout North, Central and South America. In North America, human disease occurs in the west, particularly in the range of the common wood tick *Dermacentor andersoni* and in the east, particularly in the range of the "eastern wood tick" or "American dog tick" *Dermacentor variabilis*. The actual distribution of the organism in North America appears to be incompletely known and not closely monitored. It was reported from all states except Maine, New Hampshire, Alaska and Hawaii between 1978-80. The organism is known to be present in Canada from Nova Scotia to British Columbia, but the precise distribution across the country has not been reviewed. The disease was once considered exclusive to the rocky mountain region, but it was found subsequently to cause more human illness in the states along the east coast than in the west.

Ecology and Epidemiology: *R. rickettsii* normally lives in association with wild rodents and lagomorphs and their ticks. Ticks are the only known means of transmission of the organism among susceptible host species. The most important tick vectors in Canada are *Dermacentor andersoni* and *D. variabilis*. Some other species of tick also can act as vectors. Ticks can themselves maintain the infection since the organism is transferred from infected females to their eggs and, hence, between generations of ticks. Even in areas where the organism is relatively common, however, the rate of infection among ticks is low. Humans are abnormal hosts for this organism and do not contribute to maintenance of the organism in nature. However, the organism can cause serious disease in humans. Expansion of cities and suburban developments into habitat rich in rodents, lagomorphs and ticks is thought to have contributed to the high incidence of the disease in eastern human populations. Since the introduction of antibiotics in the 1940's, the high fatality rate from this disease (average of 20%) has vastly diminished and many cases go unrecognized because effective therapy is initiated early in the illness and the diagnosis is never made. Domestic dogs may be important in human disease because they can carry infected ticks into households, thereby increasing the likelihood of human infection.

Signs and Symptoms of the Disease: **In wild animals**, disease does not seem to occur or has not been recognized. A wide range of wild rodents and lagomorphs as well as opossums and domestic dogs have been found to be infected. Wild rodents may have large numbers of the organism in their blood for long periods but show no signs of illness. Experimentally, domestic dogs have been made ill by infection with *R. rickettsii*. **In humans**, the disease was once severe and commonly fatal, and it remains so for untreated cases. Symptoms appear 2 days to 2 weeks after infection by tick bite. Initial symptoms are not at all diagnostic and consist of fever, headache, chills, and muscle and joint pain: so-called "flu-like" symptoms. The typical skin rash of small red spots develops after 3-6 days of febrile illness, and may not develop at all in cases

treated early. Characteristically these spots occur on the palms and soles, as well as elsewhere. The disease can progress to affect the brain, heart and lungs, and can last for weeks to months in untreated cases. The overall fatality rate of cases in the United States, treated and untreated, between 1978-80 was 4.5%.

Diagnosis of the Disease: Diagnosis requires isolation of the organism from the blood of patients. This can be done by inoculation into guinea pigs or into embryonated hens' eggs. Diagnosis also can be made or verified by detecting the appearance of high levels of antibodies in the blood of patients that have become ill. However, these antibodies do not appear until at least 6 days after the onset of the symptoms of the disease. Presence of *Rickettsia rickettsii* among wild animals is often assessed by detecting antibodies to the organism in the serum of domestic dogs that have access to the outdoors. The prevalence of dogs with antibodies is then used as an index to the prevalence of infection among wild animals and their ticks in the local area.

Treatment, Prevention, Control: Antibiotic therapy is effective, particularly when begun early in the course of the disease. Since this disease, like so many other zoonotic diseases, has no symptoms that are unique or diagnostic, particularly in the early stages when treatment is most effective, wildlife personnel should not ignore the onset of symptoms but should seek medical attention immediately. They should inform the attending medical personnel that they have regular contact with wildlife and wild habitat, and that zoonotic infections should be considered.

There is no method to control the infection among wild animals and their ticks. Local application of aracacide poisons has been used to eliminate ticks from small local environments. Prevention of human disease is achieved by precautions to reduce the likelihood of tick bites. Fortunately, ticks must remain attached for at least 10 hours before infection will occur. Thus, use of repellents and protective clothing and thorough searches for and removal of ticks twice a day, particularly during seasons of high tick activity, are effective preventive measures. Taking pet dogs into tick-infested areas increases the risk that infected ticks will be brought into the home. No effective vaccine against the disease is currently available.

Key References:

Acha, P.N. and Szyfres, B. 1987. Zoonoses and Communicable Diseases Common to Man and Animals. Pan American Health Organization Scientific Publication No. 503, Washington, DC.

Bernard, K.W., Helmick,C.G., Kaplan,J.E., and Winkler, W.G. 1982. Surveillance of Rocky Mountain Spotted Fever in the United States, 1978-1980. Journal of Infectious Diseases 146: 297-299.

Burgdorfer, W. 1975. A review of Rocky Mountain Spotted Fever (Tick-Borne Typhus), its agent, and its tick vectors in the United States. Journal of Medical Entomology 12: 269-278.

RINGWORM

Name of the disease: Ringworm (dermatomycosis or dermatophytosis)

Causative agent: Ringworm results from infection of the keratinized layer of the skin by fungi (dermatophytes) belonging to three genera: *Trichophyton*, *Microsporum* and *Epidermophyton*.

Distribution: Ringworm infections have a worldwide distribution and infect a number of different species of animals as well as humans. Little information is available on ringworm infections in wild animals; however, it has been diagnosed in mule deer, opossums, foxes, squirrels and other rodents. Ringworm has been frequently diagnosed in mule deer from southern Saskatchewan and Alberta. *Trichophyton verrucosum*, a species commonly found on cattle, has been isolated from the skin of affected deer.

Ecology and epidemiology of the disease: Species of dermatophytes are adapted to specific hosts and, in general, infections in these hosts tend to be less severe. Species that are adapted to humans are called anthropophilic species, the most common being species of *Epidermophyton*. Zoophilic fungi are found primarily on animals but can infect humans, and geophilic fungi are normally found in soil but are capable of infecting animals and people.

Transmission occurs by contact with fungal hyphae and their spores either directly, or indirectly in bedding, feed and on other objects. Mild abrasions of the skin are required for the fungus to become established. Fungal hyphae do not invade living tissue but are confined to the keratinized layer of the skin, hair follicles and hair shafts. Hyphae break up into small oval spores, or conidia, which remain viable in the environment for long periods of time. Ringworm is most common in young and debilitated animals.

Signs and symptoms of disease: Many ringworm infections are asymptomatic and these inapparent carriers act as reservoirs. Ringworm infections are characterized by focal expanding areas of skin inflammation, thickening, crusting and hair loss. Hair loss is due to branching fungal hyphae invading the hair shaft making it brittle and prone to breakage. Inflammation varies from mild to severe depending on the species of fungus and host involved. The stimulus for inflammation are soluble substances produced by the fungus which diffuse down into underlying layers. The disease usually regresses over a period of weeks to months.

Skin lesions in mule deer are usually confined to the face and legs. They appear as dry, grey, scurfy areas with partial or complete hair loss. Ringworm appears to have little effect on mule deer in most cases.

Ringworm in humans is similar to that in animals but infections arising from zoophilic species tend to be more severe. Skin lesions are red, expansive and may have a central healed zone, hence the name ringworm. Lesions can vary from dry and scaly to moist and exudative. Lesions may be pruritic.

Methods of diagnosis: The diagnosis can be made by skin scrapings and fungal culture and by examination of skin samples with the light microscope. Some species of dermatophytes fluoresce under UV light which may be used to diagnose infection.

