Ministry of Higher Education and Scientific Research
University of Baghdad
College of Science
Department of Biology

Disease Ecology
2020-2021

الفصل الدراسي الثاني

تدريسي المادة :

أ.م.د.حارث سعيد جعفر الورد

Disease Ecology

Course for Master Students

Department of Biology-Zoology

Dr. Harith Saeed AL-Warid
Assistant professor
Department of Biology
College of Science
University of Baghdad

Introduction

The study of disease ecology or diseases in wild animals are relatively new scientific disciplines when compared to the study of disease in humans or domestic animals. During the first half of the twentieth century a small number of scientists began pioneering studies of diseases such as tularemia and plague in wild rodents, (avian botulism in waterfowl, and rinderpest in African antelope.

Wildlife Disease Association, in 1951 marked the beginning of more organized study of disease in wild animals or the Disease Ecology, but most of the people involved in the early years of that organization would have identified themselves as members of some other discipline, such as virology, toxicology, parasitology, ecology, and pathology, who worked with wild animals rather than as wildlife disease specialists. During the past two decades, there has been a huge increase in interest in the subject. Scientists from a wide spectrum of disciplines including conservation biology, wildlife management, veterinary medicine, agriculture, public health, theoretical ecology, toxicology, animal behavior, and human medicine have become interested, on an unprecedented scale, in the particulars of disease in wild animals.

There are several reasons for this sudden increase in interest and involvement.

1- New human diseases continue to be discovered.

2- Public health officials and physicians have been forced to deal with wild animals by the discovery that most of the emerging infectious diseases of humans are diseases that are shared with animals (zoonoses), and that wild animals have a central role in many of these conditions .

Table 1.1 Emerging Diseases of Humans in Which Wild Animals Are Important

Disease in humans	Causative agent	Wild species involved
Viruses		
Hantavirus pulmonary syndrome	Sin Nombre virus and many other New World hantaviruses	Rodents
Hemorrhagic fever with renal syndrome	Puumala virus and other Old World hantaviruses	Rodents
West Nile fever	West Nile virus	Birds
Hemorrhagic fevers (Argentinean, Bolivian, Brazilian, Venezuelan)	Arenaviruses	Rodents
Australian bat lyssavirus infection	Lyssavirus similar to rabies virus	Bats
Bacteria		
Human granulocytic ehrlichiosis	*Ehrlichia phagocytophila*	Rodents, cottontail rabbits
Monocytic ehrlichiosis	*Ehrlichia chaffeensis*	White-tailed deer
Lyme disease	*Borrelia burgdorferi*	Rodents, birds, deer
Cardiopathy, endocarditis	*Bartonella* spp.	Rodents
Cestodes (tapeworms)		
Alveolar echinococcosis	*Echinococcus multilocularis*	Fox, rodents
Nematodes (roundworms)		
Visceral larva migrans	*Baylisascaris procyonis*	Raccoons

Important human diseases including severe acute respiratory syndrome (SARS), Ebola disease, and Marburg virus infection, are believed to originate in wildlife, although the specific wild animal has not been identified to date.

3- Many well-established human diseases that continue to cause problems including plague, tularemia, Lassa fever, rabies, and influenza are linked directly to wild animals.

4- Veterinarians and agriculturists also have developed a great interest in wild animals, because of the involvement of free-ranging animals in many diseases of domestic animals (table 1.2).

Table 1.2 Diseases of Domestic Animals in Which Wild Animals Are a Source of Infection

Disease	Domestic animal(s)	Wild animal(s)
Viral		
Hendra virus infection[1]	Horse	Fruit bats
Nipah virus infection	Pig	Fruit bats
Louping ill	Sheep	Red grouse, mountain hare
Malignant catarrhal fever	Cattle	Wildebeest
Foot-and-mouth disease	Cattle, sheep, pigs	African buffalo
Classical swine fever	Pigs	Wild boar
Newcastle disease	Poultry	Cormorants, other birds
Avian influenza	Poultry	Wild waterbirds
Bacterial		
Bovine tuberculosis	Cattle, deer	Badger, brushtail possum, white-tailed deer, elk, bison
Brucellosis	Cattle	Bison, elk
Anaplasmosis	Cattle, sheep and goats	Wild ruminants
Leptospirosis	Cattle, pigs, dogs	Different forms of *Leptospira* occur in a number of wild hosts
Protozoa and helminths		
Theileriosis	Cattle	African buffalo, eland
Cytauxzoonosis	Domestic cat	Bobcat
Hydatid disease (*Echinococcus granulosus*)[1]	Horse, sheep	Fox, dingo, macropods
Liver fluke (*Fascioloides magna*)	Cattle, sheep	White-tailed deer, elk
Meningeal worm (*Parelaphostrongylus tenuis*)	Llama, sheep, goat	White-tailed deer

[1]May also affect humans.

Some of the associations between wild animals and diseases of domestic animals have been known for many years, but, in other instances, the role of wild animals in the disease did not become apparent until there was effective control of the disease in domestic animals. As an example, rabies in much of North America was thought of as a disease for which the domestic dog was the principal animal host; however, when rabies in dogs was

controlled by vaccination, it became obvious that the disease was not going to disappear, because it was still cycling in wild carnivores and bats. As the disease was studied further, it was discovered that there was not one rabies virus, as had been thought, but many strains, each circulating in one principal wild species. Thus, in North America, different strains of rabies virus occur in skunks, foxes, and raccoons as well as several strains in bats. Some diseases that have been eliminated from domestic animals continue to occur in wildlife. For instance Newcastle disease in double-crested cormorants is considered a risk to North American poultry from which the disease has been eliminated.

5- Conservation biologists have become increasingly concerned about disease because of recognition that disease may play an important role in the survival of threatened or endangered species. Disease may limit captive breeding and release programs, and have devastating effects on small populations. Examples include the impact of avian malaria and poxvirus on indigenous Hawaiian birds .

Wildlife managers have been forced to become more involved with disease for several reasons. There has been an explosion of academic attention to various aspects of disease in wild animals by ecologists, behaviorists, population biologists, Microbiologist, Zoologist and public health specialist. Many of these scientists are interested in aspects of the coevolution of disease agents and animals, and the theoretical rather than the pragmatic features of disease. Their work is providing a theoretical framework for understanding host-parasite evolution, virulence, and the population effects of disease.

IS DISEASE IN WILD ANIMALS TRULY BECOMING MORE IMPORTANT?

All of the attention outlined above suggests that disease in wild animals is becoming more important or significant. One should ask if this is because there really is more disease or if disease has just become more apparent because more people are looking for it. Some of the apparent increase in disease is a result of greater surveillance. The hantaviruses provide a good example of this phenomenon. The discovery that one hantavirus, Sin Nombre virus from deer mice, caused fatal hantavirus pulmonary syndrome (HPS) in humans in a small focus in the United States led to a huge search for similar viruses. In less than a decade, more than 25 different hantaviruses, each with its own specific rodent host, have been identified in North, Central, and South America. Many of these viruses have been linked to human disease. There is no evidence that these are truly new entities. The viruses have been present but unrecognized in rodents, and the human disease, which also has been present, has now been given a name (HPS) and its cause has been identified. Some diseases do appear to have become more common or prevalent. West Nile virus is a new transplant to the New World that has spread rapidly with major consequences for wild birds, horses, and humans. It is safe to predict that disease in its many manifestations will become even more significant for wild animals in the future and that there will be increasing pressure on wildlife biologists to "manage" disease. New emerging diseases of humans will continue to be linked to wild animals as pressure from the expanding human population brings humans and wild animals into ever closer contact. The rapid movement of humans means that an individual exposed

to an infection in a wild animal in a remote part of the world can be in the middle of a city on another continent before the disease becomes apparent. The rapid and extensive movement of exotic animals for the pet, zoo, and game farm industries means that infected individuals can associate with humans and traditional domestic animals in unexpected ways, as occurred in the introduction of monkey pox to the United States in 2003 (CDC 2003). New diseases of domestic animals also will emerge that will be linked to wild animals. For instance, within the past decade three new viral diseases (Hendra virus that infects horses and humans, Nipah virus that infects pigs and humans, and Menangle virus that infects domestic pigs) have been discovered in fruit bats. Continued pressure on land for agriculture and urban development will intensify contact and exchange of disease between domestic animals and the wild animals that live in residual areas of natural habitat. The concern about some diseases such as bovine tuberculosis is that wild animals are a source of infection for domestic animals. There is also concern for transmission of disease from domestic to wild animals, as has occurred with transmission of canine distemper virus from dogs to the Ethiopian wolf. The interrelationships among wild animals, domestic animals, and humans may be complex. In some situations, domestic animals may be an intermediary by which diseases from wild animals reach humans. This occurred in Malaysia in 1999. A previously unrecognized virus of wild fruit bats became established in domestic pigs by some unknown route. There was no evidence of transmission from bats to humans, or of human to human spread, but 265 humans developed encephalitis (inflammation of the brain) in the outbreak.

Of the affected people, 93% had worked with pigs and 105 died of so-called Nipah disease (WHO 2001). Approximately 900,000 pigs were killed to control the disease.

Influenza presents a potentially even more dangerous situation. Wild water birds carry an array of all known subtypes of influenza A virus and shed the virus in their droppings. The virus can survive in surface water for an extended period. Influenza viruses recombine readily to form novel viruses and the great waves of human influenza that sweep around the world (pandemics) result from formation of a new strain. Although influenza strains from birds can infect humans directly, as occurred in Hong Kong in 1997 and is occurring in several Asian countries, the pandemic strains that affected humans around the world in the 20th century resulted from assortment that occurred in pigs infected with both a strain from humans and a strain from birds . Thus, pigs served as an intermediary between birds and humans. The discussion above dealt with infectious diseases that are shared by people, domestic animals and wild animals.

STUDY OF WILDLIFE DISEASES: AN INTERFACE AREA

The study of wildlife diseases is an interface area that can be approached from many different perspectives. The great diversity of interest in diseases of wild animals is healthy because disease usually is complex and beyond the expertise of any one discipline. Physicians, veterinarians, and public health specialists are familiar with the medical aspects

of disease but often have little understanding of the ecology or natural history of wild animals, or experience in thinking about disease as a natural component of ecosystems.

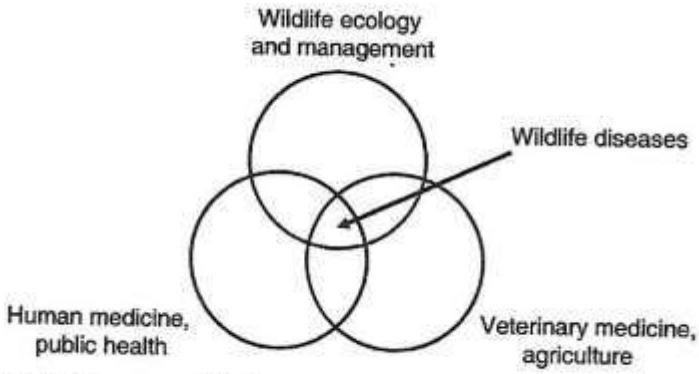


Fig. 1.1. Disease in wild animals occurs at the interface among human medicine, veterinary medicine, and ecology.

Theoretical ecologists, mathematicians, and population biologists can model how disease should behave quantitatively within populations but may have little experience with the medical aspects (physiology, anatomy, immunology, pathology) of disease, or with the practicalities of wildlife management. Unfortunately, disease has not been part of most training programs in biology and ecology so that individuals from that background have little knowledge of the range of factors that cause disease, the effects of disease agents on individual animals, or how disease agents move through populations and persist in the environment. Physicians, veterinarians, and public health specialists are familiar with the medical aspects of disease but often have little understanding of the ecology or natural history of wild animals, or experience in thinking about disease as a natural component of ecosystems.

SUMMARY

1. The study of disease in wild animals is a recent phenomenon.
2. There has been a great increase in effort in this discipline because of the recognition of the involvement of wild animals in diseases of humans and domestic animals, the impact of disease on wildlife management and conservation biology, the recognition of new forms of environmental contamination, and the academic interest in disease as an ecological factor.
3. Disease in wild animals will become even more important because of environmental, agricultural, and demographic changes as a result of growing human populations that will increase contact between wild animals, humans, and domestic animals and that will further degrade natural habitats.
4. The study of disease in wild animals must be multidisciplinary because of its complexity.
5. Wild animals are affected by a range of infectious and noninfectious factors that occur together and that interact. To consider only one or the other type of cause is to understand only part of the picture.
6. Disease is an ecological entity that should be considered in terms of life history theory and that is intimately intertwined with resources and trade-offs.

Disease Ecology

What Is Disease?

The word “disease” is used so commonly in everyday conversation that each person has his or her own understanding of its meaning. That understanding is highly variable depending upon one’s particular perspective. It is difficult to find a definition for a condition such as botulism that may kill 500,000 waterfowl on a single lake. It also is difficult to find a definition that is specific enough to clearly separate disease from conditions that we usually do not think of as disease. For instance, if a snowshoe hare dies as a result of severe intestinal damage caused by parasitic worms most people would consider this to be an example of disease. If another hare were killed by a great horned owl, we generally would consider this to be an example of predation rather than of disease. However, in each of these situations, another species extracted nutrients for its own use from the hare and in doing so caused its death. This makes the dividing line between predation and disease seem a bit hazy. The difference seems to be that the owl acted without accomplices and did the job quickly, while many worms were involved, each taking just a bit from the hare, and they did so over a period of time. We can extend this example a little further and assume that both hares had worms but in the second hare the intestinal injury caused by the worms was not so severe that the hare died because of the worms. (This would represent the more normal “parasitic" situation.) However, the worms are extracting nutrients from the hare by feeding on intestinal cells and on blood from shallow wounds in the intestinal lining, and the hare is responding by

producing inflammatory cells and antibodies to defend itself against the worm and by trying to repair the injury. Thus, the worms represent a cost to the hare. The second hare may have been able to compensate for this cost by eating more to provide both for its own increased needs (for resistance and repair) and for the needs of its uninvited lodgers, but, in doing so. it may have to spend an extra hour each day foraging for food. Hares that are moving are more vulnerable to predators than hares that are sitting motionless and hidden, and hares that are nutritionally stressed may be able to allocate less resources to antipredator behavior than well-fed hares. If the hare was killed by an owl during the extra hour of activity, should we attribute its death to disease (parasitism) or to predation? One could argue that the basic or underlying cause of death was parasite induced injury (disease) that made the bare more vulnerable to the owl and that predation was only the proximate cause of death. We might extend this example and hypothesize that heavily parasitized individuals within the hare population are more susceptible to predation than un-parasitized hares and that parasitism could be an important component in the ecology of hares and of their evolution.

It is preferred to use the following definition "***any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects; or combinations of these factors***" (Wobeser I981). Implicit within this definition are three concepts;

1- ***Disease is measured in terms of impairment of function rather than by the death of individuals.*** This distinction is important because death often has been the endpoint

used to evaluate disease in wild animals but not all dysfunctions lead to death. If we return to the hare-worm example, the location of a hare on this continuum depends upon many factors including : the number of worms present, the type of worms and their ability to cause damage, the hare's ability to resist and repair injury, and environmental factors that determine the quality and availability of food. Hares with only a few worms, of a type that causes only mild injury, will be in a high degree of relative health, particularly in a year when food is abundant. Those hares with many parasites that damage the intestine extensively may have severely compromised function, and some might die as a result, particularly in years when food is hard to find.

