# Essentials of Disease in WILD ANIMALS

GARY A. WOBESER

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This book is dedicated to three mentors: A. Bruce Stephenson, wildlife biologist, who introduced me to field research and showed me that farm boy skills were applicable to working with wild animals; Lars H. Karstad, wildlife disease specialist, who welcomed me to the then new field of wildlife diseases and gave me the freedom to explore and make mistakes; N. Ole Nielsen, veterinary pathologist and "Green Dean," who has been a tireless advocate for incorporation of environmental thinking into veterinary medicine.

# Contents

# Preface

I began this book because I perceived that many of those who are being called upon to work with disease in wild animals lack experience or training in the general features of disease as they relate to wild animals. 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. Theoretical ecologists, mathematicians, and population biologists can model how disease should behave quantitatively within populations, but they may have little experience with the medical aspects (physiology, anatomy, immunology, pathology) of disease, or with the practicalities of wildlife management.

There is no introductory level book about disease in wild animals that deals with basic subjects such as the nature of disease, what causes disease, how disease is described and measured, how diseases spread and persist, and the effects of disease on individual animals and populations. It is presumptuous of any individual to attempt to deal with all aspects of disease, and my intent is not to try to discuss any particular disease in detail.

This book developed from a graduate class in wildlife diseases that I have taught periodically over the past 30 years. The class began as a survey of the important diseases of western Canadian wildlife in which I dealt with viral, bacterial, fungal, parasitic, and toxic diseases of specific species. My approach was a rather standard veterinary one, concentrating on individual causative agents and their effect on the individual animal, in terms of the clinical disease and pathology that they produced. I initially placed relatively little emphasis on why disease occurred, or on the complex interactions that occur among disease agents, the environment, and host populations.

Embedded in my early approach was the notion that disease was somehow unique and different from other ecological factors and, as such, had more to do with medicine than with ecology. I also must admit that disease often was treated as a harmful phenomenon that should be "fought" or "managed" at every opportunity. (This was a residue of evangelical zeal from my veterinary training!) As time advanced, my interest and the class content became concerned more with general aspects of health in wild animals, such as how and why various diseases occur in wild animals, why animals and parasites appear to get along better in some situations than in others, and the effects of disease on populations rather than on individuals. There was a growing realization on my part that disease is one ecological factor among many and that disease can never be considered satisfactorily in isolation.

I have been fortunate to have been influenced during my career by many wildlife managers and ecologists. Some took a very pragmatic approach to disease while others tried to put disease into a larger ecological and evolutionary framework. From the first group, I have learned about natural history, observation, and the practicalities of working with wild animals. From the second group, I have learned that the features we observe in animals—such as their behavior, reproductive strategies, habitat selection, and susceptibility to various mortality factors—should be considered in terms of lifetime fitness, selective advantage, differential survival, and evolution.

# Acknowledgments

I am indebted to the many colleagues and students who have contributed information and inspiration for this book. I am very grateful to my departmental home for allowing me to pursue an interest in wildlife diseases for many years and to the Department of Pathobiology, Ontario Veterinary College, University of Guelph for its hospitality while I was preparing the manuscript. I particularly thank my wife, Amy, for her continuous encouragement and enthusiasm.

# Essentials of Disease in Wild Animals

# 1
# Introduction

The study of disease in wild animals is a relatively new scientific discipline 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 (McCoy 1911; McCoy and Chapin 1912), avian botulism in waterfowl (Kalmbach and Gunderson 1934), and rinderpest in African antelope (Carmichael 1938) and Elton (1931) reviewed epidemic diseases of wild animals. Formation of an international scientific body, the Wildlife Disease Association, in 1951 marked the beginning of more organized study of disease in wild animals, 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. A major factor has been a burgeoning awareness of the involvement of wild animals in infectious diseases of humans. Despite earlier optimism that infectious diseases in humans could be eliminated or controlled, it is now clear that infections have not been vanquished. "Emerging infectious diseases" have become a medical growth industry. New human diseases continue to be discovered and many old foes have returned with a vengeance because of environmental and demographic changes, declines in public health activities, and evolution of resistant organisms. 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). Many other important human diseases in addition to those shown in table 1.1, 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. Many well-established human diseases that continue to cause problems including plague, tularemia, Lassa fever, rabies, and influenza are linked directly to wild animals.