Prevention, treatment, control: Rubber gloves should be worn when handling animals with skin conditions, realizing that in a few cases animals with normal appearing skin may harbour dermatophytes. Treatment involves topical application of antifungal agents, sometimes for extended periods.

Key references:

Yager, J.A. and D.W. Scott, 1993. The skin and appendages. In Pathology of Domestic Animals, K.V.F. Jubb *et al* (ed.), Academic Press, Inc., San Diego, California. p.661-666.

Wobeser, G., S.A. Mitcham, J.R. Saunders and H.M. Hunt, 1983 Dermatomycosis (ringworm) in mule deer (*Odocoileus hemionus*). Canadian Veterinary Journal 24:316-317.

ECHINOCOCCOSIS

Name of the disease: In humans, infection with *Echinococcus granulosus* is called **hydatid disease** or **cystic hydatid disease** and infection with *Echinococcus multilocularis* is called **alveolar hydatid disease**. The term **Echinococcosis** is sometimes used to refer to both conditions.

Causative agent(s): These conditions are caused by infection of humans with the larval stage of two tapeworms, *Echinococcus granulosus* and *Echinococcus multilocularis*.

Distribution: *Echinococcus granulosus* is widely distributed across Canada and different species are involved as hosts in different regions; however, the larval form is always found in an ungulate and the adult tapeworm lives in the intestine of a canid. In the far north, this parasite cycles between caribou and wolves; in the boreal forest it occurs in moose, elk, and less commonly in deer, and in wolves, coyotes, and dogs; it also occurs in mountain goats and sheep. The parasite is uncommon in deer in southern Canada. In other parts of the world, this parasite cycles between domestic livestock (particularly sheep) and dogs, but this does not occur in Canada.

Echinococcus multilocularis has a more limited geographic distribution and a different range of hosts. The larval form occurs in rodents(usually voles and mice, but also muskrats) and the adult stage occurs in the intestine of foxes (Arctic and red), coyotes and, less commonly, in cats and dogs. The parasite is present in the Northwest Territories but the distribution has not been studied. It is present in Alberta, Saskatchewan and Manitoba. This parasite is spreading east and south in the USA.

Ecology/epidemiology of the disease: These parasites have a life-cycle in which two hosts are required. The adult tapeworm lives in the small intestine of a carnivore and produces large numbers of microscopic eggs that are passed with the animals's feces. Herbivores (ungulates in the case of *E. granulosus,* rodents in the case of *E. multilocularis*) become infected by ingesting eggs, likely as contaminants on vegetation. The eggs are extremely resistant and persist in the environment after the droppings have decomposed. Humans become infected by ingesting eggs; this usually occurs through poor hygiene in association with handling carnivore droppings, carnivore pelts that may be contaminated with feces, or from environments that are contaminated with dog feces. In herbivores (and humans) the eggs hatch after ingestion and migrate to preferred sites in the body (usually lung or liver) and begin to develop as a fluid-filled cyst containing many larval forms. Carnivores become infected by consuming the cysts while eating infected prey, e.g., moose or caribou for wolves; mice for foxes. The larvae then develop to the adult stage in the carnivore's intestine. Humans are a "dead-end" host since they are not usually consumed by carnivores.

Signs and symptoms of the disease:

Echinococcus granulosus: The adult tapeworm has no detrimental effect on the carnivore. The larval cysts cause problems because they continue to expand in size and compress tissue, e.g. cysts grow to golf-ball size or larger and may interfere with functioning of the lungs. There is some evidence that heavy infections in moose may be debilitating. In humans , developing cysts in lung may be associated with fever and respiratory difficulty. Occasionally cysts may develop in other organs, including the brain , and cause severe problems because of pressure on normal tissue.

Echinococcus multilocularis is a more dangerous and damaging parasite because the larval cysts grow rapidly, and bud externally, so that they can destroy or replace large areas of tissue. The larval parasite acts very much like a malignant cancer and can spread from one tissue to others. This parasite debilitates and eventually will kill the normal rodent host. The disease in humans is slowly progressive and often not diagnosed until well advanced. The liver is usually involved causing abdominal pain and swelling, with jaundice in some cases. In areas of the world where human alveolar hydatid disease is common, as many as 70% of untreated patients progress to a fatal outcome within 5 years.

Methods of diagnosis: Infections with either species of *Echinococcus* can be diagnosed in carnivores by finding eggs in the feces; however, the eggs are difficult to distinguish from those of other common tapeworms. The cystic larval stages can be identified on the basis of their gross appearance in ungulate and rodent hosts. Infection in humans can be recognized by radiography, CT scan, and by a variety of serologic tests.

Prevention, treatment and control : Since both forms of echinococcosis are acquired by ingestion of eggs from carnivore droppings, careful personal and food hygiene is the most important preventive measure. Care must be taken in handling carnivore droppings (scats), carnivore intestines (always handled with rubber gloves) and carnivore pelts that may be contaminated with feces. **Viscera from infected game animals must not be fed to dogs**, as the dogs will become infected and then contaminate the environment with eggs. Tapeworm infections in dogs and cats should be treated with anthelmintics. Human cases may be treated with antiparasitic drugs or by surgery.

Key references:

Rausch, R.L, J.F. Wilson, and P.M. Schantz. 1990. A programme to reduce the risk of infection by *Echinococcus multilocularis*: the use of praziquantel to control the cestode in a village in the hyperendemic region of Alaska. Annals Trop. Med. Parasitol. 84: 239-250.

Hildreth, M.B., M.D. Johnson, and K.R. Kazakos. 1991. *Echinococcus multilocularis*, a zoonosis of increasing concern in the United States. Comp. Cont. Vet. Educ., Small Animal. 13:727-741.

SARCOPTIC MANGE

Name of the Disease: Sarcoptic mange

Causative agent: Mange is caused by *Sarcoptes scabiei*, one of a group of small skin parasites called mites. The structure of mites are similar to that of ticks; however, unlike ticks they are too small to be seen with the naked eye.

Distribution: *Sarcoptes scabiei* infections have been reported in man, canids, felids, mustelids and ursids as well as in numerous noncarnivores throughout the world. In North America enzootic mange is seen in red fox, timber wolf, red wolf and coyote.

Ecology/epidemiology of the disease: Populations of *Sarcoptes scabiei* have evolved a certain degree of host specificity so that human adapted varieties spread more rapidly on humans than on other species, dog strains spread more readily on dogs, and so on. Cross infections do occur but are usually temporary and atypical. The life-cycle of *S. scabiei* is completed within burrows in the skin of infected hosts. Adults mate in moulting pockets near the skin surface. After mating the female uses cutting mouthparts and hooks on its legs to burrow through the skin. As it burrows it lays eggs at a rate of 1 to 3 per day. The eggs hatch and both larval and nymphal stages continue to migrate through the epidermis.

Mange is highly contagious. Transmission typically occurs by direct transfer of mites at any stage of their development. Transmission can also occur indirectly by contact with contaminated objects such as bedding. Mange in wildlife species predominately affects young animals and is more prevalent when populations are high.

Signs and symptoms of the disease: Mange infections in animals are characterized by oily skin, crusting, hair loss, and scab formation. Infections typically begin on elbows and pinna of ears and can progress to involve large areas of the body. Lesions are a result of physical damage to the skin, irritation caused by parasite excretions and the allergic response of the host. Affected skin is itchy or pruritic and often there is severe self trauma. Severely affected animals are frequently emaciated.