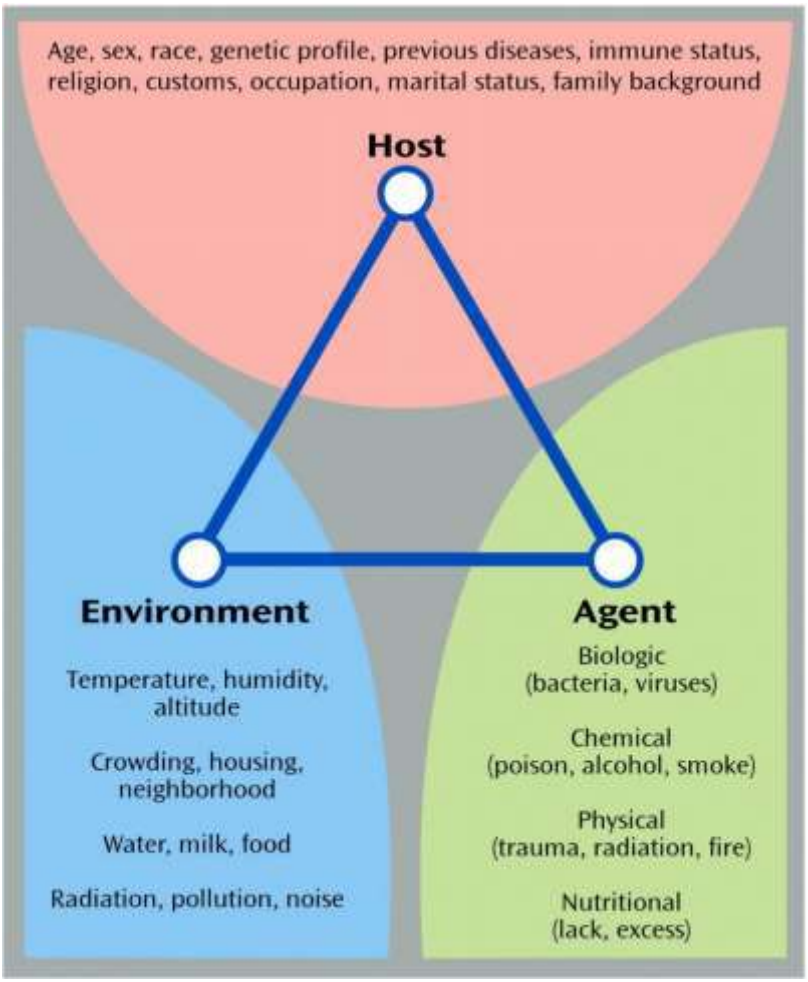
2- ***Factors that cause disease may be either intrinsic. such as an inherited defect in an animal's vascular plumbing or degenerative changes associated with aging, or extrinsic, such as a virus. bacterium, or contaminant that enters its body and causes injury.***

3-***Disease may result from factors acting alone or in combination.***

THE AGENT:HOST:ENVIRONMENT MODEL FOR DISEASE

When disease is being considered, it is tempting to simplify it to a basic equation that resembles a chemical reaction: ***causative agent + animal = disease***. If we combine the cause and the animal in this linear model, the expected result will be a dysfunction that we can label as disease. However, even in diseases in which there is only one cause and in which the disease can be reproduced by exposing the animal experimentally to that factor, the actual

occurrence of the disease in the individual animal in nature and within a population is far more complex than this simple equation suggests. The relationship between agent and animal is influenced by a great variety of factors. Some of these are features of the agent, some are features of the animal, and some are not directly related to either. The latter are taken together as environmental factors. This led to the concept that disease is the result of an interactive relationship among causative agent, animal, and environmental factors. This relationship is sometimes called the epidemiological triangle.



IDENTIFYING THE CAUSE OF A DISEASE

Some diseases are well known for years before the cause is identified. For example, at the present time a disease called avian vacuolar myelinopathy (AVM) has been described in bald eagles and a number of other, but its cause is unknown. Some diseases may be prevented or controlled effectively without the cause being known. A classic example comes from human medicine. In 1854, Dr. John Snow recognized that cholera was associated with drinking water from a particular well in London. His recommendation that the pump handle be removed from the well, so that the water could not be used, controlled the disease more than four decades before its bacterial cause was discovered.

While cause seems a perfectly adequate word, another word, "***etiology***" (from the is used commonly when discussing disease. One definition for etiology is synonymous with cause ("***the cause or origin of a disease or disorder***") but a second definition ("***the study of the factors that cause disease and the method of their introduction to the host***") deals to some degree with why and how a disease occurs as well as with what caused it. New conditions often are recognized because of some peculiar clinical signs or occurrence of the condition under specific circumstances. (The term "**clinical sign**" is used to describe objective evidence of disease, while "symptom" is usually defined as 'subjective evidence, such as the sensation that a patient may experience and relate. Symptom generally is not used in describing disease in animals.)

Various criteria have been proposed to test whether or not an agent is the actual cause of a disease. Often this involves exposing healthy animals experimentally to the putative cause. If the exposed animals develop disease that fits the case definition, causation is considered to be proven. Following the discovery that microscopic agents could produce disease, a set of rules **(Koch's postulates) was developed for establishing the relationship between a cause and a disease. The rules are:**

l. The agent must be present in every case of disease. This is usually done by "isolating" the agent in pure culture.

2. The agent must not be found in cases of other diseases.

3. The agent must reproduce the disease when introduced into experimental animals.

4. The agent must be recoverable from experimentally infected animals. These rules are useful for defining a cause: effect relationship in diseases that involve only a single cause and in which that agent is both a necessary and sufficient cause of the disease, that is, the agent is required for the disease to occur, and the agent acting by itself results in disease. The criteria served Robert Koch well for identifying the cause of some important diseases, including anthrax and tuberculosis, because the causative bacteria could be isolated from affected animals and transmitted to experimental animals, resulting in disease. Koch's postulates have been modified to examine the cause of some noninfectious conditions and experimental exposure has been used for investigating cause: effect relationships related to

many poisons and toxins. Production of appropriate disease in healthy animals exposed experimentally to a chemical is strong evidence of a causal relationship.

Koch‘s postulates are not adequate for understanding causation of many diseases of wild animals because:

(a) relatively few agents are both necessary and sufficient to produce disease over a wide range of environmental conditions.

(b) a single disease may require the interaction of several causes.

(c) a single disease may be caused by several different factors.

(d) a single agent may cause several different diseases.

A disease may have a primary cause (the principal factor contributing to the production of disease), secondary causes (that are supplemental to the primary cause), and predisposing causes (that render the animal more susceptible to a specific disease without actually causing it). The etiology of a disease might include all these factors. Changes in the agent, the animal, or other features of the environment might influence whether disease does or does not occur. The etiology of some diseases, such as the so-called lungworm-pneumonia complex of mountain sheep, involves a web of causation that may include viruses, different bacteria, lungworms, nutrition, and multiple stressors. Different methods for establishing causation are needed for diseases caused by two or more factors acting together, diseases caused by agents that may cause disease under some circumstances and not others.

Because of difficulty in fulfilling the requirements of Koch’s postulates, other criteria have been developed that incorporate epidemiologic information (the way that

diseases occur and are distributed in populations) together with information from experimental exposure to examine cause: effect relationships. Evans (1977) proposed a set of criteria related specifically to viruses, which Kelsey et al. (1996) used to develop the criteria shown below. These include some elements listed by Hill (1965) for enhancing belief in association of noninfectious disease with chemical exposure.

Criteria Used to Test Hypotheses Related to Disease Causation

1- The putative cause should be distributed in the population in the same manner as the disease.

2- The frequency of disease should be higher in those exposed to the putative cause than in those not so exposed.

3- Exposure to the putative cause should be more frequent among those with the disease than those without the disease, when all other risk factors are held constant.

4- Temporally, the disease should follow exposure to the putative cause.

5- The greater the dose or length of exposure to the putative cause, the greater the likelihood of occurrence of the disease.

6- For some diseases, a spectrum of host responses should follow exposure to the putative cause along a logical biological gradient from mild to severe.

7- The association between the cause and the disease should be found in various populations when different methods of study are used.

8- Other explanations for the association should be ruled out.

9- Elimination or modification of the cause or of the vector carrying it should decrease or eliminate the disease.

10- Prevention or modification of the host's response on exposure to the cause should decrease or eliminate the disease.

11-Where possible, in experimental settings, the disease should occur more frequently in animals exposed appropriately to the putative agent than in animals not exposed.

12-All of the relationships should make biologic and epidemiologic sense.

CAUSES OF DISEASE

The usual system for classifying disease agents has been to divide them into three groups: **Infectious diseases, Noninfectious diseases and Noninfectious transmissible diseases.**

I- INFECTIOUS DISEASES

Infectious diseases are caused by living organisms that cause harm while residing in or on an animal's body. These living agents replicate and are involved in a trophic relationship with the animal. The term "**infection**" *implies penetration and growth of organisms within an animal's body.* While the term "**infest**" and "**infestation**" *are used to described colonization of the outer surface of the body by larger organisms such as fleas and lice*, while colonization of the body surface by bacteria and fungi is described as an infection.. However, infestations are still infectious disease.

Infectious disease involves a **trophic relationship** between the causative organism and the animal, in which the organism derives benefit from the relationship while the animal is harmed in some way. This type of relationship is considered to represent **parasitism** and usually involves the agent obtaining nutrients and other biological necessities from the host animal while reducing the host's fitness. Parasitism is not a rare phenomenon. Some authors suggest that the majority of living organisms are parasitic at some time in their life. Every wild animal is host to many parasites. The list of infectious diseases continues to expand, and

many conditions that currently are considered noninfectious or of unknown etiology will be discovered to be infectious. Infectious agents generally are considered to include viruses, bacteria, fungi, certain algae, protozoa, helminthes (worms), and arthropods. Ecologists have attempted to divide infectious causes of disease into two groups (**microparasites and macroparasites**) based on biological and population features rather than on taxonomy. The general features of these two groups are shown below:

Table 3.3 General Features of Microparasites and Macroparasites

Feature	Microparasites	Macroparasites
Size	Small	Large
Generation time	Short	Long
Reproduction	High rate of reproduction within the host	Usually low or no direct reproduction within the host
Duration of infection	Short relative to life-span of host	Persistent, with reinfection being common
Type of disease	Short-lived, often severe	Chronic, usually sublethal, intensity of infection determines degree of injury
Immunity to reinfection	Long-lasting, reinfection uncommon	Depends upon continued presence of infection, reinfection common

In very general terms, most viruses and bacteria, and some protozoa, fit within the **microparasite group**, while most helminths and arthropods fit within the **macroparasite** group. This division is useful for thinking about how various disease agents act in populations, but some agents such as *Mycobacterium bovis,* the cause of bovine tuberculosis, don't fit particularly well. This organism is small and generation time may be relatively short, but the organism reproduces slowly within the host and infections are chronic and persistent.

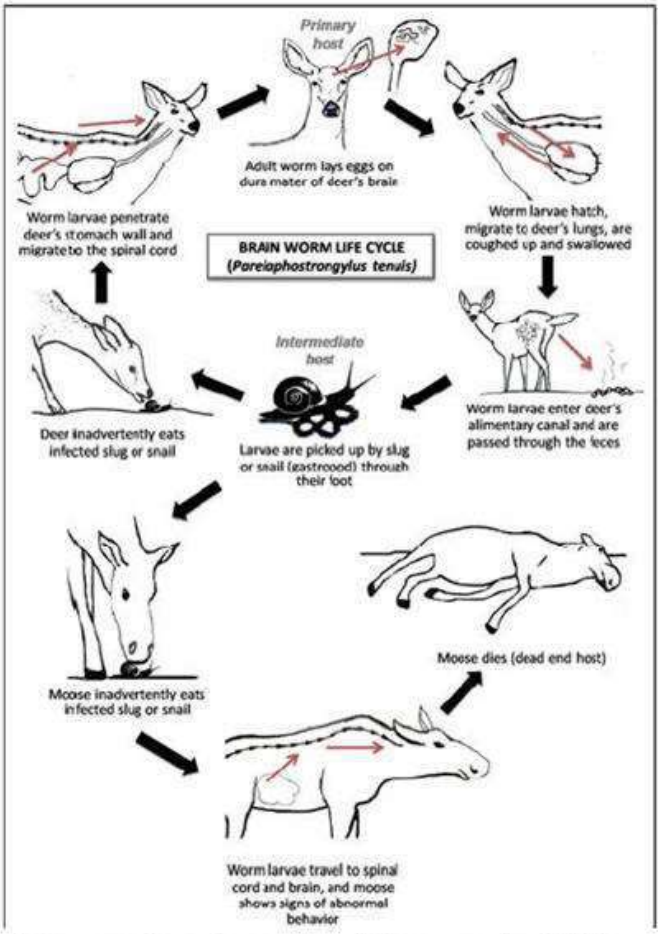
It has become clear that animals with infectious diseases often are infected with more than one genotype of the same agent. Thus, while we classify all of the bacteria causing a case of avian cholera in a snow goose as belonging to the species *Pasteurella multocida* these bacteria may represent a population of different genotypes that are competing with each other for resources from their habitat (the sick goose) in exactly the same way that individual geese compete with each other for resources from a wet land.

Infection, Infectious, and Disease: it is important to differentiate between the presence of an agent that has the ability to produce disease in or on an animal (**infection**) and the occurrence of dysfunction caused by the presence of that organism (**disease**). Many animals are infected without being obviously diseased. The point that growth of an infectious agent in or on an animal has a cost to that animal, because the agent is extracting nutrients from the animal. However, the cost may be so slight that the animal is easily able to compensate for the cost and suffers no detectable dysfunction. An agent may cause infection without disease in one species and severe dysfunction in another species. An agent also may occur as a clinically silent infection in some individuals and cause disease in others of the same species, or an individual may be infected without disease under some circumstances and become severely diseased under other conditions. One of the most basic factors that can influence whether an animal suffers dysfunction as a result of infection is the **intensity of the infection** (***the number of organisms per infected host***). Ebert *et al*. (2000) observed that " the more parasites infect a host individual, the stronger are the parasite-induced effects." The relationship between intensity of infection and the degree of dysfunction that results is

well recognized to occur in infections caused by macro parasites such as various worms and some protozoa. Individuals with few parasites are less likely to be diseased than those with many parasites. There is less information available as to whether this same principle also occurs in infections caused by microparasites. The general belief has been that, so long as the initial dose of microparasitic organisms entering an animal is sufficient to establish an infection, the organisms will give rise to a huge population that will continue to expand until they reach a certain carrying capacity or are limited by the animal's immune system .

It is important to distinguish between animals that are **infected** and those that are **infectious** to others. Not all infectious diseases are **contagious**, that is, "**capable of being transmitted from one individual to another**". Tetanus and listeriosis (bacterial diseases) and aspergillosis and histoplasmosis (fungal diseases) are caused by microorganisms that reside in the soil and that may infect animals but that do not spread from animal to animal. These agents are **infectious (capable of causing infection)** but not contagious. Even among disease agents that are contagious, not every infected individual is capable of transmitting the infection to other animals. Some infected individuals may be in the early stages of infection before transmission can occur. For example, animals are usually infected with rabies virus for 1-3 months, but transmission "only effectively occurs during a relatively short excretion period of the virus during the final stage of the disease" . The interval between infection and when infective stages are shed by worm parasites is referred to as the *prepatent period*. For instance, white-tailed deer are infected with the meningeal worm *Parelaphostrongylus tenuis* for 82 91 days before they begin to shed infective larvae in their

feces. During the 3-month prepatent period, they are infected but not infectious. Some infections enter a **latent stage** in which they are hidden, do not cause disease, and are not contagious. This is a feature of many herpesviruses, herpes hominis that causes cold sores in humans.