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). 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 and leash laws, 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.

**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 |

*Note:* An emerging disease is one whose incidence in humans has increased recently or that threatens to increase in the near future. Included are previously unrecognized infections, new infections as a result of a change in a previously recognized causative agent, infections spreading to new areas or populations, and old infections that are reemerging because of deterioration in control or public health measures.

Some diseases that have been eliminated from domestic animals continue to occur in wildlife. For instance, cattle in most of North America are free of brucellosis caused by *Brucella abortus* but remnant pockets of infection in bison and elk in a few locations are considered to be a risk to national eradication programs.

Similarly, the occurrence of Newcastle disease in double-crested cormorants is considered a risk to North American poultry from which the disease has been eliminated (Kuiken 1999). The persistence of disease in wild animals has stymied efforts to eradicate some diseases of domestic livestock. The best documented of these is bovine tuberculosis caused by *Mycobacterium bovis*. Efforts to eradicate this disease in domestic cattle have stalled in England and Ireland because of tuberculosis in badgers, in New Zealand because of the disease in brushtail possums, and in parts of the United States and Canada because of infection in wild deer and elk. New disease problems involving wild animals continue to be discovered, for example, paratuberculosis, a disease of domestic ruminants caused by *Mycobacterium paratuberculosis*, is now known to occur in a wide variety of nonruminant wild animals that may pose a risk to domestic livestock (Beard et al. 2001; Daniels et al. 2003).

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 (Daszak et al. 2000; Cleaveland et al. 2001). 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 (Atkinson et al. 1995), the near eradication of the black-footed ferret by canine distemper (Williams et al. 1988), the possible role of chytrid fungi and iridoviruses in declining amphibian populations worldwide, avian vacuolar myelinopathy in bald eagles and other species (Fischer et al. 2003), and rabies and canine distemper in Ethiopian wolves (Laurenson et al. 1998).

Wildlife managers have been forced to become more involved with disease for several reasons. There has been considerable pressure to manage wild species as part of control programs for diseases that may spread to humans and livestock, such as rabies, *Echinococcus multilocularis* infection, bovine

**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.

tuberculosis, and West Nile virus infection. Currently, there is considerable public concern and pressure for action in North America to deal with the expanding known geographic distribution of chronic wasting disease in deer and elk. Managers also have become concerned about the effects of disease on wild species per se. Recent examples of disease-related phenomena that appear to have had a serious effect on wild animals include a precipitous population crash of vultures in Pakistan caused by poisoning with an antiinflammatory medication used widely in cattle (Oaks et al. 2004); population declines of house finches as a result of eye infections caused by the bacterium *Mycoplasma gallisepticum* (Dhondt et al. 1998); massive die-offs of seals caused by morbillivirus infection (Kennedy 2001); loss of lions in the Serengeti to canine distemper (Roelke-Parker et al. 1996); extirpation of the Allegheny wood rat in part of its range by a raccoon parasite (Logiudice 2003); and the spread of bovine tuberculosis in African buffalo, other ungulates, and carnivores in Kruger National Park (Caron et al. 2003). Wildlife managers also have become more aware that their actions can contribute to disease problems ranging from simple things such as muscle injury (capture myopathy) as a result of capture and handling animals to the introduction of new diseases as a result of translocating diseased animals.