Humans infections usually occur at sites previously in contact with infected animals, such as hands and arms. The skin becomes inflamed, red and intensely itchy. Infections are usually transient.

Methods of diagnosis: Diagnosis is made by identifying the mite with a light microscope in scrapings from the margins of affected skin.

Prevention, treatment and control: Infection can be prevented by wearing gloves and coveralls when handling infected animals. Acaricides are used to treat infected animals and people. Treatments often have to be repeated over several weeks.

Key References:

Sweatman, G.K., 1971. Mites and Pentastomes In: Parasitic Diseases of Wild Mammals, J.W. Davis and R.C. Anderson (ed.), Iowa State University Press, Ames , Iowa. p.31-36.

TRICHINOSIS

Name of Disease: Trichinosis (also known as Trichinellosis)

Causative agent: Trichinosis is caused by a tiny nematode or roundworm called *Trichinella spiralis*.

Distribution: *Trichinella* infections are reported from all continents except for Australia and Antarctica. Trichinellosis is a major problem in the arctic regions where a high proportion of species are carnivorous or omnivorous thereby perpetuating the disease. *Trichinella* sp. have the widest host range of any helminth. At least 22 species of carnivores and several predatory birds have been implicated in transmission of the disease in North America. Almost all species can be experimentally infected with *Trichinella* but carnivores and omnivores, such as bears, coyotes, wolves, pigs, seals and wolverines, are most important in the natural transmission of the parasite.

Ecology and epidemiology: The life cycle of *Trichinella* is unique because both adult and larval stages occur in a single host. Transmission occurs when the meat from an infected host is eaten by a predator or scavenger. Larval stages within cysts in the muscle are released during digestion and invade the mucosa of the small intestine of the new host. Larvae develop to adults 2 days after infected meat is eaten and after 5 days females are producing live young which are deposited directly into the mucosa. Larvae migrate via the bloodstream to striated muscle. They invade the muscle, grow and eventually coil up within a small cyst. Development to the L1 stage, encysted in the muscle, takes 2 - 3 weeks at which time they are infectious to another host. Cysts remain viable in tissue for at least 6-12 months and remain infectious even after extreme putrefaction allowing transmission to occur during scavenging.

Human trichinosis usually results from eating undercooked pork, bear or seal. Meat must be at a uniform temperature of 77^{o} C throughout before larval stages of *Trichinella* will be killed. Microwave cooked meat often is not uniformly heated to this temperature allowing larvae to survive. Freezing pork products at - 15^{o} C for 3 weeks has long been considered sufficient to kill larval stages but does not work for bear strains of Trichinella. Larval stages in bear meat frozen at -20^{o} C are still infectious after 6 months.

Signs and symptoms: Clinical trichinosis is rarely diagnosed in domestic animals and its effect on wild animals is completely unknown. Disease can occur at two stages of infection, in the intestine and during muscle invasion. Infection of the small intestine by adults can result in diarrhea which may become hemorrhagic. This stage of the infection is of variable duration, lasting slightly over 1 week in dogs and 3 to 4 weeks in humans. Invasion of muscle by larval stages can result in muscle pain, edema, fever and increased eosinophils in circulation. Large doses can result in death.

Human trichinosis sufferers may develop edema around the eyes, muscle pain, fever, diarrhea, pruritis, conjunctivitis and skin eruptions. Eosinophil numbers in the blood are often extremely elevated. Infected individuals less commonly develop difficulty in breathing (due to infection of the diaphragm), encephalitis and heart failure.

Methods of diagnosis: Larval stages in muscle can be identified by squashing small pieces of muscle between glass plates and examining with the light microscope. Digesting the muscle with a solution of pepsin and hydrochloric acid followed by sedimentation can also be used to collect encysted larvae. Active muscle such as the diaphragm, tongue and masseter muscles generally have the largest concentrations of larvae. Diagnosis of infected humans is based on history, clinical signs and a rising antibody titre to *Trichinella*.

Prevention, treatment and control: Proper cooking of meat prevents infection. Infected individuals can be treated with anthelmintics to kill the nematode and with anti-inflammatories to ameliorate the symptoms.

Key references:

Dau, J. and R. Barrett, 1981. Trichinella. *In*: Alaskan Wildlife Diseases, R.A. Dieterich (ed.) University of Alaska, Fairbanks, Alaska. p151-161.

GIARDIASIS

Name of the disease: Giardiasis, sometimes called "Beaver fever".

Causative agent: Giardiasis is caused by a flagellated protozoan parasite in the genus *Giardia*. Taxonomy of this genus is important in understanding the epidemiology of this disease but is complex, controversial and likely needs to be revised. Three species can be identified based on parasite morphology. *Giardia agilis* has a long narrow trophozoite with long teardrop-shaped median bodies arranged parallel to the long axis of the body and is only found in amphibians. *Giardia muris* has 2 small oval median bodies in the centre of a pear-shaped body and is found in a variety of birds, rodents and reptiles. *Giardia duodenalis*, also known as *G. intestinalis* or *G. lamblia* has median bodies which are shaped like the claw of a claw hammer and lie transversely in the body of the trophozoite. *G. duodenalis* infects a variety vertebrate hosts including: humans, rodents, reptiles and perhaps birds. Transmission studies and molecular studies of this species indicate it likely should be separated into several new species.

Distribution: *Giardia* has a world wide distribution and infects many different species of vertebrates.

Ecology and epidemiology of the disease: *Giardia* has a simple asexual life cycle. Trophozoites, the stage responsible for disease, are found covering the mucosal lining of the small intestine. Trophozoites divide by binary fission within the intestine, reaching extremely high numbers in some cases. Poorly defined cues cause trophozoites to become encysted and pass to the external environment with feces. The cyst stage can persist in water for long periods of time, especially under cool conditions. Ingestion of cysts by a new host completes the life cycle. Understanding the epidemiology of this disease is hindered by ambiguity of taxonomy and inability to identify the host origin of cysts in water supplies associated with disease outbreaks. Direct fecal-oral transmission is the most common cause of human infections and outbreaks are well recognized in child daycares, residential schools and institutions. Contamination of water supplies and less commonly food are another important source of infection. Other infected humans are the most important reservoir of *Giardia* for humans.

Infected wild and domestic animals have been implicated in transmission of *Giardia* to humans, although to date the evidence is circumstantial. The ability of human strains of *Giardia* to infect other animals is well proven. Aquatic species such as muskrat, beaver, voles and wading birds are potential sources of human infection due to the likelihood of fecal contamination of human water supplies and the high prevalence of infection in these species. Prevalence of infection between 7-44% has been reported in beavers from different locations in North America and the prevalence of infection in muskrats and wading birds, such as herons and egrets, has been greater than 90% in some areas. In a recent outbreak in British Columbia *Giardia* isolates from humans and beaver present in the water supply have been found to be identical based on enzymatic and genetic typing. Evidence such as this indicate that, at the least, aquatic mammals may potentiate an outbreak and it is likely *Giardia* will eventually be proven to be zoonotic .

Surface water supplies are frequently found to contain *Giardia* cysts, even in pristine locations. Typically, several different strains of *Giardia* appear to be circulating in water bodies.

Signs and symptoms of the disease: Disease occurrence in Giardia infections depends on the species or strain of parasite and host. Clinical disease has not been reported from the wild.