Brain worm life cycle; courtesy of dec.ny.gov by Natalie Sacco

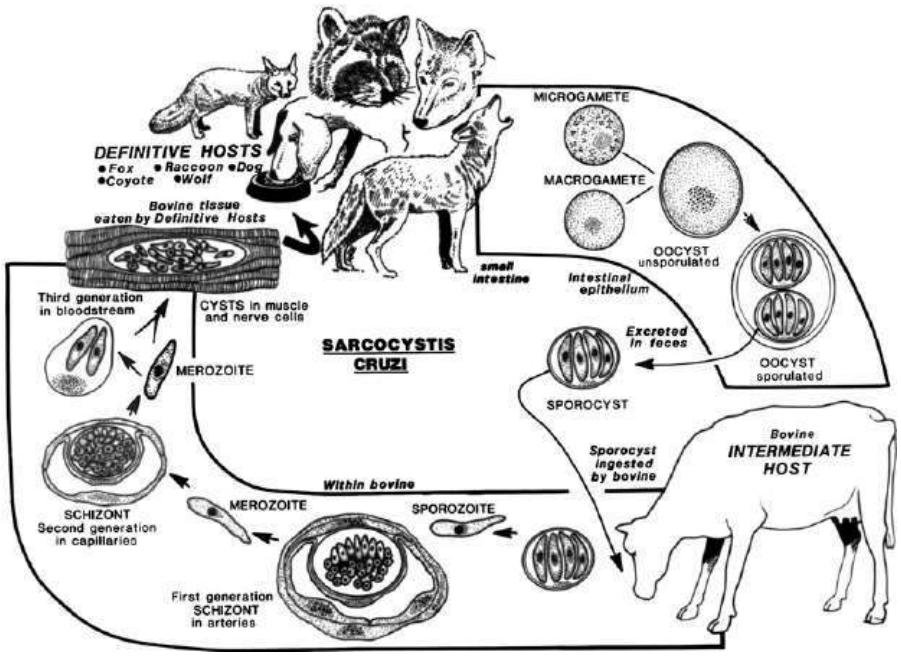
Infectious agents

- **Viruses:** "*Viruses occupy a unique position in biology. Although they possess some of the properties of living systems such as having a genome, they are actually nonliving infectious entities and should not be considered microorganisms*". Despite this assertion that they are nonliving, virus generally are considered to be infectious agents. Viruses are too small to be seen by light microscopy and contain only one type of nucleic acid, either DNA or RNA, never both. All viruses are obligate parasites. They have no functional organelles and are totally dependent upon the host cell for energy production and synthesis of macromolecules.

- **Bacteria:** Bacteria are small (0.5 to 5 pm long), single-celled organisms with considerable morphologic diversity. Most have a rigid cell wall and multiply by binary fission. Many bacteria can be grown on inert artificial media, although two groups (rickettsia and chlamydiae) only grow in living cells. Bacteria are large enough to be seen with the light microscope and most bacteria can be separated into one of two groups based on the way that they stain when exposed to a particular stain. Bacteria that stain blue with this stain, such as staphylococci and streptococci, are said to be gram positive; those that stain pink, such as Esrherichia coli and salmonellae, are said to be gram-negative.

- **Fungi:** Fungi are eukaryotes with a distinct nucleus, cell and nuclear membranes, Golgi apparatus, mitochondria, and cytoskeleton.

- **Protozoa:** The protozoa are a large, diverse group of single celled eukaryotic organisms, many of which have a complex life cycle. It is estimated that there may be 50,000 species, of which about 20% are parasitic in vertebrates and invertebrates. Their classification is confusing, and molecular biology is leading to major changes. From a practical perspective, it is easier to discuss these parasites based on the part of the animal's body that is infected rather than on a taxonomic basis. Some protozoa that require more than one host species to complete their life cycle occupy different niches in their different hosts. For example, parasites of the genera *Sarcocystis* and *Toxoplasma*, require two different host animals (a predator and one or more prey species) to complete their life cycle. They inhabit the intestine of the predator and other body tissues of the prey host (which is usually a herbivore). Most protozoa behave like microparasites in that they are small, have a short generation time, have high rates of reproduction in the host, and tend to produce immunity to reinfection in hosts that survive. A major feature of protozoal infections is that the severity of disease is related directly to the intensity of the infection; the more organisms that are present, the more severe the disease. The intensity of infection is usually determined by the number of infective forms that enter the animal initially (the infective dose).



- **Metazoa:** The metazoa are an artificial and extremely diverse assemblage of multicellular animals that includes arthropods and various types of worms (helminths). These are the organisms that are usually thought of as "parasites" when different types of diseases are being discussed. In an ecological sense, most are macroparasites in that they are visible with the naked eye, are long-lived, usually do not replicate extensively within the host, result in infections that tend to be chronic, and provide short-lived immunity with reinfection occurring commonly. As with protozoa, the effect of metazoa on the host is highly dependent upon the intensity of infection. Individuals with few parasites are unlikely to have overt disease, while those with many parasites may be harmed severely. For example, moose calves that received 164 or 200 infective larvae of *P. tenuis* rapidly developed severe fatal disease, calves given 15 or 30 larvae developed moderate to severe non resolving neurological signs, and calves given 3-10 larvae developed clinical signs that abated or disappeared . A general feature of parasitism by metazoa is that the distribution of parasites

is aggregated across the population. Most members of a population have few or no parasites of a particular species, while the majority of the population of the parasite is concentrated in a few animals.

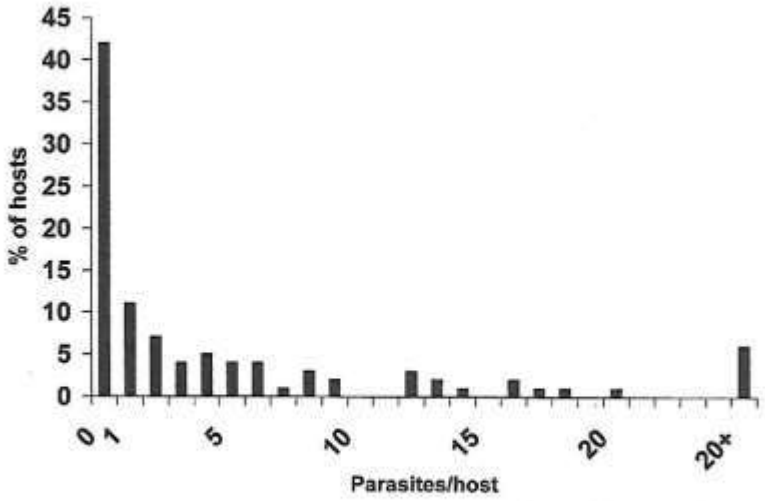


Fig. 3.6. Distribution of a hypothetical nematode parasite within a population of wild animals. A highly aggregated distribution of this type is typical of many parasites, with most animals having few or no parasites, and a few individuals having a large number of parasites.

This has important consequences including the following:

1- Severe negative effects (detectable disease) are concentrated in the few heavily infected individuals.

2- A small proportion of the host population is responsible for most of the parasite transmission.

3- It is difficult to measure parasitism rates and the effects of parasitism in populations without examining large samples of animals. Because of the diversity of taxa, lifestyles, and ecological niches filled by metazoa it is difficult to give a simple overview of the group.

II- NONINFECTIOUS DISEASES

Noninfectious diseases are caused by factors other than living organisms that cause harm while living in or on an animal's body. (Some noninfectious diseases, such as botulism and cyanobacterial poisoning, are caused by toxins produced by living organisms but these organisms do not usually live in or on the animal)**.**

The term "noninfectious disease" is a general term for conditions not caused by living organisms that colonize the body. Included within this group are diseases caused by genetic disorders; physical agents including heat, cold, trauma, and radiation; metabolic alterations; degenerative changes; deficiency of required nutrients; and chemicals including human-made contaminants and natural toxins.

- **Genetic disorders** are relatively common in domestic animals as a result of inbreeding and selection for specific traits and also occur in small, closed human societies. It is unlikely that genetic disorders are common in large out-bred populations of wild animals because of negative selective pressure, although individual animals are found with conditions that are suggestive of a genetic disorder. Genetic disorders are likely to be more common in small populations and to become more important as habitats for wild animals become increasingly fragmented, resulting in isolated subpopulations in which inbreeding may occur. It is more difficult to identify genetic disorders in wild animals than in humans or domestic animals because of the difficulty in identifying individuals and establishing their genealogy. It is important to distinguish between "congenital" and inherited disorders. The term

"congenital" simply means present at birth or hatching, and congenital disorders may be caused by a great variety of factors including various types of infection of the mother or the developing embryo/fetus, certain contaminants, and physical factors such as overheating or cooling of eggs. Inherited disorders are the result of a genetic abnormality.

- **Physical Agents:** Physical agents including cold, heat, and trauma from natural events such as hail storms can usually be linked to injury by association in time and space. These stochastic events are unlikely to be important at a population level except at the extremes of a population's range or for very small populations.

- **Deficiency of Required Nutrients:** Food supply is one of the basic limiting factors for wild populations. Food supply may influence both survival and reproduction negatively, but the effects of reduced or diminished supply are not easy to quantify. Some wild animals die directly of starvation (loss of body condition and functions to the point of death). Others may develop specific dysfunctions, such as the occurrence of osteoporosis in malnourished moose calves in Norway, but protein-energy malnutrition often is intertwined inextricably with predation and other forms of disease. As discussed earlier, it may be impossible to decide if individual animals are malnourished because of the demands of parasites or if parasites have flourished because of reduced resistance as a result of malnutrition.

- **Chemicals:** There is a huge literature on the association between chemical substances and wild animals. A cause: effect relationship between exposure to the chemical and disease has been established clearly for some substances. Some of these chemicals may

be acting as precipitating or contributing causes to other conditions, for example by modifying immune or hormonal function, but associations of this indirect type are difficult to prove conclusively or to separate from the effects of other nonspecific stressors, such as malnutrition, that might have similar effects.

III- NONINFECTIOUS TRANSMISSIBLE DISEASES

Noninfectious transmissible diseases This basic subdivision is challenged by our current knowledge about a class of diseases called the transmissible spongiform encephalopathies (TSE). These diseases, which include chronic wasting disease of deer (CWD) and bovine spongiform encephalopathy (BSE) among others, are believed to be caused by proteinaceous agents called prions that lack nucleic acid and, hence, do not fit the definition of living organisms. However, the TSE are transmissible and behave like infectious agents in many respects. There is no established category for the TSE, and they often are considered with infectious diseases. (Wobester , 2006) believed that it is more appropriate to place them in a separate category that he called “noninfectious transmissible diseases.” .

Discovery of the TSE and the importance of diseases such as chronic wasting disease of wild cervids (CWD) and bovine spongiform encephalopathy (BSE) have confused the definition of what is an infectious or noninfectious disease. The currently accepted wisdom is that these diseases are caused by a change in structure and function of a normal host cell protein called the prion protein (PrP). Normal PrP (often called PrP^{C}) is found at highest concentration in nerve cells, and its biological function has not been defined. Disease

is associated with accumulation of a form of the protein that is structurally different from PrP^{C} and that is resistant to enzymes (proteases) that break down normal proteins. This abnormal, resistant form of the protein, often called PrP^{res}, has identical amino acid sequences to PrP^{C} but differs in the way that the protein molecule is folded. The unique feature of the TSE is that once PrP^{res} enters a suitable animal either by some natural route or experimentally, it may promote production of PrP^{res} from PrP^{C}. There is good evidence that the condition can be passed from one individual to another in some TSE, particularly in CWD. Although the method by which transmission occurs is unknown, deer may become affected by CWD from exposure to environments in which affected deer had lived or died previously. Because of their transmissibility, various TSE may behave like an infectious disease but, because the agent has no genetic identity (no nucleic acid), the disease also might be perceived as a form of intoxication rather than an infectious disease. Miller and Williams (2003) used the term "**contagious prion disease**." It is likely more correct to describe animals with a TSE as affected rather than infected.

FINDIN G DISEASE

The vast majority of disease occurrences in wild animals is invisible and unrecognized, even for those diseases that may be lethal. Disease among wild species often is likened to an iceberg in that only a tiny tip projects above the water to be visible while the bulk of disease is hidden from view. Detecting covert disease is complicated further because very few people are looking for or observing wild animals on a regular basis. Even when large conspicuous species are involved, obvious disease may go unrecognized.

The circumstances in which diseased wild animals are most likely to be detected include:

1- Mass die-offs of conspicuous species.

2- Individual large animals that are conspicuous because of abnormal behavior, such as moose with neurologic disease caused by infection with the nematode *Parelaphostmngylus tenuis.*

3- Animals with conspicuous external abnormalities that are observed at close range, for example skin tumors in hunter-killed deer.

4- Species that are the subject of intense observation or interest. Often this occurs when disease in wild animals is linked with a human health risk.

Many methods have been used to search for disease in wild populations, but these can be categorized into four basic types of surveillance :

1. Searching for sick or dead animals

2. Searching for the causative agent

3. Searching for a physiologic response to the causative agent

4. Searching for evidence of the disease or the causative agent in a species other than the primary species.

SEARCHING FOR SICK OR DEAD ANIMALS

Finding animals that are sick with or that have died of a disease is the most direct evidence that disease (i.e., dysfunction) is occurring in an area or population. However, this technique has limited application and invariably results in an underestimation of the actual frequency of occurrence of disease. Sick and dead wild animals are very difficult to find. Sick animals are likely to hide, and clinical signs of disease are often short-lived in wild animals so that there may be only a transient window of time during which a sick animal might be detected. Wild animals seem to be good at disguising illness, probably because animals with obvious dysfunction are likely to be recognized by predators and removed from the population. Sick animals that die of disease or that recover also are lost to this form of surveillance.

The clinical signs of many diseases are not sufficiently specific or distinct so that an observer can be confident that the animals actually have the disease in question. For

instance, raccoons infected with canine distemper virus act similarly to animals infected with rabies virus and the diseases cannot be reliably differentiated on the basis of clinical signs.

Carcasses of animals that have died of disease are removed rapidly by scavengers and become unavailable for detection. The rate of removal is likely to be more rapid when the animals involved are small and when only a few animals die, so that the scavengers are not satiated. Even when many carcasses are available, they may disappear rapidly.

SEARCHING FOR THE CAUSATIVE AGENT

Surveying for the causative agent may provide a more complete picture of the frequency with which a disease occurs in the population than looking for sick or dead animals. This is because animals are often infected with agents, have elevated levels of toxic substances in their tissues, or are deficient in some required nutrient before they appear ill or die, and because disease may be subclinical or fleetingly clinical in many diseases. Samples can be collected from either dead or live animals for this purpose. Many factors must be considered when using the presence of a causative agent to detect or monitor the occurrence of disease in an animal population.

1- Infection or contamination with residues is not synonymous with disease. Detecting the agent in an animal does not mean that the animal is currently, or will in the future become, diseased.

2- The number of agents or the amount of residue detected in samples may not relate directly to the intensity of infection or contamination. This is best

documented for helminth parasites in which the number of eggs in an animal's feces may not be indicative of the number of worms present in that animal.

3- Not all the individuals that are infected or that have a causative factor in their tissue will be detected. There are a number of reasons why some animals may be missed including the following:

a- Recently infected animals may not have eggs, larva, oocysts, bacteria, or virus in the tissues or extract that are tested.

b- Infectious agents may be present only periodically or sporadically in the tissues or excreta examined.

c- The tissue distribution of agents and residues may vary with the length of time since exposure. For instance, the tissue distribution of lead varies with the time since ingestion. Lead residues in liver and kidney are indicative of recent exposure, and lead residues in bone are indicative of exposure in the more distant past. If only bone is examined, animals exposed recently to lead may be missed.

d- Agents may be present but latent or hidden and undetectable with most techniques. This is particularly a problem with herpesvirus infections,

e- The tissue or excreta sampled may be inappropriate for detecting the agent.

f- The way that the samples are handled after collection may reduce the likelihood of detecting the agent.