Toxicologists have been interested in wild animals for many years and effective control measures have been developed for some diseases such as those caused by organochlorine insecticides, mercurial seed dressings, and lead shot. Some of the emphasis in wildlife toxicology has shifted from the more overt poisons to compounds, such as endocrine-disrupting chemicals (Ottinger et al. 2002), that may have sublethal effects on immune function, behavior, and reproduction. Contaminants of various types often appear to interact with other potential disease-causing agents, closing the gap between infectious and noninfectious diseases. For instance, during an outbreak of phocine distemper (caused by a morbillivirus), seals from the heavily polluted Baltic Sea appeared to be most severely affected (Kennedy

1990) and seals fed contaminant-laden fish from the Baltic had reduced immune function compared to seals fed fish from the less-contaminated Atlantic (Swart et al. 1994). Contaminants also may interact synergistically with other mortality factors such as predation (Relyea 2003).

Interest in disease in wild animals also has increased for an entirely different reason. There has been an explosion of academic attention to various aspects of disease in wild animals by ecologists, behaviorists, population biologists, and modelers. 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 (Nichol et al. 1993) 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 (Mills and Childs 2001). 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. For instance, it is unlikely that massive die-offs of waterfowl similar to those that have occurred during the past three decades as a result of avian cholera would have gone unrecognized earlier in the century, but the disease was not known to occur in wild birds in North America prior to 1943 and widespread large outbreaks have only been recognized since the mid-1970s (Friend and Franson 1999). Eye infections of house finches caused by *Mycoplasma gallisepticum* appear to be a completely new disease that has spread widely in North America since it was first recognized in 1994 (Dhondt et al. 1998). Canine parvovirus 2, which infects several wild canid species, appears to have arisen from a virus of cats and spread rapidly in domestic dogs and wild canids around the world about 1978 (Barker and Parrish 2001). 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 mingle with humans and traditional domestic animals in unexpected ways, as occurred in the introduction of monkeypox 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 (Laurenson et al. 1998), African wild dog (Alexander and Appel 1994), lions in the Serengeti (Packer et al. 1999), and seals in Lake Baikal (Mamaev et al. 1995).

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 waterbirds 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 as this is written, the pandemic strains that affected humans around the world in the 20th century resulted from reassortment that occurred in pigs infected with both a strain from humans and a strain from birds (Kida 2003). Thus, pigs served as an intermediary between birds and humans. The combination of wild waterbirds, intensive poultry production, intensive pig production, and dense human populations that occurs in some areas of the world provides the ideal milieu for generation of new influenza viruses.

The discussion above dealt with infectious diseases that are shared by people, domestic animals, and wild animals. Disease also is likely to have a greater direct effect on wild species in the future. New agricultural and industrial practices will result in exposure of wild animals to new contaminants, often with unexpected results. For example, introduction of "second-generation" anticoagulants for control of rats and mice has resulted in secondary poisoning of carnivores. Acidification of soils by acid rain may be leading to calcium deficiency in passerine birds and cadmium poisoning of mammals. Some diseases that are unimportant currently are likely to become significant because of continued human pressure on natural habitats. Diseases are like weeds in that both thrive in disturbed environments. Just as weeds have great difficulty gaining a foothold in an established forest or grassland, diseases have difficulty being perpetuated in stable systems, but both weeds and some forms of disease quickly invade and proliferate following disturbance.

Human history is replete with examples in which pestilence has followed social and environmental disruption. It is useful in this regard to compare human and wild animal populations. Improvements in sanitation, shelter, nutrition, and water supply have been central to the control of important infectious and noninfectious diseases of humans. When these improvements are disrupted by social or natural disasters, disease follows rapidly. Few wild animals live in undisturbed environments or in circumstances in which the level of sanitation, shelter, nutrition, or the quality of water have improved. Diseases such as measles have emerged in epidemic form in human populations as a result of the large, dense populations that occur in cities. Refuges on which wild waterfowl are crowded together for months and artificial feeding areas on which some wild species congregate seem very like cities to me, but they are cities without the benefit of sewage disposal, clean water, and the immunization programs that protect us from many diseases. It should not be surprising that avian cholera has emerged in the past few decades on these refuges, or that tuberculosis has become a self-sustaining infection among artificially fed white-tailed deer (Miller et al. 2003), or that salmonellosis occurs among passerine birds congregated at bird feeders (Daoust et al. 2000; Refsum et al. 2003).