Three forms of the disease occur in humans: subclinical or asymptomatic, acute and chronic. Mild or absent symptoms is the most common outcome of infection. Acute disease is characterized by sudden onset of watery diarrhea which may develop to steatorrhea (fatty stool), nausea, abdominal discomfort, bloating and weight loss. Symptoms develop 3 to 20 (mean 7)days after exposure to the parasite. The majority of cases are self-limiting within a 2-4 week period; however, in up to 25% of cases symptoms may persist for 7 weeks or more. In an estimated 30-50% of cases patients will develop chronic diarrhea, with profound weight loss, malabsorption and in some cases symptoms of vitamin and protein deficiencies.

Methods of diagnosis: Diagnosis is based on clinical symptoms and identifying trophozoites or cysts in feces using the light microscope. A diagnosis can also be made based on the presence of an antibody titre to *Giardia* in serum or by identifying *Giardia* antigen in the stool using immunological techniques.

Prevention, treatment and control: Good hygiene prevents transmission by the direct fecal-oral route. Conventional water treatment plants employ coagulation, filtration and chlorination to ensure 99.9% removal of cysts. Chlorination by itself is insufficient. Boiling the water will destroy Giardia.

Key references:

Erlandsen, S.L., 1994. Biotic transmission - Is Giardiasis a zoonosis? In: *Giardia*: From Molecules to Disease, R.C.A. Thompson *et al.*(ed.) CAB International, Wallingford, UK. p.83-97.

Feely, D.E., D.V. Holberton, and S.L. Erlandsen. 1990. The biology of Giardia: In: Human Parasitic Diseases, Vol. 3: Giardiasis, E.A. Meyer (ed.). Elsevier: New York. pp 11-49.

***Baylisascaris procyonis* Larva Migrans**

Name of the Disease: *Baylisascaris procyonis* larva migrans.

Baylisascaris procyonis larva migrans is a disease of animals and people caused by immature stages (larvae) of a parasitic nematode (roundworm) which migrate in tissues. As a disease potentially transmissible from animals to people, it is known as a "zoonosis".

Causative Agent: Adult *Baylisascaris procyonis* worms are found in the small intestine of the raccoon (*Procyon lotor*). Similar worms infect skunks (*B. columnaris*) and bears (*B. transfuga*). They resemble in general appearance and life cycle the common ascarid roundworms of domestic dogs and cats (*Toxocara* spp. and *Toxascaris* sp.).

In the raccoon, *B. procyonis* is transmitted principally by a direct life cycle. Adult worms produce eggs which are shed in the feces of the raccoon. In the external environment, infective larvae develop in the eggs in approximately a month. Infective eggs ingested by susceptible raccoons hatch in the gut, and larvae migrate via the bloodstream through the liver to the lungs. They are then coughed up and swallowed, maturing to the adult stage in the small intestine. Raccoons may also become infected by eating another animal which has ingested infective eggs, and, as a result, has *Baylisascaris* larvae in its tissues.

Susceptible Species: The raccoon is the normal host for this worm, and in this species, it is rarely the cause of disease. Occasionally, localized areas of inflammation and tissue damage will occur, caused by migrating larvae, and there have been reports of intestinal obstruction due to abnormally high numbers of adult worms in the small intestine.

When infective eggs are ingested by other species of warm-blooded animals, the larvae which hatch in the gut may undertake an aberrant migration through tissue, causing widespread damage along their path. Many organs, including lung, liver and heart may be affected, but the most important damage usually occurs in the central nervous system. Species in which brain damage has been reported include human beings and a large number of wild and domestic mammals and birds. Raccoons tend to use communal sites for defecation. These latrines, which may be on the ground, in lofts, caves or the crotches of trees, provide potential sites of *Baylisascaris* infection for animals which forage in these areas. Thus, disease has been reported commonly in woodchucks, grey squirrels, red squirrels, porcupines, cottontail rabbits, and a number of species of ground-foraging birds.

Geographic Distribution: The geographic distribution of the parasite likely mirrors that of its raccoon host. The raccoon is a member of the family Procyonidae, which is primarily a New World tropical group, originating in the South American continent. The raccoon is the only species in this family that occurs in Canada, and it is primarily found in the more southerly portions of the country. Population densities are high in southern Ontario, particularly in urban areas. Its range extends a limited distance into the boreal forest of Northern Ontario and Quebec, and as far east as Cape Breton Island, but not to Anticosti Island or the province of

Newfoundland. In the prairie provinces, it is found in the aspen parkland. Raccoons also occur in the lower mainland of British Columbia and on Vancouver Island. The presence of raccoons at high densities in urban areas is of obvious significance in assessing the zoonotic potential of this parasite.

Significance: As the larval *B. procyonis* migrate through the brain of a susceptible host, they cause extensive destruction of brain tissue, and elicit a strong inflammatory response from the host, which itself causes further damage. The result is the development of severe neurological signs, such as imbalance, circling and loss of normal fear responses. Often, affected animals are suspected of being rabid. Death is the usual outcome, due to any of the direct effects of the worm, increased susceptibility to predation, or through human intervention.

Usually, individual animals are affected, but there are reports of outbreaks of disease due to this parasite affecting many small mammals in a particular locality. There also may be effects on a population level, if susceptible populations are small or isolated. The extirpation of the Allegheny wood rat in New York State has been attributed to mortality caused by this parasite.

These worms constitute a significant zoonotic hazard, and may cause severe damage to the eye and brain of infected people. It is likely an important cause of ocular and visceral larval migrans in people, and there are two reports in the medical literature of deaths in children due to the effects of migrating larvae. In one instance, the child was thought to have become infected by playing with, and likely chewing on, wood from a woodpile used by raccoons. Given the propensity of raccoons to inhabit abandoned or unsealed buildings and to live in close proximity to human beings, and the frequency with which attractive juvenile raccoons are adopted as "orphans", the risks for human infection are obvious.

Method of Diagnosis: In the live raccoon, a routine fecal flotation will detect eggs in a large proportion of infected animals. Juvenile raccoons are more likely than adults to be infected. Presumably, resistance develops in raccoons following initial exposure and infection.

In abnormal hosts infected by larvae, necropsy is the primary way of confirming infection. Gross necropsy findings are often unremarkable, aside from occasional evidence of localized inflammation due to the passage of larvae. Microscopically, in organs such as liver, lung and heart, tissue destruction and characteristic eosinophilic inflammation may be present. Damage due to the growth and movement of larvae, and the host animal's inflammatory response to them, is often most severe in the brain and spinal cord. Microscopic larval worms are occasionally seen in tissue section, and they can be distinguished from other roundworms which are capable of causing similar lesions.

In people, blood eosinophilia and clinical signs compatible with the effects of migrating parasites will suggest infection with *B. procyonis* or a similar parasite, but no definitive diagnostic test is available.

Management Implications: The most serious management concerns are the protection of the public from this zoonotic hazard, by minimizing the potential exposure of people to raccoon feces. Exclusion of raccoons from dwellings, outbuildings and other potential den sites in proximity to human habitation is an important step. Public education concerning the risks of

raccoon feces, and attention to children's hygiene in high-risk environments is also required. Wildlife rehabilitators need to take particular care in the handling and disposal of raccoon feces in order to avoid potential exposures. Most immature raccoons are infected, and they may shed many hundreds of thousands eggs per day. These eggs will persist for years in the environment, and are resistant to common disinfectants. Raccoon feces should never be used as manure, and material which may be contaminated with raccoon feces should not be used as feed or bedding for other animals.