SEARCHING FOR A PHYSIOLOGIC RESPONSE TO THE CAUSATIVE AGENT

One of the most commonly used methods of disease surveillance is to search for some physiologic indicator of exposure to an agent. The most frequently used technique of this type is to test for evidence that the immune system has been activated. There are many tests of this type but all are based on detecting either antibody (these tests are broadly classed as serologic tests because blood serum is the usual sample) or evidence of a cell-mediated response, including elevation in substances such as cytokines produced by the immune response. Several factors must be considered when interpreting the results of tests that measure a physiologic response:

1- ***Recent exposure and test results***. Animals exposed recently to the causative agent may not have had sufficient time to respond and will test negative. For instance, antibodies are usually not detectable in serum until about a week after first exposure to an agent.

2- ***Exposure versus infection***. The presence of an immune response indicates that the animal has been exposed to an agent at some point in time. It does not necessarily indicate that the animal is infected when the sample was taken.

3- ***Exposure response and level of detection***. Animals may have responded in the past to exposure to an agent but the response has waned below the level of detection of the test. We do not know how long physiologic responses to many diseases persist at detectable levels in wild animals.

4- ***Antibodies in young animals***. Young animals may have antibody acquired passively from their mothers. Presence of this antibody indicates that the mother and not the offspring was exposed to the agent.

5- ***Generic versus specific responses***. Many physiological responses are generic rather than specific. This is a problem with some biomarkers such as activation of hepatic detoxifying enzymes that _may be elevated after exposure to several types of compound. So-called cross~ reaction with other organisms also occurs in some serological tests. .

6- ***Responses by different species***. The response by different species to an agent may be substantially different so that tests that are useful in one species may be worthless in another species.

SEARCHING FOR EVIDENCE OF DISEASE IN SPECIES OTHER THAN THE PRIMARY SPECIES

It may be more convenient or practical to look for evidence of a disease in a species other than the one that is affected by the disease or that is important in its maintenance or perpetuation. Often this technique takes advantage of a trophic relationship in which predators or scavengers are examined for evidence of a disease that occurs in their prey. The method is effective because predators and scavengers examine or “screen" a large sample of prey animals, and because predators are generally longer-lived than prey. Plague (*Yersinia pestis* infection) is primarily a disease of rodents, but carnivores including coyotes, dogs, badgers, and bobcats have been used to monitor plague activity in the western United States.

SAMPLING TO DETECT DISEASE

All types of sampling used to detect or monitor disease are potentially compromised by several basic problems, and these often are more profound and difficult to deal with in wild animals than in either humans or domestic animals. These will not be dealt with in detail here, but anyone contemplating a disease survey should be aware of the basic concerns and try to address those relevant to their situation.

SAMPLE SIZE

Sampling often is done to answer two basic questions about a disease:

1. Is the disease present in the population?

2. How common is the disease in the population?

Although the questions appear simple, obtaining a useful answer is not a simple task. For both questions the confidence one can have In the answer de pends upon the number of animals examined (sample size) and the accuracy (validity) of the test that has been used. A basic fact that often is not recognized is that the more rarely a disease occurs in the population, the larger the sample that is needed to answer either question adequately. Determining whether or not a disease is present in wild animals is a particularly perplexing problem, especially when the disease in question affects humans or domestic animals and there is pressure for an absolute yes or no answer. One can state unequivocally that the disease is present if even a single animal with the disease is found, but how confident can one be that the disease is not present if no diseased animals are detected? A useful rule of thumb when confronted with this question is the “rule of three” which is based on the

observation that “if none of n patients shows the event about which we are concerned, we can be 95% confident that the chance of the event is at most 3 in n (i.e.. 3/n)." For example, assume that 40 elk (n = 40) from a large population have been tested for tuberculosis and all were negative. Based on this sample of 40 animals, how confident can we be that the population is actually free of tuberculosis? Assuming‘ that the test is 100% accurate (which is highly unlikely) and that the sample is representative of the population (also highly unlikely), the answer is that we can be confident, at the 95% level, that the prevalence of tuberculosis in the population is not greater than about 3/40 or 7.5%. This is a very long way from being certain that the population is free of the disease! The formula can be rewritten n = 3/p to give a rough estimate of an approximate sample size needed to detect a particular prevalence (p) of disease.

VALIDITY OF TESTS

The validity of a test is defined as ***the ability to distinguish between those that have the disease and those that do not.*** Validity includes two components: **sensitivity and specificity**. Sensitivity describes ***the ability of a test to correctly identify those with the disease and is expressed as the proportion of affected animals that is identified as positive by the test***. Specificity ***is the ability of the test to correctly identify those that do not have the disease and is expressed as the proportion of animals that do not have the disease that are identified as negative.*** In order to calculate the specificity and sensitivity of a test, we obviously must know which animals actually do have the disease. This usually is done by comparing the test results with the results of some so called gold standard. For instance the

gold standard for tuberculosis is isolation at the causative bacteria from specific tissues following detailed necropsy. The methods used and the tissues to be cultured are rigidly defined, and if bacteria are not isolated, the animal is considered to have been free of the disease. In the case of tuberculosis, bacteria cannot be isolated from a small proportion of animals that are known on other grounds to be infected. The method used to determine sensitivity and specificity is shown in table below:

	Diseased	Not diseased	Total
Test positive	81	100	181
Test negative	37	782	819
Total	118	882	1000

Note: Data from a hypothetical situation in which 1000 animals were tested. On the basis of another "gold standard" test. 118 of the animals were considered to be diseased and 882 were considered to not have the disease. The sensitivity of the test is the proportion of the diseased animals that was correct1y identified (81/118 = 68.5%), the specificity of the test is the proportion of the animals that were not diseased that tested negative (782/882 = 88.7%).

To see how sensitivity and specificity affect the use of a test, let us assume that the test described in the table (sensitivity = 68.5%, specificity = 88.7%) is to be used as the basis

for removing bison from a wild herd in which about 40% of the animals are infected. Every animal that tests positive will be removed from the herd (culled) in an attempt to control the disease. We can predict that if 1000 bison are tested, about 400 of them actually will be infected. Since the test is 68.5% sensitive, it will detect 274 bison that have tuberculosis (these are true positives) but it will miss 126 infected animals (false negatives) that would remain in the herd. The other side of the test is that 600 of the 1000 bison are not diseased. Because the specificity is 88.7%, 532 of these will test negative (true negatives) but 68 will test positive (false positives) and these will be culled in error.

If we examined dead mice for the larval stage of the tapeworm *Echinococcus multilocularis* , animals either do or do not have visible cysts. But even in this disease, there is some likelihood that very early infections in which the larvae are minute and buried deep in the liver parenchyma will be mistakenly classed as not being infected, so the sensitivity of this simple test is likely to be <100%. Animals misidentified in this way are false negatives and result in underestimation of the actual frequency of occurrence of *E. multilocularis*. Larval cysts of other tapeworms might be wrongly identified as *E. multilocularis* so that the specificity might be <100%, resulting in false positives and overestimation of the abundance of *E. multilocularis*. (The sensitivity and specificity of this test could be improved by thorough dissection of the liver to detect small cysts and by microscopic examination of the cyst contents to confirm their identity) The potential for making mistakes in identification of affected and non-affected animals is much greater for most tests than in the test just described for *E. multilocularis*.

TERMINOLOGY & CALCULATIONS

Sensitivity: the ability of a test to correctly identify diseased animal with a disease.

Specificity: the ability of a test to correctly identify animals without the disease.

True positive: the animal has the disease and the test is positive.

True negative: the animal does not have the disease and the test is negative.

False positive: the animal does not have the disease and the test is positive.

False negative: the animal has the disease and the test is negative.

Sensitivity is calculated based on how many animals have the disease (not the whole population). It can be calculated using the equation: ***sensitivity=number of true positives/(number of true positives+number of false negatives)***.

Specificity is calculated based on how many animals do not have the disease. It can be calculated using the equation: ***specificity=number of true negatives/(number of true negatives+number of false positives)***.

Example:

A new test is developed for the detection of disease called (A). When it is tested in a group of 115 animals with disease (A), 80 have a positive test. In a group of 215 individuals without disease A, 5 have a positive test. *What is the specificity and sensitivity of the test?*

sensitivity=number of true positives/(number of true positives+number of false negatives).

Sensitivity= 80/80+35

=69.56

specificity=number of true negatives/(number of true negatives+number of false positives).

Specificity= 210/210+5

=97.67

Pathogens Population

Ecologists need the ability to describe an organism's surroundings. Parasites are not fundamentally different from other living organisms in this regard, but, because parasites often have complex life cycles, various descriptive terms can be rather misleading.

(1) **Site, location, and habitat**. The terms site and location have a long history of use among parasitologists, and, to them, the terms may carry specific connotations. To others, such connotations are unknown or vague. In disease ecology, it is considered that the site or location of a parasite to **be the topological or spatial location in a host where a particular sample of parasites is collected**. **Site and location are thus anatomical parallels to geographic locality.**

Habitat refers to the typical local environment in which parasites occur.

Remarks: In disease ecology, we disagree with Margolis et al. (1982) that habitat should not be used to refer to "the tissue, organ or part of the host in [on] which a parasite was found." We suggest that, because organs and tissues provide the local environment (including physical, chemical, and biological surroundings), they are appropriately called habitats. In fact, we feel that the term habitat is preferable because it has a long history of similar use in the literature on free-living forms and it can easily be compounded, e.g., microhabitat, habitat-specific. Further, recognizing the complexity of many parasite life

cycles, it will often be necessary to describe the surroundings of free-living phases, particularly in autecological studies. The use of habitat (rather than site or location) seems to make better sense when describing eggs in fecal pats, juveniles on blades of grass, or miracidia in a water column. As with most ecological words, scaling is important for the term habitat, and it is appropriate to refer to the intestinal veins as well as a rice field as habitat for *Schistosoma mansoni*.

(2) **Locality**. The term locality refers to a geographic locale of the external environment where the parasite is found.

Remarks: Locality is used widely for geographic position, and the term should be restricted to an identification of where, geographically, the individual, population, or community is obtained. Locality might be the spatial region where a host (or hosts) is (are) collected or it might refer to the spatial region where a substrate (or substrates) is (are) examined for parasites.

(3) **Niche**. The niche of a parasite refers to its role, and how it fits, within a particular community.

Quantitative descriptors of parasite populations

Most quantitative descriptors such as prevalence and mean abundance are point estimates based on samples from the whole population of hosts.

(1) **Prevalence**. Prevalence is the number of hosts infected with 1 or more individuals of a particular parasite species (or taxonomic group) divided by the number of hosts examined for that parasite species. It is commonly expressed as

a percentage when used descriptively and as a proportion when incorporated into mathematical models. Prevalence is intended as a descriptive statistic for presence-absence data on parasites in a sample of hosts and is used when it is desirable to classify hosts into 2 categories, infected and uninfected, without regard to when the infected hosts acquired their infection.

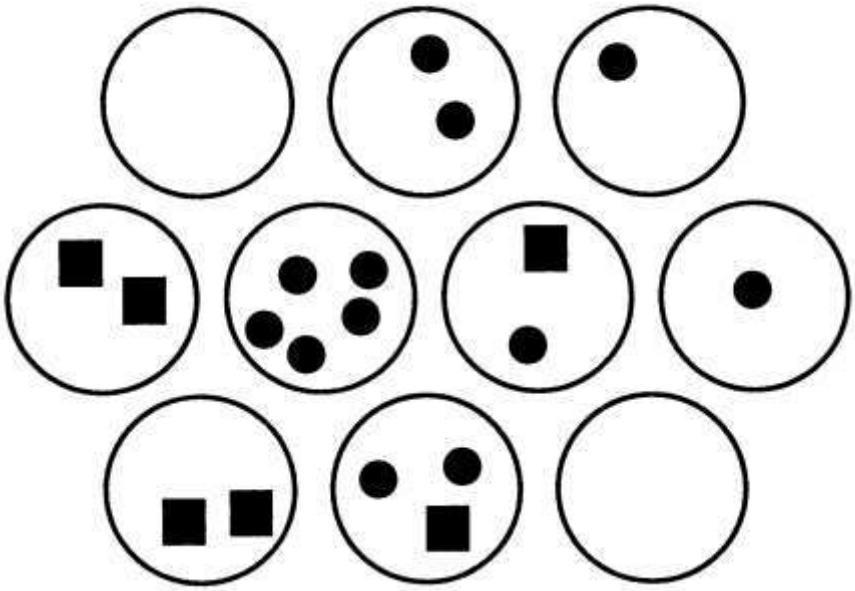


Figure 1

In Figure 1, 10 host individuals are infected with none, 1, or 2 species of parasites. The prevalence of the circle parasite is 6/10 = 0.6 (or 60%)

The prevalence of the square parasite is 4/10 = 0.4 (or 40.

(2) Incidence. Incidence is the number of new hosts that become infected with a particular parasite during a specified time interval divided by the number of uninfected hosts

present at the start of the time interval. Remarks: Incidence is a descriptive statistic used to determine the risk of acquiring new infections by individuals in a population of hosts. Incidence is applicable only to the uninfected individuals in the host population, without regard to the number of hosts with existing infections. An example of the calculation of incidence. "In an epizootic of an acute viral disease on a dairy farm with a population of 100 susceptible cows, 7 became sick on day 1 of the outbreak, 15 on day 2 and 10 on day 3. The daily attack rates would be: 7/100 = 0.07 (7%), 15/93 = 0.16 (16%), and 10/78 = 0.12 (12%), respectively." Note the decrease in the denominator as hosts acquiring the infection in 1 time interval are now removed from the susceptible pool for subsequent intervals. Incidence is most commonly used to monitor the spread of clinical disease in populations of humans or domestic animals because determining the number of preexisting cases of the disease is relatively.

(3) Density. Density is the number of individuals of a particular parasite species in a measured sampling unit taken from a host or habitat, e.g., in units of area, volume, or weight. Remarks: Density is used widely in the ecological literature and can be equally applied to parasites. When the sampling unit is an individual host, it would be proper to report infections as "A density of X parasites per infected host (or per host)." However, because of the frequency with which parasitologists use the host as the sampling unit, the terms intensity and abundance, with their implied sampling units, are more concise and seem preferable. We, therefore, recommend that density be used when an accurate census of all parasites in a host is difficult or impossible to make. For example, an efficient density measure of

Trypanosoma lewisi might be the number of flagellates per milliliter of rat blood. In any case, it is important to specify the denominator to avoid confusion.

(4) Intensity (of infection. Intensity (of infection) is the number of individuals of a particular parasite species in a single infected host.

Intensity is a form of density with the sampling unit specifically defined as an individual infected host. Therefore, intensity is a convenient measure for parasitologists because hosts are discrete and natural sampling units. In Figure 1, 6 hosts are infected with circle parasites, and the intensities are 1, 1, 1, 2, 2, and 5. Four hosts are infected with square parasites, and the intensities are 1, 1, 2, and 2.

The potential confusion of intensity with other forms of density makes it necessary to define it following initial use. Alternative terms for intensity: Although a few authors will likely continue to use synonyms (worm burden, parasite load, and degree, level, or extent of infection), we recommend the use of intensity.

(5) Mean intensity. Mean intensity is the average intensity of a particular species of parasite among the infected members of a particular host species. In other words, it is the total number of parasites of a particular species found in a sample divided by the number of hosts infected with that parasite. Remarks: In Figure 1, the mean intensity for circle parasites is 12 divided by 6 or 2, whereas for square parasites it is 6 divided by 4 or 1.5.