## STUDY OF WILDLIFE DISEASES: AN INTERFACE AREA

The study of wildlife diseases is an interface area (fig. 1.1) 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. For example, I am part of a group struggling to develop an appropriate strategy for the management of bovine tuberculosis in elk and deer within and outside a national park, and in cattle in the vicinity of the park. In developing this plan, wildlife managers, conservation ecologists, geographers, agriculturalists, foresters, rural sociologists, veterinarians, modelers, biometricians, historians, and laboratory scientists have made a significant contribution, because their skills are complementary.

It is my perception that many of those who are being called upon to work with disease in wild animals lack experience or training in the general features of disease as they relate to wild animals. 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

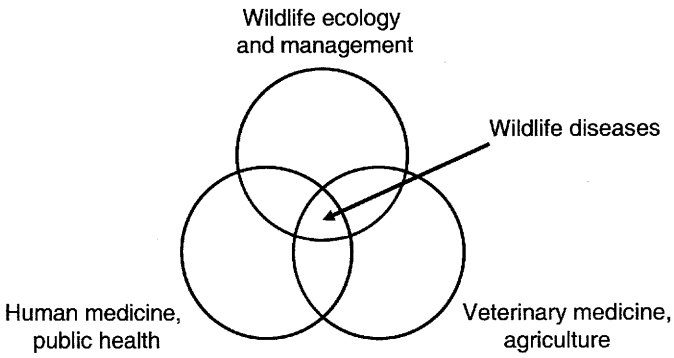


**Fig. 1.1.** Disease in wild animals occurs at the interface among human medicine, veterinary medicine, and ecology.

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. 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.

In the chapters ahead, I will discuss the nature of disease, what causes disease, how disease is described and measured, how diseases spread and persist, and the effects of disease on individual animals and populations. It is presumptuous of any individual to attempt to deal with all aspects of disease, and my intent is not to try to discuss any particular disease in detail. I will use examples from wild species to provide basic information about the nature of disease in its many forms and the range of factors that result in disease. I hope to provide some familiarity with the vocabulary of disease (e.g., the difference between infection and disease, and between prevalence and incidence), some understanding of the intricacies of detecting disease (e.g., the specificity and sensitivity of tests), and the difference between humoral and cell-mediated immunity. I will stress that all disease, regardless of cause, begins at the cellular level, and that changes at the cellular level result in functional changes in the individual that have population effects. If nothing else, I hope that the reader will gain an appreciation that disease is one environmental feature among many that affect animals, and that it is impossible to understand disease without considering the interactions among disease agents and with other factors such as nutrition, predation, climate, and reproduction.

Over the past few decades, technical books have appeared that describe many individual diseases that occur in wild animals. Some deal with a single species, such as *Diseases and Parasites of White-tailed Deer* (Davidson et al. 1981); groups of related species, such as *Diseases of Wild Waterfowl* (Wobeser 1997); or larger taxa, such as *Infectious Diseases of Wild Mammals* (Williams and Barker 2001) and *Parasitic Diseases of Wild Mammals* (Samuel et al. 2001); or diseases that occur in a geographical area, for example, *Parasites and Diseases of Wild Birds in Florida* (Forrester and Spalding 2002). These are excellent references on clinical, epidemiological, and pathologic features of important diseases with a heavy emphasis on game species and on diseases that cause conspicuous mortality. *Ecology of Infectious Diseases in Natural Populations* (Grenfell and Dobson 1995) and *The Ecology of Wildlife Diseases* (Hudson et al. 2001) deal with more general aspects of infectious disease in wild animals with a particular emphasis on mathematical aspects of disease in populations as demonstrated in models.