Control of infections in wild raccoons is impossible. However, each year, many raccoons come into the hands of animal shelters and wildlife rehabilitators. It should be a policy of these organizations to regularly deworm all raccoons that come under their care, and to take measures to minimize human exposure to infective eggs.

Key References:

Fox, Amy S., Kevin R. Kazacos, Nevenka S. Gould, Peter T Heydemann, Chinnamma Thomas and Kenneth M. Boyer. 1985. Fatal eosinophilic meningoencephalitis and visceral larva migrans caused by the raccoon ascarid *Baylisascaris procyonis*. New England Journal of Medicine. 312 (25): 1619-1623.

Kazacos, Kevin R. 1989. *Baylisascaris* larva migrans. Journal of the American Veterinary Medical Association. 195(7): 894-903.

INSECTICIDES

Hazardous Chemicals: Insecticides

Names of Insecticide Chemicals:

There are many insecticides on the market that are members of these categories of chemicals. Organophosphates and carbamates are among the most widely used insecticides in North America, but the pyrethroids are becoming widely used as well. Organochlorines are generally used in smaller quantities, but their use continues. Each insecticide has an official common name and is sold in one or more commercial products or formulations, each of which also will have a commercial or trade name. The official common name will appear on the label in the list of ingredients. (Some fungicides and herbicides of the same chemical classes are included in the lists that follow.)

Organophosphates:

Acephate-met
Akton
Azinphos-methyl
Bomyl
Bromophos
Carbophenothion
Chlorphenvinphos
Chlomephos
Chlorpyrifos
Coumaphos
Crotoxyphos
Crufomate
Cyanophenphos
Cyanophos
Cythioate
Demeton
Demeton-methyl
Dialifor
Diamidfos
Diazinon
Dicrotophos
Dimefox
Dimethoate
Dioxabenzophos
Dioxathion
Disulfoton
Ditalimphos
DMPA
Edifenphos
EPN
Ethion
Ethoprop
Etrimfos
Famfur
Fenamiphos
Fenitrothion
Fensulfothion
Fenthion
Fonofos
GC 6506
Malathion
Methidathion
Methyl parathion
Mevinphos
Monocrotophos
Naled
Omethoate
Oxydemeton-methyl
Parathion
Phorate
Phosalone
Phosmet
Phosphamidon
Phoxim
Pirimiphos-ethyl
Pirimiphos-methyl
Ronnel
Sulfo TEPP
Sulprofos
Temephos

Organophosphates (Cont'd)

Dicapthon
Dichlophenthion
Dichlorvos
Isazophos
Isofenphos
Leptophos
TEPP
Terbufos
Tetrachlorvinfos
Triaziphos
Trichlorfon
Vamidothion

Carbamates:

Aldicarb
Aminocarb
Bendiocarb
Bufencarb
Butoxycarboxim
Carbanolate
Carbaryl
Carbofuran
Dioxacarb
Diram
Ethiofencarb
Formetanate
Hydrochloride
Methiocarb
Methomyl
Mexacarbate
Oxamyl
Trimethacarb

Pyrethroids

Allethrin
Barthrin
Bifenthrin
Bioallethrin
Bioresmethrin
Cismethrin
Cyfluthrin
λ-cyhalothrin
d-cis,trans-allethrin
Deltamethrin
Dimethrin
Esbiothrin
Fenpropathrin
Fenvalerate
Flucythrinate
Fluvalinate
τ-fluvalinate
Kadethrin
Permethrin
Phenothrin
Resmethrin
S-bioallethrin
Synthetic pyrethrum
Synthetic pyrethrins
Tefluthrin
Tetramethrin
Tetramethrin (1R)-isomers
Tralomethrin

Organochlorines

Aldrin	Dicamba	Methylene chloride
Benzene hexachloride	Dichloropropane	Mirex
Chlorbenside	Dichloropropene	PCNB
Chlordane	Dicofol	Pentachlorophenol
Chlordecone	Dienochlor	Tetrachloroethylene
Chlorfenethol	Endosulfan	Tetradifon
Chlorobenzilate	Endrin	Toxaphene
Chloroform	Epichlorohydrin	Triclopyr
Chloroneb	Ethylan	
Chloropicrin	Ethylene dichloride	
Chloropropylate	Heptachlor	
DBCP	Hexachlorobenzene	
D-D	Lindane	
DDT	Methoxychlor	

Poisonous Properties:

NOTE: Commercial pesticide formulations contain many chemicals besides the listed pesticide(s) itself. Some of these additional chemicals are highly toxic and are under review for possible discontinued use. These chemicals often are listed simply as "inert ingredients" on product labels and their identities are considered trade secrets. These chemicals may add to the general toxicity of commercial pesticide preparations either by enhancing the toxicity of the pesticide or simply because they are themselves toxic in ways unrelated to the toxicity of the pesticide.

Organophosphates and Carbamates

Organophosphates and carbamates are nerve poisons. They block the action of a particular enzyme in the brain, in nerves and at the connection between nerves and muscles. The enzyme is called acetylcholinesterase and these two classes of chemical poisons are often referred to as anti-cholinesterase pesticides. When the enzyme is blocked, many nerve impulses can not be transmitted, particularly those that go to muscles. The usual fatal result is paralysis of muscles, especially breathing muscles, and suffocation, but there are also more general effects on the central nervous system such that abnormal behaviour of various kinds occurs in victims poisoned sublethally or prior to death in lethal poisonings. Some organophosphates also can produce permanent damage to nerves by a different toxic mechanism. This nerve damage develops a few weeks after exposure to the organophosphate poison, and is known as 'delayed neurotoxicity'.

Pyrethroids

These compounds also are neurotoxins. They alter the normal conduction of nerve impulses and the result is tremors, abnormal motion, seizures and convulsions. Pyrethroids appear to be less toxic to mammals than the other classes of insecticides.

Organochlorines

Organochlorine pesticides affect the central nervous system most importantly, particulary nerve fibres in the brain associated with movement and sensation. Non-fatal exposures may cause toxic damage to other organs, particularly the liver.

Routes of Exposure:

Insecticides are applied as agricultural sprays, granular formulations for soil treatment, as seed coatings, as powders, as paints, as dusts, as pour-on treatments, dips or rub-on preparations for livestock, as household or garden sprays, as veterinary products for pets and as baits of various kinds. Formulations at very high concentration are sold for use as sprays and are either diluted in water or oil, or are applied with equipment that dispenses very small quantities as spray. These concentrated formulations are particularly dangerous. Exposure by contact with the skin can result in a lethal dose. Discarded containers of such formulations will contain potentially lethal doses on the walls of the container. Significant exposure of the skin can occur through gloves if gloves are re-used regularly, since glove material will be penetrated eventually. Incautious removal of gloves or protective clothing also can result in significant exposure of the skin, as can re-use of protective clothing without adequate laundering between uses. Pour-on veterinary products are specifically formulated to be absorbed by the skin. Inhalation of vapours, dusts or sprays or inadvertent ingestion of chemicals allowed to contaminate hands, utensils or food material are additional routes of exposure.

Signs and Symptoms of Poisoning:

In all species, the signs and the duration of illness or the speed with which death results will depend on the particular poison and the dose which has been absorbed. Low doses may lead to mild symptoms and these may continue for long periods if exposure is regular or continuous. More severe effects occur with larger doses. Anyone experiencing symptoms or observing them in others should immediately consults with medical personnel and the most available Poison Control Centre. Any possible exposure to a hazardous chemical should be explained and, the name of the chemical product(s) should be determined and made known to the medical personnel. Emergency treatment may be indicated on the product label as well.