(6) Abundance. Abundance is the number of individuals of a particular parasite in/on a single host regardless of whether or not the host is infected. Remarks: Abundance is also a form of density, and it differs from intensity in that, by definition, an intensity of 0 is

not possible whereas an abundance of 0 is appropriate. In Figure 1, the abundances of the circle parasites are 0, 0, 0, 0, 1, 1, 1, 2, 2, and 5, and the abundances of square parasites are 0, 0, 0, 0, 0, 0, 1, 1, 2, and 2. We find the distinction between intensity and abundance to be useful because, in some studies, only the infected host sub-population is of interest; in other studies, the whole host population is of interest. Often in community studies, one might wish to examine phenomena such as cooccurrences where 0-sized populations are important.

(7) **Mean abundance**. Mean abundance is the total number of individuals of a particular parasite species in a sample of a particular host species divided by the total number of hosts of that species examined (including both infected and uninfected hosts). It is thus the average abundance of a parasite species among all members of a particular host population. For example, in Figure 1, the mean abundance of circle parasites is 12 divided by 10 or 1.2; for square parasites, it is 6 divided by 10 or 0.6.

The nesting of parasite populations

(8) Infrapopulation. A parasite infrapopulation includes all individuals of a species in an individual host at a particular time. Remarks: In Figure 1, there are 10 host individuals, but only 8 hosts have infrapopulations; there are, however, 10 infrapopulations: 6 of circle parasites, 4 of square parasites.

(9) Component population: all individuals of a parasitic species in/on all members of a host population at a particular time.

(10) Suprapopulation: all individuals of a parasitic species (whether on or off a host) at a particular time.

(11) Infracommunity: A community of parasite infrapopulations in/on a single host.

Remarks: In Figure 2, there are 15 hosts and 11 infracommunities. If individuals do respond to the presence of other species, it is at this level that any selection pressures will occur.

(12) Component community: all infrapopulations of parasites in/on all members of a host population at a particular time.

Remarks: In Figure 2, there are 5 component communities: triangle and square final hosts, octagonal and circle intermediate hosts, and free-living phases.

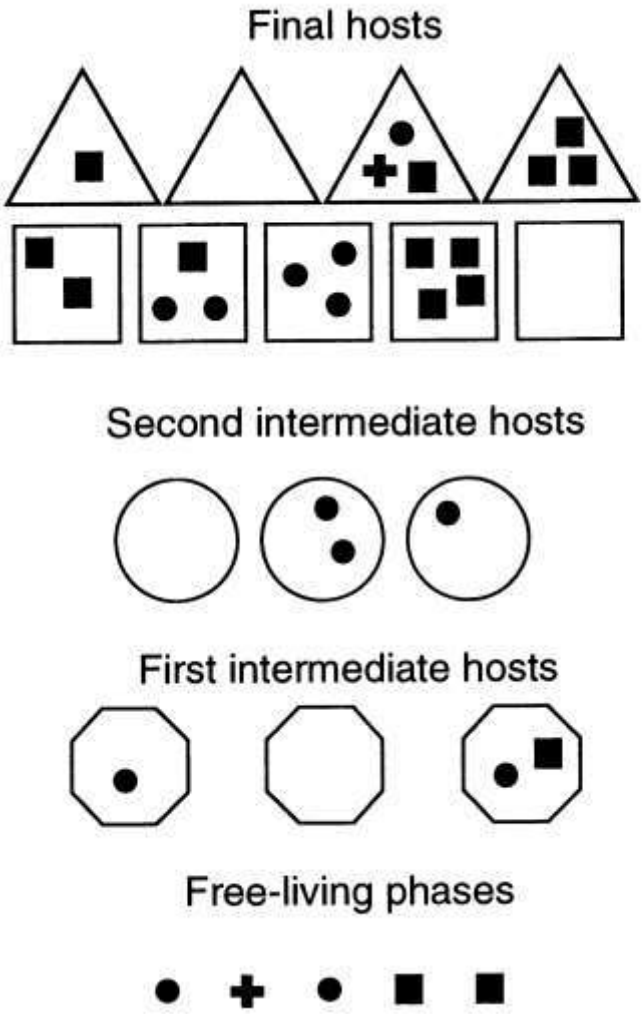

Final hosts
Second intermediate hosts
First intermediate hosts
Free-living phases

Figure 2

Parasitic systems and the structure of parasite populations

Every population system is part of a certain community. In a community, the population system interacts with the population systems of other species. These interactions can be more or less close and regular. In the case of stable and regular interactions between individuals of two species, the stable community connection arises between the population systems of these species.. The stable complexes of the population systems are formed also by other types of connections between individuals, i.e. competition, predator–prey interactions, etc. In general, the more important the organism of one species is for the organism of another, the greater the entirety of the complex that makes up their population systems. Population systems of the parasitic organisms in the community are in contact with the population systems of other species: firstly, of their hosts, but also of predators, competitors and so on. The interactions of the individual hosts and parasites are very close and include a complex of the topic, trophic interactions allowing us to define a host organism as the environment for the parasite . At the population level, these interactions are reflected in the stable existence of the complex of several population systems. This complex involves a population system of the parasite and the population systems of all the host species connected with this parasite. A complex of the population systems of hosts united by the population system of a parasite was designated as a parasitic system.

A minimum of two population systems is required for a parasitic system – a population system of the parasite and that of the host. However, this is a rare situation in nature that appears only in the case of a monoxenic life cycle and strict specificity of a parasite. As a rule, a parasitic system consists of more elements (= population systems): parasites of a single developmental stage may use several populations of the different host species which in this case are termed paraxenic hosts. Additional extension of the number of interacting populations occurs if the parasite has a di-, tri or tetraxenic, etc. life cycle.

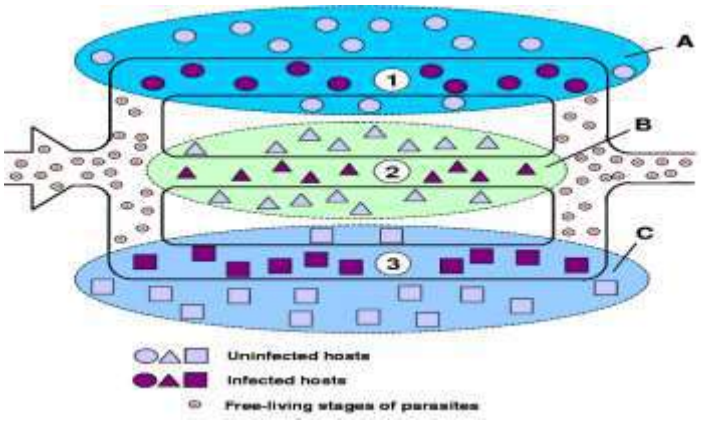
The population system is a functional part of a particular community. Steady interrelationships between population systems of different species within the community (referred to as "**community links**") appear to be a prerequisite for the formation of a complex of population systems. A prominent example of this is the parasitic system. **The parasitic system *is the population system of a parasite with all the connected populations of its hosts.*** The complexity of a parasitic system depends on:

(1) peculiarities of the life cycle of the parasite, since its population system is the organizing component of the parasitic system

(2) subdivision of the environment for the parasites. The first trait is discussed from the standpoint of the phase structure of populations, which is clearly seen in parasites. The second one comprises the organization of the parasites' environment according to the scale of variability of hosts. These make it possible to recognize spatial and functional parts in the framework of the parasitic system.

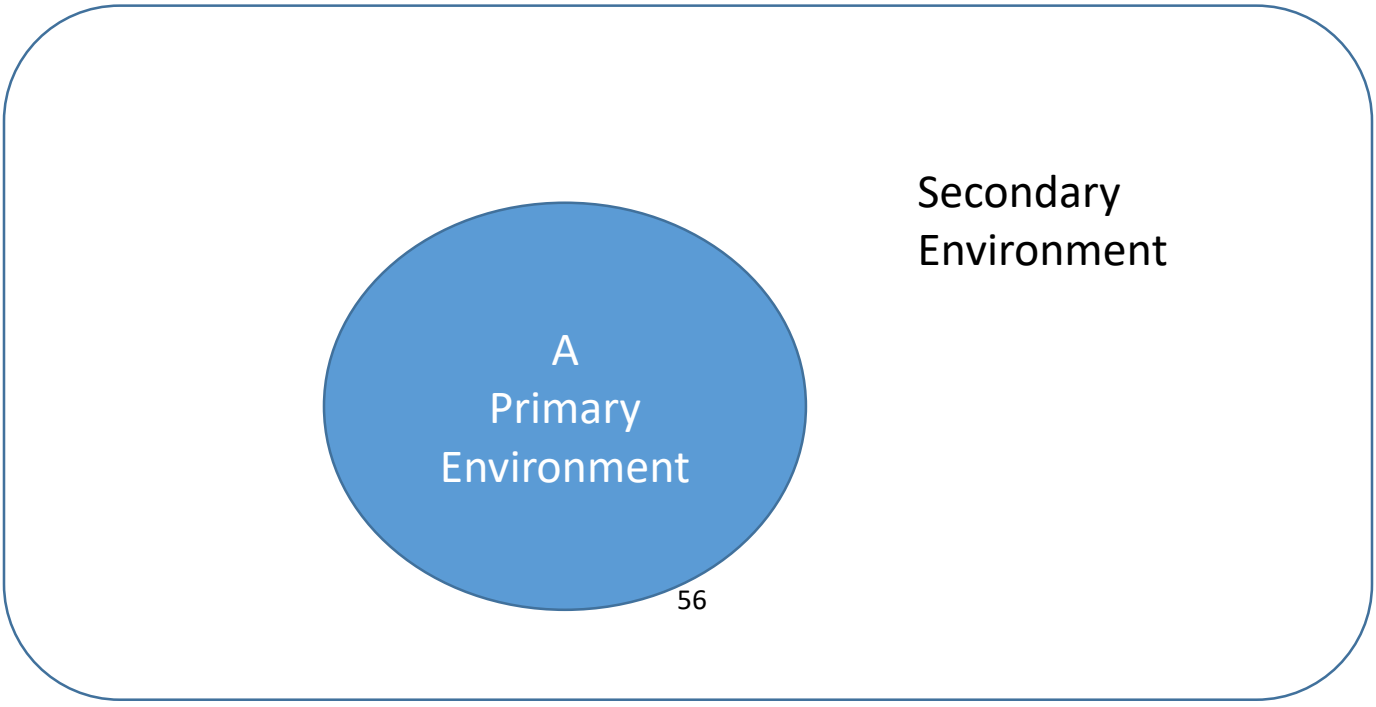
Community connection – interrelations of population systems of two species in the community that are stable in time due to stable and regular interactions between individuals of these two species (predator–prey, parasite–host, mutualistic, competition and other types of interindividual interactions).

Parasitic system – complex of population systems of hosts, united by the population system of their parasite on the basis of stable parasite–host community connections



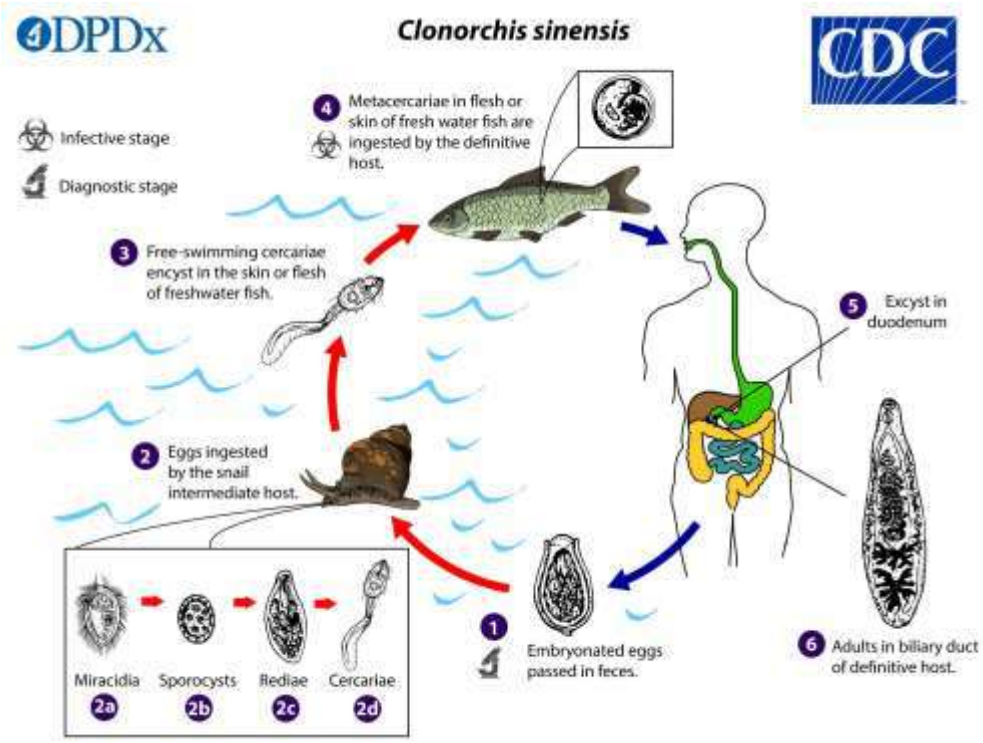
Primary environment for the parasite – organism of the host.

Secondary environment for the parasite – the external environment of the host.

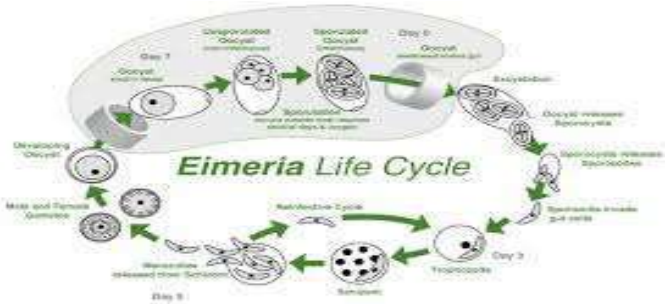


Complex life cycle – type of life cycle with two or more distinct, regularly alternating generations. These generations are usually characterized by different modes of reproduction.

Example : *Clonorchis sinensis*



Monoxenic life cycle – for the parasites – ability of the parasite to complete its full life cycle in/on the organism of only one host species.