A feature of most books that deal with wildlife disease is a distinct separation of subject matter based on causation. Noninfectious diseases caused by poisons and contaminants are almost never discussed together with infectious diseases. Noninfectious diseases caused by factors such as nutrition, aging, and genetic defects have received almost no attention. Diseases caused by living organisms usually are separated into those caused by "little" organisms (viruses, bacteria, fungi) and those caused by larger organisms that are visible to the naked eye such as fleas, lice, and various worms. (Protozoa seem to float between the two main groups.)

Diseases caused by the little organisms (*microorganisms*) are usually termed *infectious,* and diseases caused by the larger animals are generally referred to as *parasitic*. However, at the ecological level, all of the infectious agents are parasites and diseases caused by both big and little organisms are infectious.

I have tried to incorporate both infectious and noninfectious conditions in the discussion throughout this book, because the two types of disease occur together in nature, because the basic principles are the same, and because I believe that there is merit in trying to use the same ecological construct for looking at disease of all types. Wild animals seldom are exposed to just one disease-causing agent at a time, or to just infectious or just noninfectious factors. As a simple example, Pawelczyk et al. (2004) examined one tissue (blood) from common voles by one method (light microscopy). They identified at least five different microscopic organisms, including some that generally are classified as parasites and some that are considered infectious agents. At the instant that they were sampled, about 50% of the voles had two agents visible in their blood and 1% was infected concurrently with four different agents. One can assume that these voles also were infected with a range of infectious agents in tissues other than blood, that they had agents that were not visible with the light microscope, and that they carried residues of potentially harmful substances in their tissues, because that is the "usual" situation in wild animals. The voles may or may not have been suffering dysfunction as a result of these agents.

Different types of disease-causing agents often interact and many diseases are caused by combinations of agents rather than by a single factor. Noninfectious factors affect the ability of animals to respond appropriately to infectious agents and infections may compound or confound the effects of abiotic factors. As a diagnostic pathologist, I often have been confronted with dead animals that had elevated residues of several classes of chemicals, greater than usual numbers of worms, evidence of exposure to one or more viruses, infection with potentially damaging bacteria such as *Salmonella* spp., and evidence of malnutrition. In such situations, selecting any one of the chemicals, parasites, bacteria, viruses, or nutrition as the main cause of the problem is naive.

I have tried to insinuate some basic life history theory in various parts of this book. The most important single feature is that natural selection favors evolution of physiological mechanisms to ensure optimal allocation of limited resources to competing activities. "Success" is the result of making the most appropriate trade-offs, and disease is all about resources and trade-offs. Infectious agents and host animals must both make trade-offs. Bacteria, viruses, and larger parasites trade off the amount of nutrients that they can extract from an animal against the probability of being transmitted to another animal. If they are too greedy and cause too much damage to their host, they may compromise their own survival and fitness. Similarly, host animals make many trade-offs related to disease (e.g., should they graze in a lush area where worm larvae are abundant or move to another area where parasites are less numerous but the plants are less nutritious? Should they use resources to resist a disease or put those resources toward growth and reproduction in the hope that the disease won't occur or that they can survive its effects? Should they allocate resources toward reproduction now if doing so compromises their resistance to disease and decreases the chance of surviving to reproduce again later? How many resources should they allocate to resisting the effects of one disease agent compared to those devoted to defense against another agent?).

Most of my experience has been in the cooler parts of North America so that many of the examples I use reflect my familiarity with that environment. I have tried to incorporate references to work dealing with experimental systems for studying basic aspects of disease and mathematical modeling of disease situations, because I believe that theory developed by laboratory studies of creatures such as *Daphnia* and field studies such as those of wild rodent populations infected with cowpox virus are relevant for understanding and management of problems such as West Nile virus infection and bovine tuberculosis.