Organophosphates and Carbamates

In animals, signs of acute poisoning range from mild disorientation or other behavioral abnormalities to muscle twitching, perspiration, salivation, diarrhea, nasal and tear discharges, constricted pupils and finally to paralysis proceeding to death. **In humans**, the symptoms that have been associated with the onset of acute poisoning include headache, nausea, dizziness, restlessness, weakness, muscle twitching and tremors, disorientation, incoordination, abdominal cramps, diarrhea, perspiration, salivation, excess tear production, constricted pupils, blurred vision, slurred speech, confusion and hypertension. Signs may progress to paralysis, convulsions and coma.

Pyrethroids

In experimental mammals, the signs of toxicity are tremors or stiff gait progressing in intensity until convulsions and/or prostration develops. Symptoms in humans appear not to be clearly defined. A range of symptoms associated with dysfunction of brain and nerves is anticipated. The mode of action of the pyrethroids is similar to that of the organochlorines, and overlap in symptoms of poisoning between these two classes of agents is to be expected.

Organochlorines

The general symptoms of poisoning are similar for the various compounds in this class. None are diagnostic or specific. Tingling of the lips and tongue, dizziness, irritability and tremors progressing to convulsions have been described in human poisonings with DDT, while immediate onset of convulsions is more usual with aldrin, for example.

Prevention and Treatment:

Wildlife personnel can protect themselves from exposure by common-sense precautions that take into account the highly toxic nature of pesticides. Avoidance of exposure is the key theme. Fields that have been recently sprayed should not be entered for several hours so that vapours or residual air-borne particles will not be inhaled. Protective clothing should then be worn when entering such areas. These should include a chemical-filtering mask, disposable or readily washable boots or foot coverings, complete external covering such as coveralls and substantial water-proof gloves that will the discarded after use. Specimens collected in contaminated areas should be placed in sturdy plastic bags in the field and in a second plastic bag once the specimens have been retrieved from the contaminated area. Labels should include the information that the specimens may be contaminated with a poisonous chemical and the name or class of the chemical suspected. Protective clothing should be removed cautiously to avoid contamination of skin or other objects or materials. All contaminated clothing should be placed in plastic bags until discarded or washed. All exposed skin should be washed and any sites of

any accidental exposure of the skin should also be thoroughly washed with soap and warm water. Protective clothing should not be used twice unless it is properly washed between uses. Wildlife personnel required to use pesticides as part of their work should receive special training in the handling, preparation, use and disposal of the particular pesticide in question. National occupational health and safety regulations require that employees receive such training and no person should agree to handle dangerous chemicals without this mandatory instruction.

Since poisoning by insecticides is a life-threatening and complex toxicological problem, effective treatment requires immediate medical attention and careful medical monitoring of the patient as the various possible treatments are applied. There are no home remedies.

Treatment of poisoning by organophosphate and carbamate insecticides is directed at relieving the symptoms that are amenable to treatment and, for organophosphates only, in re-activating the acetylcholinesterase enzyme which the insecticide has affected. There is no true antidote. Similarly, only symptomatic and supportive treatment are available for poisonings with pyrethroid and organochlorine insecticides.

Key References:

Briggs, S.A. Basic Guide to Pesticides. 1992. Taylor & Francis, Washington, DC.283 pp.

Klaassen, C.D., Amdur, M.O. and Doull, J. 1986. Casarett and Doull's Toxicology. (3rd Ed.). Collier Macmillan Canada, Inc, Toronto. 974 pp.

RODENTICIDES AND OTHER POISONS FOR MAMMALS

Hazardous Chemicals: Rodenticides and other Poisons for Mammals

Routes Of Exposure: Most rodenticides are presented as baits, but the poisonous principles may be sold as concentrated powders or liquids. Cyanide compounds may be dry powders, bait materials or gasses used for fumigation. Explosive devices, sometimes called cyanide guns, have been used to discharge cyanide compounds into the mouths of predatory mammals.

Chemical Names: Poisons used to kill mammals belong to several different chemical classes. Each pesticide has an official common name and is sold in one or more commercial products or formulations, each of which also will have a commercial or trade name. The official common name will appear on the label in the list of ingredients. The major classes and official common names of agents within each class are listed below.

Coumarins (anti-coagulants)

Brodifacoum	Dicoumarol
Bromadiolone	Difenacoum
Coumafuryl	Warfarin

- Poisonous Properties: Coumarins prevent the body from making proteins required to enable the blood to clot. Death results from fatal hemorrhage, which is usually internal and not evident unless the animal is dissected.

- Treatment: Large doses of vitamin K can counteract the effect of the coumarins. This treatment must be administered and monitored by medical personnel.

Vitamin D_3

Cholicalciferol

- Poisonous Properties: Toxic overdoses of vitamin D_3 result in large mineral deposits in many organs and tissues, particularly the kidney. Failure of these organs and tissues leads to death. Death will occur in 24 hours with high doses or may take several days with lower doses.

- There is no effective treatment or antidote

Cyanide

Acrylonitrile	Calcium cyanide	Sodium cyanide
Ammonium thiocyanate	Hydrogen cyanamide	
Calcium cyanamide	Hydrogen cyanide	Trichloroisocyanuric acid

- Poisonous Properties: The active poison in all these is cyanide gas which may be inhaled directly or released from dry material that is ingested. Cyanide prevents tissues from using oxygen and causes whole-body asphyxiation

- There is no treatment or antidote

Fluoroacetate

Compound 1080 (sodium fluoroacetate)
Compound 1081 (fluoroacetamide)

- Poisonous Properties: These compounds block important enzymes of energy metabolism. They affect the whole body but especially the heart and brain. Onset of effects occurs 15 minutes or so after exposure. Death may be delayed 1 to 24 hours from the onset of signs of poisoning, which include anxiety, hyperactivity, convulsions and paralysis of respiratory muscles.

- There is no effective treatment or antidote.

Strychnine

- Poisonous Properties: Strychnine affects the normal control of nerve activity and results in excessive nerve stimulation. Signs progress from restlessness and anxiety to convulsions and death from respiratory failure.

- Effective treatment is sometimes possible by use of muscle relaxants, sedatives, anaesthetics and mechanical respiration until the poison is eliminated by the body. Treatment requires hospitalization and intensive care. There is no antidote.

Key References:

Briggs, S.A. Basic Guide to Pesticides. 1992. Taylor & Francis, Washington, DC.283 pp.

Klaassen, C.D., Amdur, M.O. and Doull, J. 1986. Casarett and Doull's Toxicology. (3rd Ed.). Collier Macmillan Canada, Inc, Toronto. 974 pp.

HAZARDS OF WILDLIFE IMMOBILIZATION

Several hazards may be encountered during the immobilization of free roaming wildlife. For the purpose of this discussion the hazards will be divided into equipment hazards, animal hazards, and drug hazards.