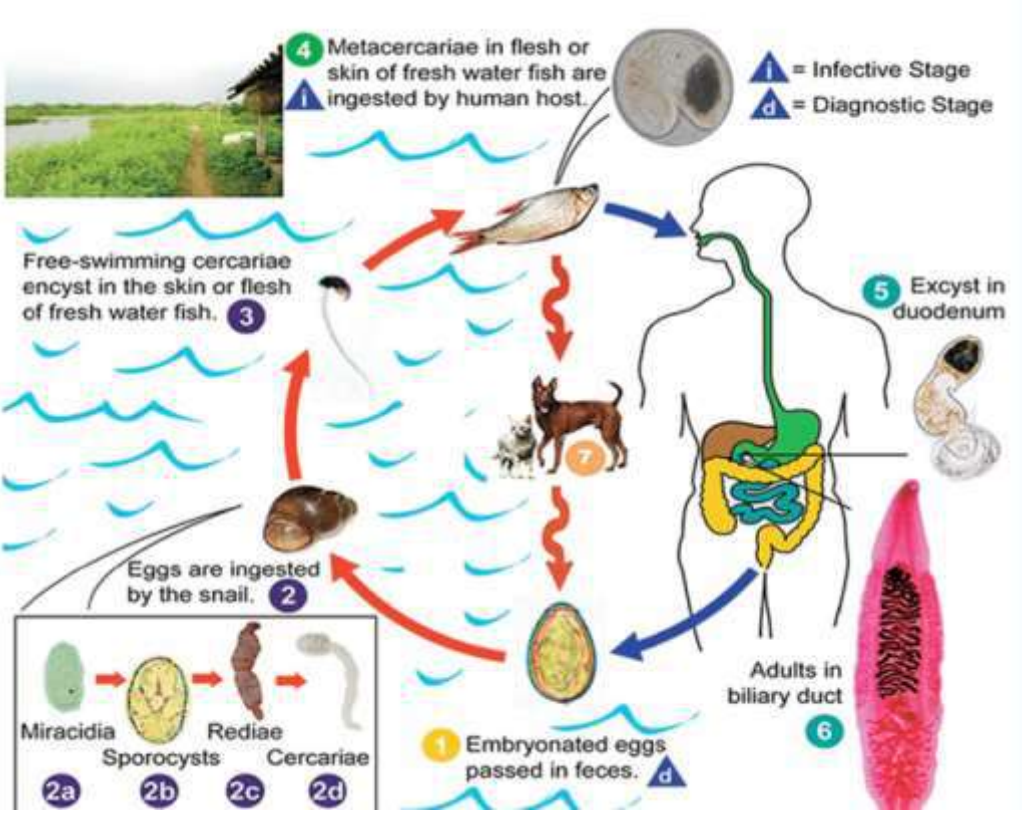


Di-, tri-, tetraxenic life cycle – for the parasites – necessity of more than one host for the full completion of their life cycle; i.e. necessity of two metaxenic hosts for a dixenic life cycle, three metaxenic hosts for a trixenic life cycle and so on.

Homoxenic life cycle – for the parasites – ability of the parasite to use only one host species for every stage of its life cycle. This is the case of extremely strict specificity of parasite.

Heteroxenic life cycle – for the parasites – ability of the parasite to use more than one host species for some or all stages of its life cycle. A heteroxenic life cycle comprises several paraxenic hosts.

Example : *Opisthorchis felineus*



Paraxenia – ability of the parasite to use several species of hosts for the development of the same stage of its life cycle.

Paraxenic host – different species of hosts which are used by the parasite at the same stages of its life cycle. In this case strict specificity is absent.

Metaxenic host – different species of hosts, which are used by successive stages of the life cycle of the parasite.

Aggregation of parasites

Parasites are generally **aggregated** among hosts, and it is commonly believed that aggregation is an important feature of the population biology of these organisms. Parasites aggregation creates variability among hosts in the effects of parasites, so the net effect of parasitism on the population of hosts often depends not just on mean parasite burdens, but also on the variability of burdens. From an individual parasite's point of view, aggregation creates variability in the number of other parasites occurring in the same host. If parasites experience density dependence in reproduction or mortality, either due to direct interactions or interactions mediated through the host, then aggregation may change the parasite population growth rate.

Processes that lead to aggregated parasite distribution can broadly divided into two categories: those that produce variability among hosts in exposure to parasites, and those that create variability either in host acceptability to the parasites or in host immune responses.

A fundamental aspect of the relationship between parasites and hosts is contained in the distribution of parasites amongst hosts. This distribution has repeatedly been shown to be clustered or aggregated in the sense that typically, ***a few hosts harbor many parasites, while the remainder of the hosts are virtually parasite free***. Aggregation has very significant implications for both hosts and parasites because of the following:

1- It affects their genetics and evolution.

2- It has been recognized to have many consequences for public health management.

3- It has been recognized to have many consequences for livestock management.

4- Aggregation has been shown to affect parasite ecology by stabilizing host-parasite population dynamics.

5- Aggregation has been shown to affect parasite ecology by facilitating interspecific co-infection as a result of increased host susceptibility.

6- Aggregation also influences parasite evolution by, e.g., increasing the level of intra-specific competitive interaction .

7- Aggregation of parasites amongst hosts affects the transmission of infectious human diseases.

A quantitative understanding of the mechanisms which lead to the observed levels of aggregation is thus essential for the knowledge of parasite ecology and evolution. One of the most fundamental issues in the field is to what extent differences in parasite loads reflect differences in the exposure of hosts to the infective stages of a parasite, or differences in the

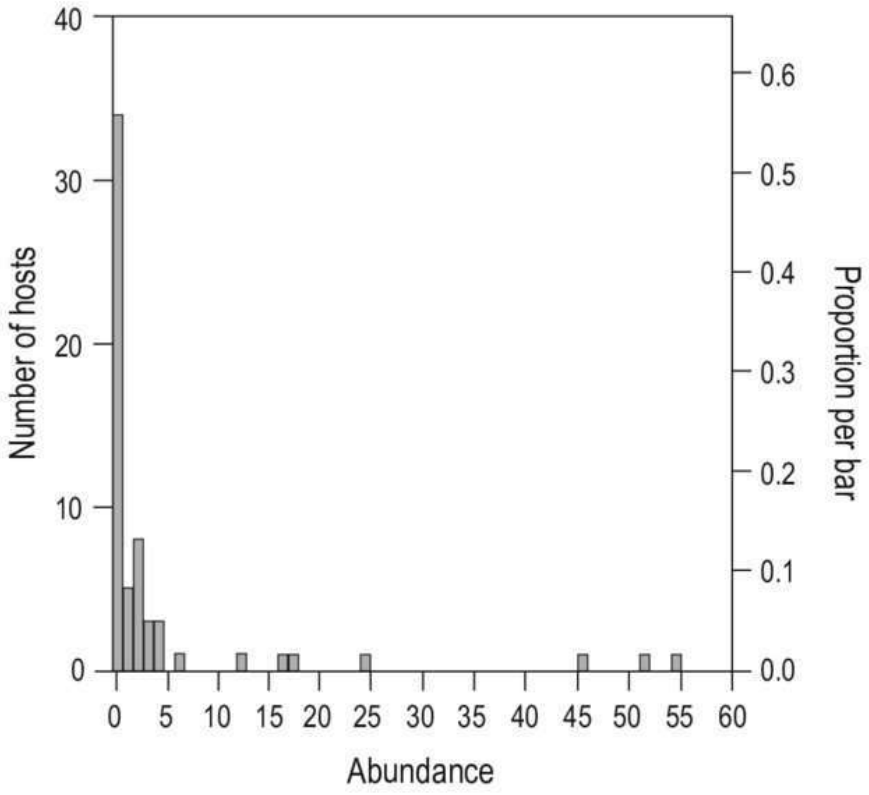
success of a parasite in infecting its hosts . The processes that are potentially involved in producing the distribution of parasites amongst hosts are two types:

(i) The number of encounters between a host and a parasite or a source of parasites

(ii) The number of parasites that are ultimately carried by a host, which result from a single encounter with a parasite or a source of parasites.

These two processes, henceforth referred to as ‘encounters’ and ‘success’

Exhaustive empirical surveys have shown that, almost without exception, macroparasites (parasitic helminths and arthropods) are aggregated across their host populations, with most individuals harbouring low numbers of parasites, but a few individuals playing host to many.Parasites are invariably aggregated across the host population, with the majority of the parasite population concentrated into a minority of the host population. In human communities, for example, generally less than 20% of individuals harbour 80% of the helminth parasite population. Thus, a relatively small number of individuals in the ‘tail’ of the parasite distribution are responsible for most parasite transmission and play an important role in the persistence of the parasite .



Box 2.1. Measures of aggregation

If the parasite population was distributed randomly amongst hosts, the variance (s^2) of the parasite distribution would be approximately equal to its mean (m), i.e.

Random distribution: $s^2 = m$ (1)

For an aggregated distribution, the variance is greater than the mean, i.e.

Aggregated distribution: $s^2 > m$ (2)

Thus, we can quantify the degree of aggregation simply as the ratio of the variance to the mean:

Variance-to-mean ratio = s^2/m (3)

You will notice that this ratio varies from zero (when parasites are uniformly distributed amongst hosts), through unity (for a truly random distribution of parasites), to a number equal to the total number of parasites (for a maximally aggregated distribution).

- Exercise: A dataset is comprised of information collected on 12 host, each of which was necropsied and the number of roundworms counted. The number of infected hosts were 7.
- One host infected with 10 *(Adult worm)*

- One host infected with 5 *(Adult worm)*
- One host infected with 1 *(Adult worm)*
- One host infected with 1*(Adult worm)*
- One host infected with 1*(Adult worm)*
- One host infected with 1 *(Adult worm)*
- One host infected with 1 *(Adult worm)*
- **Calculate the aggregation**

  Solution:

  Aggregation = variance to mean ratio

Mean abundance should be calculated

=20/12

=1.66

Variance =S^2

$$s^2 = \frac{\sum (X - \overline{X})^2}{N - 1}$$

=8.78

Variance/ mean

= 8.78/1.66

= 5.4875

Environmental Interactions

Environmental factors may affect the animal or the agent, and the action may be to modify animal abundance and density, the degree and type of exposure to the agent, the degree of resistance of the host, and even the virulence of the agent. It is not possible to identify or consider all of the possible environmental factors that might modify any animal-agent relationship.

Subject can be divided into **abiotic** and **biotic** factors, but it is important at the outset to state that the effect of human population is an overarching feature involved in every disease relationship. Even climate, that most basic of abiotic factors, has been modified by humans, and the effects of humans on water, land, vegetation, and animals are obvious. One of the most basic features of the investigation of any disease occurrence is to set it in **time and place**. Determining the spatial and temporal relationships among the factors involved in a disease often provides important clues as to what is causing the disease and why the disease occurred. Where a disease occurs, the location of affected individuals and outbreaks, and its general geographic distribution are determined by the presence of factors that are suitable for the animal(s) involved, for the agent(s).and for agent and host to interact.

ABIOTIC FACTORS

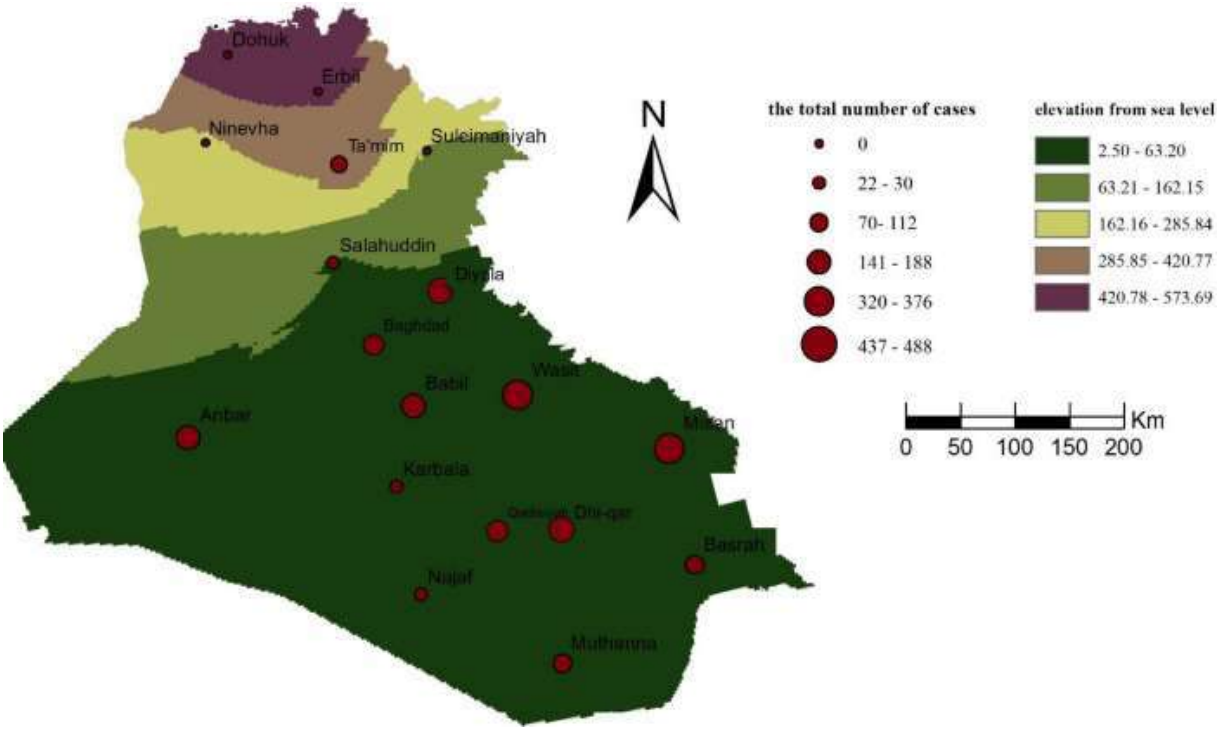
The principal abiotic factors that are going to be discussed here are **topography, climate and weather, water, bedrock, and soil**. These are not independent, for example, topography influences local climate, soil formation, and the distribution of water on the landscape. Nor are abiotic factors independent from biotic factors, for example, soil nutrients, aeration, texture, and microclimate have a major effect on the vegetation that grows on a site and on the animals that inhabit the area, in considering the distribution of disease events.

Topography influences distribution of contaminants and infectious agents by altering wind direction, speed, and dispersion of air-born materials and by influencing water flow. Topographical orientation influences the degree of solar irradiation, precipitation, vegetation, and how land is used by humans. In some situations, it may be difficult to untangle the interrelationships among factors, one of which might be altitude. See table below:

Disease (species)	**Altitude effect**
Anthrax (livestock)	Not seen above 300 meter, perhaps because temperature are not warm enough for significant sporulation.
Elaeophora schneideri	Prevalence of infection related to preferred altitude of the horse fly intermediate host.

Hanta virus pulmonary syndrome	Most exposure between 1800 and 2500 meter, non >2500 meter (corresponds with altitude with highest density of rodent host.

Al- Warid *et al*., (2019) showed that there was a significant relation ($p<0.05$) between elevation and the occurrence of kala-azar, with a decrease in elevation correlated to increases in occurrence. Overall, 96.7% of the cases occurred in provinces with an elevation ranging from 3–63 m above sea level. that leishmaniasis was not normally found at high-elevation because of decreases in sandfly density.



Climate is used here as the generally occurring weather conditions of a region averaged over many years. Climate has a major effect on the geographic distribution of

animal and plant species, disease agents, and human activities. The effect of climate on distribution is most obvious in those infectious diseases that involve an invertebrate as a vector or intermediate host, because poikilothermic animals are particularly sensitive to temperature, precipitation, and humidity. These affect the rate of invertebrate development, their longevity, their activity. and the rate of development of disease agents within them. For example. “geographically variable tick population dynamics are determined principally by climatic factors, temperature that drives development, and moisture availability that determines mortality” (Randolph et al. 2001). Because climate has a major effect on human activities, including agriculture, it also has an important indirect effect on the distribution of toxicants such as insecticides. Prevailing wind are responsible for the long distance transport of contaminants and the distribution of these in wild animals. Precipitation (rain and snow) has a major influence on the distribution of animals and disease agents.

Weather conditions have a strong effect on the local and immediate occurrence of disease and can also be a direct cause of disease in individual or groups of animals. Ambient temperature may interact with disease agents in many ways. Some effects of temperature may be related to energy metabolism and trade-offs. Much of the recent emphasis on temperature as a factor in disease has been on high temperature, but low temperature also may be related to disease. Cold ambient temperatures prolong the survival of many disease agents in the external environment, and disease may render affected animals more susceptible to the effects of cold weather.

Factor affected	General direction of change
Biting rate by blood-feeding arthropod vector	Increases with increasing temperature up to a point and then may decline.
Proliferation of cyanobacterial blooms in water	Increased at higher temperature
Rate of development of agents within vector or intermediate hosts	Increased with increasing temperature
Population of blowfiles on carcasses	Increased with increasing temperature

Water is involved in some manner in the ecology of virtually every disease and must be considered during the investigation of any condition.