From time to time, I will make reference to Aldo Leopold, the "father" of wildlife management in North America, because many of his views on the place of disease in wild animal ecology, as expressed in *Game Management* (Leopold 1933), remain relevant. When referring to research on disease he wrote, "It is a pity that the narratives of scientific exploration in this field—as fantastic a romance as any Arabian Nights—should either be masked by such technical verbiage as to mean nothing to the thinking layman, or translated for the popular press in such kindergarten terms as to be no longer true." My sincere hope is that I can avoid either extreme in discussing disease.

## SUMMARY

- The study of disease in wild animals is a recent phenomenon.
- 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.
- 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.
- The study of disease in wild animals must be multidisciplinary because of its complexity.
- 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.
- 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.

# 2
# 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. For most people who live in an urban setting, reference to disease usually relates to the human condition, as in "she died of heart disease," "alcoholism is a disease," or "gum disease is a serious problem that needs attention." I find it interesting that the examples that came to mind while writing the above were all noninfectious entities related in some way to lifestyle. This reflects my perspective as a member of a society in which most infectious causes of human disease have been removed from everyday thought. In contrast, if I were writing this book from the perspective of someone dealing with human ailments in some of the poorest parts of the developing world, the examples that would spring to mind would be diseases resulting from communicable infections, parasitism, malnutrition, and perinatal conditions (Murray and Lopez 1997).

If one lives in a rural community, many everyday references to disease relate to livestock or to crops. In veterinary medicine, one also sees a disparity in the type of disease that is important based on the purpose for which the animals are kept and the level and intensity of management of the animals. The diseases of pet animals (dogs and cats) are similar to those of their owners, with the added factor of many genetic disorders related to human selection for traits that have negative survival value. The owner of a large herd of intensively managed dairy cows usually is most concerned about so-called production diseases. These are conditions that result in decreased milk yield or lower conversion of feed into milk, or that extend the time period between calves from each cow. This same type of production disease is important in intensively managed pig and poultry operations. Most of these conditions, as in diseases of humans in affluent societies, are related to the lifestyle of the cows, pigs, and chickens. However, the animals have little choice in the food they eat, the amount that they exercise, or the company that they keep, unlike their human counterparts. In contrast, many of the disease problems of less intensively managed livestock such as beef cattle or sheep living on range relate to various infectious agents, poor nutrition, and intoxications from plants. The ecological concept of *fitness* as it relates to lifetime reproductive success has no meaning in most domestic species, because the animals seldom are allowed to live their full life span or to reproduce at will.

Because the perception of what constitutes disease is highly variable, it is difficult to find a definition for the word that is inclusive enough to encompass both the irritation of receding gums (i.e., "gum disease") and 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 as-

sume 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 hare 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 unparasitized hares and that parasitism could be an important component in the ecology of hares and of their evolution.

A similar example is the severely emaciated coyote that has stopped hunting and has removed most of its hair coat in a frantic attempt to reduce the intense irritation caused by *Sarcoptes scabei* mange mites (fig. 2.1). When this distracted, starving animal wanders into the path of a passing automobile or ventures into a farmyard and is killed by the dog, should its death be attributed to disease or to simple bad luck?

Because disease comes in many forms with degrees of severity, and has many causes, I think it can be defined most adequately in terms of the effect on normal functions of the individual. The definition that I prefer is that disease includes "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 1981).

**Fig. 2.1.** Coyote with severe hair loss as a result of infestation with the mange mite *Sarcoptes scabei.* Severely infected coyotes are usually emaciated and many have secondary bacterial skin infection. Some die of starvation and others die as a result of misadventure (hit by car, killed by dog, shot by farmer) because of abnormal behavior.

Implicit within this definition are four 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. For instance, a condition that results in reduced milk production by female elk so that their calves grow less well is disease, as is infestation with mites that causes a bird to be less attentive to its nest and, hence, results in poor reproductive success. The animals did not die of disease in either of these examples. I find it easiest to think in terms of a continuum between two endpoints: absolute health (a state in which all functions are optimal) and death, which occurs when functions are so severely compromised that life is impossible (fig. 2.2). Between these two points there is a region of