EQUIPMENT HAZARDS

Many remote delivery systems are available on the market for remote delivery of wildlife immobilizing drugs. Equipment used for immobilization of free roaming wildlife often requires a heavy steel dart propelled by a .22 Calibre charge. The range of the dart can be adjusted either by adjusting the power setting on the rifle, or by using a higher powered charge. At close range these darts have considerable penetrating power, and have the potential to produce severe harm or death depending on the location that the dart penetrates. These rifles should be treated with the same respect as any other firearm.
Knowledge of the darting equipment is also essential. Darts must be assembled and loaded properly, and if air or butane charged darts are used they should always be pointed away from the operator during charging, and should only be charged immediately prior to use. Darts should not be stored on the person if at all possible. if darts must be carried close to the body, they should be in a tightly sealed container. Always wear gloves when darts are loaded or cleaned, have plenty of water close by when cleaning darts, and work in pairs.

ANIMAL HAZARDS

Hazards to personnel can occur from inadequately immobilized wildlife. The potent narcotics often produce an excitement phase prior to immobilization, this is particularly pronounced in ruminants. Large ruminants may pose a significant threat during this time period.
With some agents little warning may be given by the animal prior to recovery, and sudden recoveries may occur. This has been noted with xylazine-ketamine and medetomidine-ketamine in bears. Reversal agents may produce extremely rapid recoveries if they are administered by the intravenous route. When working with potentially dangerous animals, one should retreat to a safe place as soon as the reversal agent is administered, and the animal should be monitored from a distance. Lightly anesthetised animals can pose a risk as sudden movement of the animals limb or head could cause injury to people working around the animal. Potentially dangerous animals should always be approached cautiously, and re-darted if the plane of anesthesia appears to be too light. Alpha-2 agonists such as

medetomidine or xylazine should **Never** be used as the sole immobilizing agent in potentially dangerous wildlife. These agents may produce an animal that appears to be deeply sedated or anesthetized, but as soon as the animal is approached it may arouse and become aggressive.

DRUG HAZARDS

Probably the most serious hazard encountered by people involved in wildlife immobilization are the wildlife immobilizing drugs. Some of these drugs are extremely potent and exposure to even a minute volume of some of these drugs could result in death. Accidental darting, could result in general anesthesia in an uncontrolled environment, without even basic support such as oxygen or equipment for ventilation. Anesthetic overdose may occur from drugs that are potentially safe in low dosages. Some of the drugs, such as ketamine, could produce behavoiral changes if accidentally administered at a low dose. Many of the agents would produce respiratory depression and death if they were accidentally administered at the doses required for wildlife immobilization. Several of the agents used for wildlife immobilization are not encountered in human anesthesia, and overdose with these agents may produce confusion among medical personal who have not been acquainted with the pharmacology of these agents. Often these drugs are used in combination, and toxicity will result from the combined effects of the mixture. It is extremely important to have a full knowlege of the potential adverse effects of these agents prior to their use, and to be aware of how exposure to these drugs should be treated. The following section deals with individual drugs, and combinations. The final section will discuss how to avoid toxicity from these agents

CARFENTANIL

- Extremely potent narcotic 10,000 times the potency of morphine.
- Very small volumes are required.
- In several species excitation reactions can be seen on induction, hackney gait and aimless wandering are characteristic
- Underdosing can cause severe exitement reactions.
- Respiratory depression.
- Variable induction time, usually about 8min.
- Long duration of action, usually greater than 3hrs.
- Renarcotization due to enterohepatic recycling.
- Problems with hyperthermia.
- Often combined with xylazine.
- Extreme human safety hazard.

Reversal: Naltrexone; the half life of this drug is about 4 hours, it outlasts carfentanyl making it ideal for reversal. Other reversal agents are to short acting.

ADVANTAGES:
- Small injection volume
- Reliable anesthesia in a wide variety of wildlife species
- Readilly Reversable.

DISADVANTAGES:
- Human safety hazard.
- Abuse potential.
- Animals may regurgitate
- Can cause hypoxemia and hypercarbia.
- Hyperthemia, may lead to capture myopathy.
- Renarcotization

HUMAN SAFETY:
- Extreme human safety hazard, can possibly be absorbed percutaneously, or via mucous membranes. A few drops on the skin, or on a mucous membrane, could lead to severe narcosis. Three milligrams of carfentanil is equivalent to 30 grams of morphine. Accidental darting would probably have a very grave outcome. First aid would include the immediate administration of the reversal agent naloxone. The Naloxone should be administered at 2 mg stat and repeated as needed. AR or CPR may also be required. The victim should be removed as quickly as possible to hospital. The drugs should go with the victim.

ETORPHINE

- Synthetic derivative of thebane.
- Extremely potent opiod 5000 times as potent as morphine.
- onset of action 10-15 min. following I.M. injection.
- Inhibition of respiratory center leads to respiratory acidosis and hypoxia.
- Passive regurgitation can occur.
- Most often used in combination with acepromazine or xylazine to produce neuroleptanalgesia.
- Very dangerous to handle, accidental injection or contact with the eyes can result in profound respiratory depression
- Reversal: Diprenorphine; M50-50 at 2 times the etorphine dose

ADVANTAGES:
- Relatively rapid induction
- Smaller injection volume than some of the other agents
- Reliable anesthesia
- Readilly reversible

DISADVANTAGES:
- Quality of anesthesia is not always the best, animals may be rigid and can move extremities
- Must monitor for signs of respiratory depression, animals may become hypoxic.
- Bradycardia can be extreme, should respond to atropine.
- Recycling can occur.
- Human safety hazard

HUMAN SAFETY: Extreme human safety hazard. Must avoid contact with the skin, eyes, etc. If contact with the skin, flush immediately with cold water. If signs of toxicity develop narcan should be administered (SEE CARFENTANYL OVERDOSE)

KETAMINE

1. Phencyclidine derivative; produces a state of cataleptic anesthesia.
- Mechanism of action may be due to an increasd release of dopamine in the brain or to a change in seratonin metabolism.
- Increased heart rate, arterial blood pressure and cardiac output.
- Pharyngeal and laryngeal reflexes remain somewhat intact.
- Can cause clonic-tonic convulsions in carnivores.
- No specific antagonist available.

ALPHA 2 AGONISTS

- Act at pre and post synaptic alpha 2 receptors in the CNS and PNS.
- Alpha 2 receptors in the CNS are involved in the modulation of sympathetic outflow regulating cardiovascular and endocrine functions, vigilance, emotion, cognition, and pain perception.
- Must be used in combination with other drugs. **Large cats may appear very sedate but may be stimulated to a purposeful aggressive state.** Hoofstock may flee when approached.
- Decrease heart rate, blood pressure and cardiac output.
- decreased resp. rate.
- Impair thermoregulatory ability
- Emetic effect in dogs, cats, and bears
-bloat and regurgitation in ruminants.

SPECIFIC ALPHA 2 AGONISTS

1. XYLAZINE

2. DETOMIDINE: inability to obtund righting reflex
- 4 times more potent than xylazine.

3. METDETOMIDINE: much more selective Alpha 2 agonist.
- 40 times more potent than xylazine.
- Greatly decreases ketamine requirements.
- Readily reversible with Atipamezole.

ALPHA 2 ANTAGONISTS

- Block alpha 2 receptors centrally and peripherally results in reversal of alpha 2 agonistic actions.
- Old drugs such as Yohimbine aare less specific in their actions, block alpha 1 as well as alpha 2 receptors. Some effects on dopaminergic, seratonin, opiod and cholinergic pathways.
- Newer drugs are much more selective for alpha 2 receptors.
- Yohimbine
- Tolazeline
- Idozoxan: Alpha 2/alpha 1 selectivity 170.
- Atipamezole: alpha 2/alpha 1 selectivity 1399.