Anthrax is excellent examples of the many ways in which water may influence disease. Anthrax caused by the bacterium *Bacillus anthracis* is primarily a disease of herbivorous. The bacterium has a spore stage that is extremely resistant in the external environment. Animals become infected by ingesting or inhaling spores. Anthrax outbreaks often occur in hot, dry weather following heavy rains and may be associated with low-lying areas. One explanation is that light spores in soil are carried to the low- lying areas by running water and become concentrated there as the water evaporates. Spores also may be dispersed from carcasses by water, and outbreaks in bison in northern Canada, perhaps because of dispersal of spores concentrated about carcasses into the soil. Tabanid flies are important

transport hosts for anthrax in some areas, carrying the bacteria on their mouthparts. These flies require aquatic environments for breeding, so that their distribution and abundance is dependent on surface water.

Water mechanism	**Example**
Breading habitat for vector	West Nile virus
Carrier of dissolved toxin	Selenium poisoning
Habitat for intermediate host	Trematodes that use aquatic snails
Habitat for toxin-producing agents	Cyanobacteria, algae that produce domoic and other marine toxins

The soil of an area reflects the parent bedrock and the long-term climate of the region and has a major influence on the distribution of vegetation and animals. Soil and bedrock can influence disease in many ways, and important features include its chemical, physical, and moisture content.

Soil Features	**Disease relationship**
Physical features	Sedimentation of lead pellet
Moisture and nutrient content	Reservoir for disease agents
Microorganisms	Conversion of toxin (methylation of mercury in sediments)

BIOTIC FACTORS

VEGETATION

Many diseases are associated with a particular type of vegetation. In some instances, this may reflect simply the distribution of the animals that are affected, or it may be related to the particular climatic or soil features of the area. In other cases, the type of vegetation may have an effect on survival of a disease agent while it is outside the host. For instance, both *lxodes scapularis* and *I. ricinus* (the vector tick for Lyme disease in North America and Europe, respectively) rarely are found in open grassland areas, probably because ticks that fall there are exposed to desiccation and die. Forested areas buffer climatic extremes and, because leaf litter is important for the survival of immature ticks, these species are more abundant in deciduous forests than in coniferous forests .

ANIMALS

This section will deal briefly with intraspecific and interspecific relationships among larger animals.

Intraspecific Interactions

In many diseases, other members of the same species are the most important environmental influence on disease. Their abundance, distribution, density, susceptibility to disease, and current disease status may be important. Other members of the population may

influence the rate of exposure to the agent and the relative resistance of individuals to the disease. (**Population a group of animals of the same species**.) Population size and density are subjects of great interest to those working with disease, and there is a general belief that disease in wild animals is "**more important**" when animal populations are large and/ or dense. A disease might be considered to be more important when populations are large or dense because:

(1) it is more apparent

(2) it occurs more commonly

(3) it involves a larger proportion of the population

(4) the effect on individuals is proportionately greater.

To illustrate the first of these, assume that on a wetland in 1 year there are 100 ducks of which 10 die of disease X. A few years later, the duck population is much larger and more dense with 10,000 birds on the same area, of which 1000 die of disease X. In the second year, the absolute number of ducks that died was much larger (100 times that in year 1), but the mortality rate, the proportion of the population that died, is the same (10%). Because a die-off of 1000 birds is much more likely to be detected than one involving 10 birds, finding dead birds in the second year might lead to the mistaken conclusion that disease X only occurs in large/dense populations.

In some situations disease may occur more commonly over time in a large population than in a small population. A minimum number of animals may be needed to allow the agent of some directly transmitted diseases to persist in the population. For

example, the minimum population size required for measles to persist in a human population is estimated to be about 300,000 to 500,000 persons (Black 1966). If the disease is introduced into a smaller population, the disease dies out and disappears because there are not enough susceptible individuals to maintain the infection. In this type of disease, the disease agent is present all the time in large populations, while it may only be present periodically or sporadically in small populations. To return to the wetland example, disease X might be present consistently when the population is 100,000 but only occur periodically when 100 ducks are present. Disease might be more important in dense populations if a greater proportion of the individuals that make up dense population is affected, that is, the occurrence of the disease is density-dependent.

Interspecific Interactions

Interactions with other species may influence disease in a variety of ways

Type of interaction	**Effects**
Competitor	Reduce availability of resources of target species.
Predator	Increases mortality rate and may reduce prevalent of agent in population
Scavenger	1- Remove potentially infectious carcasses. 2- May transfer agent to new sites.
Intermediate host	Facilitates transmission
Vector	Facilitates transmission

Transport host	Facilitates transmission
Bioaccumelator	Concentrate agent and pass to next trophic level.

Transmission of Infectious Disease

Transmission of infectious agents consists of three steps: **exit from the host**, **passage across the external environment** to find or be found by a new host, and **entry and colonization of the new host**. Success of transmission is dependent upon the number of organisms that exit. their persistence in the external environment and success in finding a new host, and the number that are able to initiate an infection in that host.

EXIT FROM THE HOST

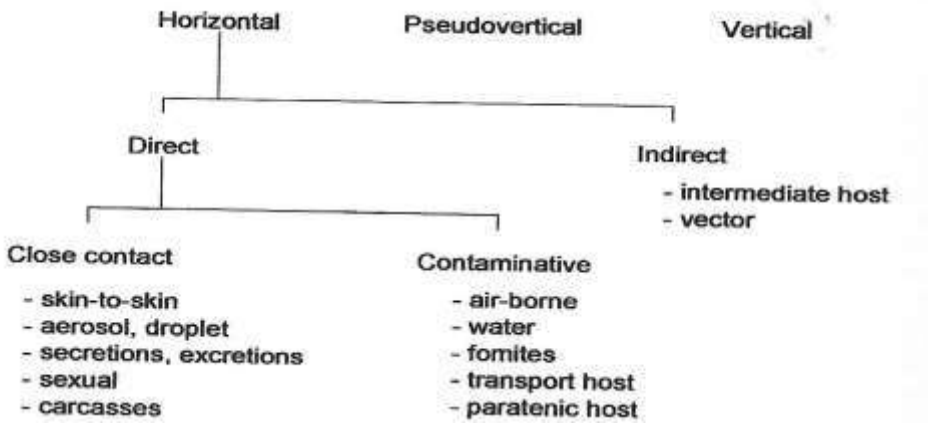
Infectious agents have to leave the body of the host for transmission to occur, except for those agents that are transmitted through predation or scavenging, in which the agent can wait in one host until consumed by the next. Routes of exit used by disease agents together with examples of diseases in which each route is important are shown below:

Rout of exit from host	**Example of disease**
Respiratory tract secretion	Tuberculosis
Saliva	Rabies

Feces	Many helminthes
Skin	Poxvirus infection
Blood	Malaria
Genital secretion	Brucellosis
Urine	Renal coccidiosis
Milk	Tuberculosis
Ocular discharge	Canine distemper
Via fetus or egg	Pestvirus infection
Postmortem decomposition of carcasses	Anthrax
Consumption of body by predator/ scavenger	*Sarcoystis* spp

CROSSING THE EXTERNAL ENVIRONMENT AND FINDING A NEW HOST

Infectious agents have developed an amazing diversity of methods for voyaging from one host to the next.



VERTICAL TRANSMISSION

Vertical transmission involves passage of an agent from parent to offspring. This may occur through passage of agents in semen, through the placenta or milk, or through the egg in egg-laying species. Toxoplasmosis can be transmitted vertically.

PSEUDOVERTICAL TRANSMISSION

Pseudovertical transmission involves passage of agents to neonates shortly after birth or hatching and before they have been exposed extensively to the outside world. Transmission is often from parent to offspring, because of their close association, but parents are not the only source of infection. Pseudovertical transmission has been used to describe transfer of bovine tuberculosis

HORIZONTAL TRANSMISSION

Horizontal transmission involves transfer of infection from one animal to other individuals in the population, independent of their parental relationship. This is by far the

most common method of disease spread and takes many forms. Several methods for subdividing horizontal transmission have been described.

Those dealing with microparasites tend to use the term:

"**direct transmission**" for situations in which transmission occurs by "close physical contact/proximity between individuals or by common use of an enclosed airspace."

"**Indirect transmission**" is used when there is "intermediary involvement or action of another individual, object or substance"

I- Direct transmission

The simplest forms of direct transmission are those in which intimate contact between infectious and susceptible animals allows exchange of infectious particles through skin-to-skin contact or in excretions or secretions. Because the agent is exposed to the external environment only briefly, agents transmitted in this manner may be rather fragile. This form of transmission is more likely to occur within a social group (e.g., a family or territorial group) than among unrelated animals of the same species or between species, because of the greater frequency and intimacy of contact.

1- Close contact

a- Skin-to-Skin Contact.

Although some fleas, lice, ticks, and mites have developed alternate strategies for moving among hosts, most ectoparasites are dependent upon close physical contact for transmission. For example, *Sarcoptes scabei*, the cause of sarcoptic mange in wild canids.

b- Short-Range Airborne Transmission.

Transmitsion via aerosols or droplets is the principal method of transmission for many respiratory infections in which the agent leaves one animal on the expired air and enters another in inspired air. However, only droplets<5 micron in diameter are capable of remaining suspended in air for an extended period of time.

c- Secretions and Excretions.

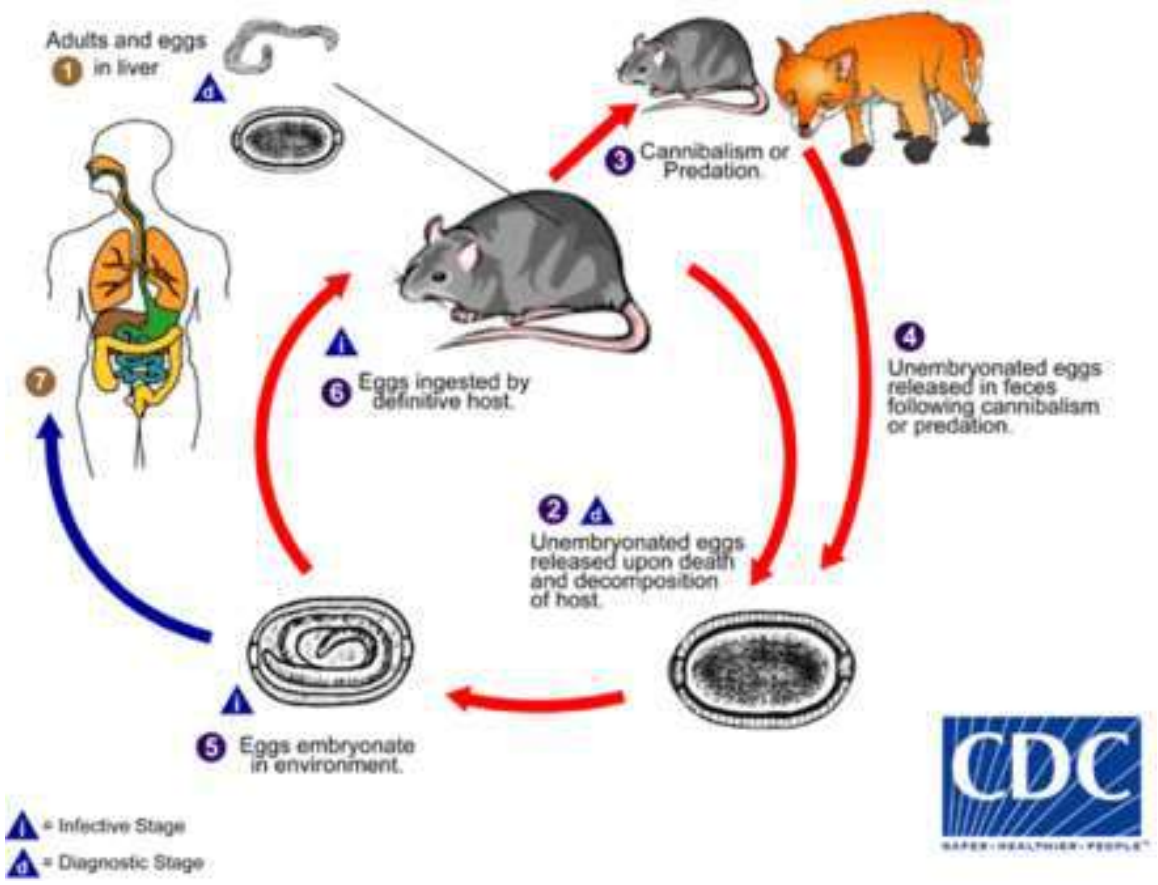
Secretions and excretions often contain many agents that can be transmitted through close contact. These might be inhaled in the form of aerosols generated by sniffing or, more commonly, ingested through licking and grooming, aggressive behavior, or from contamination of the immediate surroundings.

d- Sexual or Genital Discharges.

Transmission of bacteria of the genus *Brucella* among terrestrial mammals is primarily through contact with fetal fluids, vaginal exudates, and aborted fetuses. This takes the form of licking an infected fetus, placenta, or the genital region of an animal after abortion or parturition.

c- Contact with Carcasses

The carcass of an animal that died of infectious disease may contain massive numbers of infectious agents. Direct contact with carcasses is probably not a common route of intraspecific disease transmission. Cannibalism (intraspecific scavenging and predation) is probably infrequent as a method of disease spread, but it is important in the transmission of *Capillaria hepatica*, a nematode that occurs in rodents and less commonly in other species. The adults of this worm live and deposit eggs within the liver of the host, and the eggs remain in the liver until released by predation, decomposition, scavenging, or cannibalism. Cannibalism is a major method of transmission of this parasite among deer mice .



2- Contaminative Transmission.

The term “contaminative transmission” is used here for forms of direct transmission in which there is separation in time and/or space between the infectious individual and susceptible animals. For this to occur, the agent must be sufficiently robust to survive in the external environment. In many situations, some intermediary or “vehicle“ is involved. This might be water, air, inanimate objects, or a living organism of another species. In the latter case, participation by the other species is not required for transmission.

a- Long-Distance Airborne Transmission.

Most droplets originating from the oral or nasal cavities are only infectious within a short distance from their origin. However, particles <5 pm may remain in suspension for extended periods and may be carried long distances

b- Water

Many diseases are transmitted through contaminated water, and disease agents that are not normally thought of as being aquatic often survive longer in water than in the terrestrial environment The bacteria survive in water for days to weeks and accumulate near the surface of the water column.

c- Contamination of Food, Vegetation, Soil, and Other Abiotic Elements. Consumption of contaminated food and exposure to vegetation and soil contaminated by organisms, particularly those shed in feces, m common routes of transmission for many important diseases of wild animals, including diseases caused by viruses, bacteria, protozoa, and helminths.

e- Transmission by Other Species.

There are two forms of direct transmission that involve species other than the usual host for the disease. The first of these is exemplified by the carriage of some viruses on the mouthparts of mosquitoes .The second form of animal transport is through “paratenic hosts." These are hosts that become infected with the organism but in which reproduction of the organism does not occur. Paratenic hosts are not required, but they may enhance the probability of transmission. Paratenic hosts usually are only recognized for macroparasites.

II- Indirect Transmission

Indirect transmission requires the involvement of more than one species. The “other” species may be a vertebrate or an invertebrate, and more than two species may he required in some diseases. When two or more species are involved in the life cycle of a disease agent, the nomenclature used to designate the status of the different hosts is a bit problematic.