ADVANTAGES OF ALPHA 2 AGONISTS AND ANTAGONISTS
- Readilly and rapidly reversable, especially with the new highly specific antagonists
- Provide much more sedation than any of the other tranquilizers/sedatives
- Occasionally can be used by themselves

DISADVANTAGES
- Should not be used as the sole agent, unreliable, animals may appear sedate but may become aroused.
- Hemodynamic instability. When used alone or in combination with opiods can see bradycardia and hypotension. Used in combination with ketamine these effects are not so bad.

HUMAN SAFETY: injection of these drugs would probably result in severe bradycardia and hypotension, or hypertension with high doses of potent agonists. Coma and cardiovascular collapse would probably occur with high doses. Treatment would consist of airway management, supportive treatment for shock, If a specific antagonist is available its administration may be considered by trained medical personnel.

KETAMINE-XYLAZINE

- Xylazine is commonly used in combination with ketamine to decrease the convulsive effects of ketamine and produce general anesthesia. This combination has been used in a wide variety of species, and is particularly useful in carnivores. Xylazine decreases sympathetic outflow in the CNS, resulting in hypotension, and bradycardia. Ketamine is a sympathetic stimulant,and tends to counter these effects. Hemodynamic and respiratory perameters are relatively stable with this combination. Animals immobilized with this combination tend to maintain some degree of airway protective reflexes. Immobilization usually occurs 10-15 min. post injection.

ADVANTAGES:
- Hemodynamic and respiratory stability
- Good quality of anesthesia
- Maintain laryngeal reflexes.
- Handler safety

DISADVANTAGES:
- Large injection volume
- Not fully reversible
- Occaisionally see sudden recoveries, particularly in bears.

HUMAN SAFETY:
- relatively safe to handle, Ketamine used for IM induction in people at 6.5-13mg/kg. Biggest danger would come from the xylazine. It is not used in people and its effects are unknown. Treatment: maintain airway and ventilation, remove to a hospital for treatment.

MEDETOMIDINE-KETAMINE

-Medetomidine is a new alpha 2 agonist with a potency about 40 times that of xylazine.
- Medetomidine is very selective for the alpha 2 receptor, administration produces analgesia, sedation, and at high doses medetomidine is reported to produce anesthesia.
- Side effects include bradycardia, hypotension, and hypoventilation.
- Ketamine should be included in combination with medetomidine. Ketamine tends to increase the heart rate and counter some of the hypotension produced by medetomidine.
- Low doses of ketamine are required allowing a good recovery following reversal of the alpha 2 agonist with atipamazole
- A very low dose of medetomidine is required, approximately 80 μg/kg in many ruminants. This can be combined with powdered ketamine to produce a small injection volume.

ADVANTAGES
- Small injection volume.
- Reliable anesthesia.
- Can be used in a wide variety of species.
- Less abuse potential than the potent narcotics.
- Hemodynamic and respiratory stability.
- Handler safety.
- Some maintenance of airway protective reflexes.

DISADVANTAGES
- Not yet available in North America.
- Few studies have been performed on the hemodynamics and respiratory stability in wildlife

HUMAN SAFETY
- Low doses of medetomidine have been used in human medicine, Ketamine is used in human anesthesia. Low dose exposure percutaneously would probably be harmless if the drug was washed off. Large dose parenterally would probably produce severe hypotension and respiratory depression. The victim should be treated for shock and AR or CPR administered immediately. The victim should be removed to hospital immediately. The drugs and a description of the drugs should be included.

TELAZOL

- Marketed as a combination of tilletamine (a drug similar to ketamine) plus the benzodiazepine drug Zolazepam. Onset of activity 5-10 min. following IM injection. Used in a wide variety of species.

ADVANTAGES
- longer duration of action than ketamine- xylazine, smoother more predictable recoveries.
-Smaller injection volume than xylazine/ketamine.
-Relatively safe to handle
-Hemodynamic and respiratory stability
-Maintain laryngeal reflexes

DISADVANTAGES
-No antagonist available.
-Prolonged recovery
-Can see some convulsive activity in canids.

HUMAN SAFETY: These drugs are not liscenced for use in humans, the benzodiazepines and the phencyclidine derivatives are both relatively safe and have a high margin of safety. Accidental injection would probably produce anesthesia. Supportive care, management of airway, ventilation if required, removal to a hospital.

.OTHER AGENTS USED IN RESTRAINT

1. Innovar Vet: May be used in small carnivores and ruminants, Innovar has been used in humans, exposure to low doses would produce a condition known as neuroleptanalgesia. The condition is similar to light anesthesia.

2. Acepromazine. useful in combination with narcotics to produce neuroleptanalgesia. In the past it was commonly combined with etorphine, now it is largely replaced with xylazine. Acepromazine is a relatively innocuous drug. Adult humans have survived doses of chlorpromazine up to 10 grams. Administration may result in behavoiral toxicity. Overdosage may result in extra pyramidal side effects (similar to convulsions)

3. Azaparone. very similar to acepromazine, butyrophenones and phenothiazines are used to treat psychosis and aggressive behavoir in people.

4. Butorphanol. A kappa agonist mu antagonist. May be useful in combination with xylazine to produce neuroleptanalgesia. Relatively safe drug to use. Human dose is 2-3 milligrams. Severe overdose in people would require high doses of antagonist. People overdosed with this drug should be removed to a hospital as quickly as possible. Narcotic antagonists and AR should be administered if signs of respiratory depression are apparent

PRECAUTIONS TO USE WHEN DEALING WITH IMMOBILIZING AGENTS

The following is a list of precautions that should be followed when these drugs are used. The list is adapted from similar suggestions advised by Dr. Jerry Haigh who has had extensive experience working with these agents.

1) Prevention:

Avoid contact with any of these agents, paticularly the potent narcotics. Skin contamination can be avoided by wearing disposable gloves. Avoid contact with eyes, nose, and mouth, as mucous membrane exposure will result in a rapid uptake of the drug.

- Work carefully with darts, and needles, have large volumes of water available when loading or cleaning darts, dispose of needles and syringes in sharps containers labled with a warning to avoid exposure to contents.

- Never work alone.

2) Prepare for the worst scenario and be informed.

- Meet with medical personnel in the are you are working in. Advise them of the drugs that you are using, the doses of the drugs, and have a monograph available for each drug that is used.

- Members of the immobilizing team should be well versed in first aid, and should have a knowlege of artificial respiration, and CPR.

- First aid for drug exposure, and evacuation procedures for an affected individual should be discussed prior to going in the field. Everyone working around the drugs should have a knowlege of the potential hazards of the drugs.

- A first aid protocol should be present in the drug box.

First Aid & Antidote:

- If an antidote is available it should be drawn up and ready to administer, sufficient antidote should be available.

- Administer the antidote immediately. Narcan should be administered at a dose of 2 mg initially and repeated to effect. It may require 6 - 12 mg of narcan (1.5-3 bottles) to reverse the effects of one drop of carfentanil. Antidote should be repeated as required.

- Carry out artificial respiration, or CPR as required.

- If medical facilities &/or ambulance/paramedics are available they should be contacted immediately and the victim should be evacuated to a medical facility.

- In remote locations immediate evacuation should be considered to the closest medical facility.

- The effects of high dose narcotics &/or high dose alpha 2 agonists may be irreversible, & AR, CPR, & mangement of shock may be the only hope for survival.