Examples:

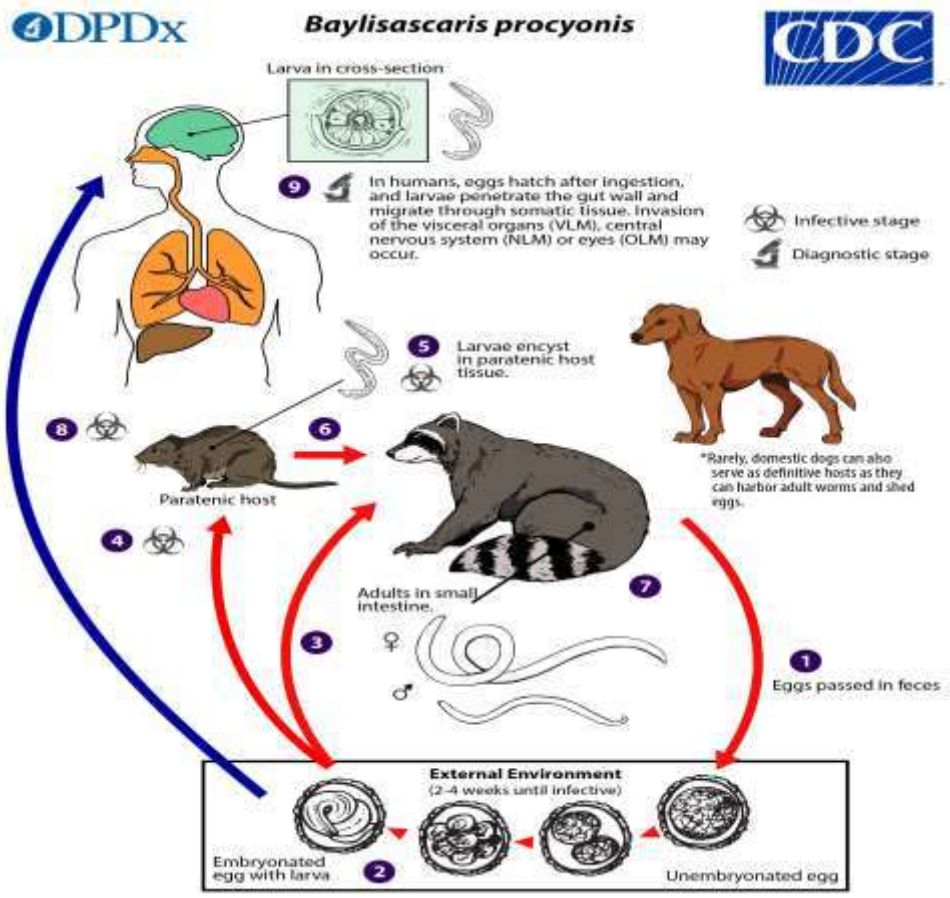
Disease agent	Definitive host	Intermediate host
Fasciola magna	Elk, deer	Snail
Echinococcus granulosus	Wolf	Moose, deer, elk
Disease agent	Wildlife host of concern	Vector

Lyme Disease *Borrelia burgdorferi*	Rodent, birds	*Ixodes scupularis, Ixodes ricinus*
West Nile virus	Wild birds	*Culex*

MULTIPLE ROUTES OF TRANSMISSION

Many infectious diseases have more than one route of transmission. The route that is most important may vary in different situations. This is obviously of great importance in understanding how a disease is maintained. As an example, the nematode *Baylisascaris procyonis* is transmitted by different routes to juvenile and adult raccoons. Adult worms live in the intestine and eggs are passed in the raccoon's feces. After a period of development in the external environment, the eggs are directly infectious if ingested by another raccoon. This is the route by which most young raccoons become infected. If eggs are ingested by animals of other species (usually a rodent or small bird), the eggs hatch and the larvae migrate through the animal's tissue (a process called "visceral larva migrans") and some reach the brain. The larvae do not complete development in these paratenic hosts, but the resulting debilitation

and/or death of the animal facilitates its being found and consumed by a raccoon. This is the route by which most transmission to adult raccoons is thought to occur.



ENTRY INTO THE NEW HOST

The third step in transmission is invasion and colonization of a new host. Much of what we consider to be inside the body is in fact directly connected with the outside. The most important points of entry for agents are via ingestion and inhalation. Less commonly, organisms enter through the mucous membranes of the conjunctiva, the urogenital system. and the skin. Some agents never penetrate through the epithelium lining the body's surfaces.

Some of these live on epithelial surfaces (e.g., *Giardia* spp. in the intestine), and others such as coccidia enter epithelial cells but do not penetrate more deeply into the body.

Mims et al. (2001) listed four general mechanisms by which agents enter the body.

1. Agents may have special mechanisms for attaching to and penetrating the body surfaces. These allow them to adhere to specific molecules, called receptors, in specific host cell membranes. (Receptors were not designed for disease-agent attachment, they serve other functions in the cell.) Some organisms can bind to more than one cell type.

2. Agents may be introduced through the skin by a biting arthropod or may penetrate directly as is done by some macroparasites such as the hookworms of seals and canids.

3. Agents may be unable to penetrate the body surfaces and depend upon some damage to a surface to allow them to enter. The damage may be a mechanical break in the skin or a mucous membrane, or a bite wound as used by the rabies virus.

4. Agents may enter when there is a local or generalized defect in the body's defenses.

Not all disease agents that reach a new host are successful in establishing an infection. The number of agents arriving at a new host usually is quite small and may be inadequate to overcome local defenses. In diseases caused by microparasites, a threshold number of organisms, the "minimum infectious dose," is required to result in infection. Little is known about the minimum infectious dose required for transmission of most diseases of wild animals, but the approximate infectious dose of some agents for humans is known, illustrating the degree of diversity. For example, *Salmonella typhi* ≤105 bacteria, *Shigella*

dysenteriae = 10 bacteria, Giardia lamblia = 10 cysts, and Mycobacterium tuberculosis = 1-10 bacteria.

The actual dose required to result in infection varies with the species, age, sex, genotype, and general condition of the animal, the particular strain of agent, the route of infection, and many other factors.

Wildlife Disease Management

INTRODUCTION

Disease is a natural part of any ecosystem; however, it is also a global problem for human health and wellbeing. Characterised as any impairment of normal bodily function, over 1400 diseases have been identified that directly affect humans, 60% of which are known zoonotics (Delahey *et al*. 2009). The cause of disease can come from an external source such as a virus, bacteria, fungi or protozoa or an internal dysfunction such as a cancer. But more diseases can affect humans indirectly, having damaging effects on agriculture, economics and biodiversity. These diseases and those that directly threaten human health become the target of management programmes. During the last century there has been mounting evidence and recognition of the role that wildlife play in the epidemiology of problem diseases, acting as a reservoir, vector or simply a victim (Tompkins & Wilson 1998; Artois *et al*. 2001). Human intervention to manage these issues can be problematic and complicated. This type of human-wildlife conflict is increasingly exacerbated as the world is developed and the interface between humans and wildlife intensifies via urbanization, agricultural intensification, habitat degradation and the changing climate (Tompkins & Wilson 1998). This review focuses

on the various management strategies and considerations for the control of problematic infectious diseases in wildlife with reference to relevant cases.

WHY MANAGE WILDLIFE DISEASE?

The decision to intervene with an ecosystem and manage a wildlife disease is made when the cost to the human way of life in considered too high, be this a risk to human health, economic well-being or the threat of loss of biodiversity through species extinction.

(1) Human health

Many diseases are worldwide public health concerns. Those that are zoonotic diseases can be transmitted to humans from domestic or wild animals. Mosquitoes transmit malaria through bites; rabies can be transmitted by domestic dogs and red foxes (*Vulpes vulpes*) amongst others toxoplasmosis (*Toxoplasma gondii)* which affects 500 million people around the world is transmitted from felines. Governments have to act to manage these threats to human health.

(2) Domestic animal health

Animal welfare legislation dictates that animals under our responsibility should be cared for fully, including the treatment and prevention of illness (OPSI 2006).

Livestock animals are economically valuable and can be threatened by a number of diseases. This is compounded by animal diseases being regarded as an unacceptable limitation to trade or movement (Thomson *et al*. 2004).

(3) Conservation of species

The protection of healthy ecosystems and their biodiversity are connected to human well-being for a sustainable future (Chapin *et al*. 2000). Pathogens and infectious diseases are of increasing concern for conservation as they pose a significant extinction risk for a number of endangered species, particularly those in reduced fragmented populations with reduced genetic resistance. The Ethiopian wolf (*Canis simensis*) and African wild dog (*Lycaon pictus*) are but two canid species that are threatened by rabies and canine distemper virus. The Ethiopian wolf has suffered at least two outbreaks of rabies in the last ten years, resulting in >70% mortality during each event, and long term demographic impact.

Prevention, control or eradication

The objective of a disease management program may be to

Prevent, control and eradiation

Prevention = proactive strategies that are aimed at averting the occurance of a disease into unaffected areas, individuals or populations (Wobeser 1994). Quarantine for domestic dogs travelling between countries has helped keep the UK a rabies free area.

Control = activities designed to reduce or maintain the prevalence of a disease to a

tolerable level. This includes finding the balance between cost and benefit to stakeholders with an understanding that some level of disease will persist with the consequence of continuous disease control management (Wobeser 1994; Artois *et al*. 2001).

Eradication = involves the total elimination of a disease, ensuring an absence of continued transmission. This would usually have a time limit and designated area and require extensive resources. For eradication to be a success the correct identification of all sources/reservoirs of infection most be known, which is not always as necessary for prevention or control strategies (Wobeser 1994; Delahey *et al*. 2009).

The choice of management level decided upon will depend on the particulars of each disease problem and more importantly, on the correct identification of the reservoirs of infection and budget available (Delahey *et al*. 2009).

	Control	Eradication
Definition	To reduce incidence to acceptable level e.g. malaria control	Total cessation of disease agent, e.g. Small Pox eradication
Objective	To reduce mortality and morbidity	To uproot the disease
Area of operation	In high incidence area	Total coverage
Duration of operation	Long follow up	Time limited
Economic aspect	Expensive	Cheap
Case finding, confirmation, Epidemiological investigation	Not important	Very important

MANAGEMENT STRATEGIES

(1) Manipulating host population size

Reducing infected and susceptible individuals in a population can help with density dependent transmission and contact between wildlife and domestic animal populations (Artois *et al* 2001). This can be achieved by culling or restricting the reproductive rate of the target population through fertility control measures.

(a) culling

Culling can be achieved by different methods, trapping, shooting, gassing or poisoning of infected domestic animals or wild animals (Artois *et al*. 2001). Culling has been used to attempt to control Rabies in the red fox throughout Europe, rabies in striped skunks Culling can also have important ecological side effects as removing a carnivore from a food web can induce competitive release resulting in an increase in other carnivores (Cross *et al*. 2007; Trewby *et al*. 2008).

(b) fertility control

As a non-lethal technique for managing wildlife populations, fertility control is less controversial than culling although a slower and more lengthy process. While it is used to control pest species .

As fertility control only reduces the number of new individual uptake into a population it does not deal with susceptibility to pathogens, the individuals' already diseased or further transmission. Smith & Wilkinson (2003) use fertility control combined with vaccination within their predictive model for controlling rabies in red foxes against alternative methods of vaccination alone or culling. Vaccination combined with fertility control was found to be more effective than vaccination alone. It is believed that the use of fertility control will increase the effectiveness of vaccination programs as it restrains the recruitment of new susceptible individuals to the target population (Delahey *et al*. 2009). As a means of reducing population levels, fertility control has advantages over culling as it does not remove individuals from delicate social structures.

(2) Immunization

Vaccinating individuals within a population also reduces the number of susceptible animals and controls the disease by controlling the threshold population level required for disease persistence (Artois *et al*. 2001). For the strategy to be successful a sufficient proportion of the population need to be vaccinated during a particular period of time. In fox vaccination programs models showed that where there was a sub-optimal number of vaccinated foxes this led to the slowing of natural spread of the infection but extend the duration of infection. In this case, increasing the number of individuals vaccinated would lead to the disappearance of rabies from the area (Artois *et al*. 1997; Tischendorf *et al*. 1998).

In Ethiopia domestic dogs are the known reservoir for rabies and are the most abundant carnivore, with population levels well above the threshold needed for rabies persistence. Vaccination of the domestic dog population was a management option in this case but proved problematic due to cultural attitudes of dog handling, a high population growth rate and missing out the families that translocate seasonally, proving the strategy to be logistically and financially difficult (Randall *et al*. 2006). In conjunction with this an emergency vaccination trial in Ethiopian wolves was necessary and restrained the spread of the rabies epidemic. Models suggested that only 20%-40% coverage of vaccination in the wolves would reduce their risk to extinction considerably. This protected individuals and whole packs from rabies infection and stopped the spread of the outbreak (Randall *et al*. 2006). However, failure of a vaccine to protect against

rabies has been recorded in wild and captive African wild dogs (*Lycaon pictus*) leading to the hypothesis that booster doses may be required (Randall *et al*. 2006).

(4) Behavioral modification

The change of behavior of humans, domestic animals or wild animals can be an effective and less costly strategy than other methods of controlling wildlife diseases.

West Nile Virus (WNV) infects humans, domestic animals and a large variety of wildlife. In the United States advice leaflets were distributed to the public with suggestions of precautions that could be implemented to help combat the mosquito born disease. These suggestions included the removal of any standing water to hinder mosquito reproduction (CDC 2009). In the fight against bTB, cattle-to-cattle transmission is a problem and changes in farm management such as bio-security provide an opportunity for solutions to transmission routes.

Changing the behavior of wildlife can consist of deterring them from areas where they come into contact with other susceptible animals. This could include dispersing animals more widely or using deterrent or scaring devices. In the USA hunters had recently taken to putting out large piles of feed to attract larger numbers of deer, however, in doing so they increased the contact and therefore the transmission of pathogens between the deer . In response the USDA encouraged hunters to scatter the feed patches and use smaller quantities, to disperse the deer and reduce transmission instances. Scaring devices can also be employed to deter deer away from cattle ranch feeding areas to reduce species-species transmission of pathogens .

(5) Combining strategies

The methods mentioned within this lecture all have their limitations and a number of wildlife disease management teams and researcher are now suggesting that integrated management strategies would be much more effective.

(6) Monitoring

Surveillance of disease in wildlife is poorly developed in most countries with few currently having their own wildlife disease surveillance network (Artois *et al*. 2001; Delahey *et al*. 2009). It is often the people on the ground, such as hunters or wildlife conservationists who report cases and remove sick animals (Artois *et al*. 2001).

The monitoring of disease in wildlife can encompass the use of sentinels. In the USA, wildlife surveillance programs identified relatively high disease prevalence in coyotes (*Canis latrans*), highlighting them as a potential sentinel species for detecting bTB prevalence in white-tailed deer (Atwood *et al*. 2007). Corvids and chickens have been used as sentinels for monitoring the progress of WNV across the United States (Komar *et al*. 2003; Brault *et al*. 2004).

The Devil Disease Project Team intends to monitor six sites including areas that are non-diseased, recently diseased and long term diseased. They will also monitor regions near the disease frontline with the aid of camera traps (Hawkins *et al*. 2006).

The Ethiopian Wolf Conservation Group intend on monitoring disease in the species by radio collaring one wolf in each of the packs, ensuring the rapid detection of any

outbreaks by observation of clinical signs and discovery of carcasses (Randall *et al*. 2006). Epstein (2001) and Simpson (2002) maintain that global climate change will have implications for ecosystems and their pathogens, increasing the chances of emergence and re-emergence of pathogens and their vectors in new geographic areas and hosts.

It is widely believed that a system for the routine monitoring of wildlife needs to be in place as an essential tool for future wildlife disease management with greater coordination between national agencies .