ONE

Introduction

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The study of wildlife diseases encompasses the health, disease, and fitness of wildlife, and the broad range of factors that can potentially affect their well-being. These factors include a wide array of infectious organisms such as helminths, arthropods, and microorganisms, as well as toxins, traumas, metabolic dysfunctions, genetic problems, and habitat fragmentation. Such factors not only act individually, but also may interact synergistically in complex ways to affect wildlife health. In this first chapter we give a brief summary of the basis for the emergence of wildlife diseases as a discipline and provide some key general concepts used throughout this text.

WHY STUDY WILDLIFE DISEASES

A focus on the health and well-being of wildlife themselves is of relatively recent origin. Historically, much of the initial interest in the health and diseases of wildlife stemmed from other concerns, particularly human health and the health of domestic animals (Friend 1976, Gulland 1995, Simpson 2002). In time, more direct interest emerged in the wildlife themselves, with an effort to gain a more complete biological and ecological understanding of how diseases interact with host populations; this is exemplified by the emergence of wildlife health and conservation medicine as distinctive disciplines.

More broadly, because parasitism is such a common mode of life, with parasites comprising the majority of species on earth (Zimmer 2000), there is considerable interest in understanding the basic biological relations parasitism entails, so as to better understand evolutionary history and ecological principles. In conventional Darwinian theory, natural selection adapts creatures to their immediate local environments through a process of specialization, operating to produce features that reduce organismal flexibility for future evolution; for example, specialization through simplification and loss of structures among helminth parasites often has been extreme (Gould 2002). Wildlife provide important models to better understand key host–parasite relationships.

In recent years, an increased sense of human responsibility toward the natural world, including a greater concern for wildlife, has emerged. Two examples of these concerns are the growing interest among the public in wildlife rehabilitation, as well as concerns over environmental issues such as oil spills and other forms of pollution. In recent years, increased tensions in global political relationships and a desire for greater national and international biosecurity (Dudley 2004) have further stimulated interest in a number of wildlife diseases.

Human Health

Public health risks were an early concern, and they remain a significant source of interest in the study of wildlife diseases (Beran and Steele 1994, Ashford and Crewe 2003). These interests have been focused primarily on wildlife diseases to which humans are susceptible, such as rabies, bubonic plague, avian influenza, Lyme disease, hantavirus pulmonary syndrome, and West Nile fever in North America. Such diseases transmitted between humans and other vertebrate animals are termed *zoonoses* (sing. zoonosis) (Soulsby 1974). We apply the term zoonosis as including (a) diseases common to both humans and nonhumans and which may be transmitted from nonhumans to humans or from humans to nonhumans, (b) diseases that are transmitted to humans and for which humans typically are a dead-end host (zooanthroponoses), and (c) diseases transmitted from humans to nonhumans and for which nonhumans typically are a dead-end host (anthropozoonoses). Interestingly, in Russian literature the opposite definitions are given for each term (Bender 2007). Estimates for the number of zoonotic diseases range from as low as 100 (Benenson 1990) to as high as 3,000 if all 2,000 serotypes of *Salmonella* spp. are counted as separate species (Beran and Steele 1994, Ashford and Crewe 2003), with new zoonotic pathogens being described every year. In one detailed count, an estimate of 816 zoonotic pathogens was made after omitting the numerous *Salmonella* spp. serotypes (Woolhouse and Gowtage-Sequeria 2005).

Along with their health impacts, some zoonoses have had particularly significant sociological impacts. Bubonic plague, caused by the bacterium *Yersinia pestis,* is considered to have given rise to at least three major world-wide epidemics (Stenseth et al. 2008) and to be one of the greatest natural disasters in human history (Fig. 1.1). In the fourteenth century alone, it is estimated that plague caused or contributed to the death of one-fourth of the western European population and of 2 million persons in England alone (Poland et al. 1994). Equally important was the nearly complete social disruption that accompanied many plague epidemics, with consequent shifts in world politics and power (McNeill 1977). On a historical note, the old children's nursery rhyme "Ring-around-the-Rosie" sometimes has been described as a poem stemming from plague epidemics. However, this appears to be fallacious, as the poem was not recognized until the eighteenth century, long after the most significant plague epidemics in humans; also, the poem has many variations that contain little apparent connection to plague (Munro 1996) and include one proposed to depict a smallpox epidemic (Glickman 1987).

Rabies is another disease that has had a significant impact on human cultures.

FIGURE 1.1 Plague cemetery in Nürnberg, Germany. Headstones often were laid on top of graves to prevent wild pigs and other animals from scavenging the dead (photo by R. Botzler).

The "big bad wolf" from the Little Red Riding Hood fairy tale may play on the fear many of European ancestry held for wolves *(Canis lupus);* this fear has been traced to a severe wolf rabies outbreak in Europe during the 1760s (Clarke 1971). However, in a more extensive study, Linnell et al. (2002) considered most wolf attacks on humans in France during the 1760s to be predatory rather than rabies-induced. The authors cite a number of reported wolf attacks on humans, especially in Finland, France, and Estonia, and more recently India, Russia, and additional regions of Asia; but they also assert that attacks by normal, healthy wolves are quite rare and unusual, and have not been reported in North America (Linnell et al. 2002).

Public health concerns have played an important role in the study of many other wildlife diseases. For example, most emerging human diseases have been identified as zoonotic (Lederberg et al. 1992); one estimate is that about three-fourths of the emerging diseases in humans are zoonoses (Taylor and Woodhouse 2000). Many emergent diseases in humans have followed changes in habitat or populations of wild hosts, with rodent species frequently being of special concern (Mills et al. 1994, Poss et al. 2002). Hantavirus infections and avian influenza both can produce significant human mortality and loom as potential sources of serious zoonoses.

Besides giving insights into the sources and risks of zoonotic diseases to humans, the study of diseases in wildlife also may provide models for better understanding similar human diseases. Alternatively, the understanding of human diseases can provide important insights into diseases transmitted to nonhumans (anthropozoonoses), including other primates (Cranfield et al. 2002).

Domestic Animal Health

In addition to disease agents shared with humans, wildlife also share many disease agents with domestic animals. Rinderpest (literally "cattle pestilence"), caused by a morbillivirus closely related to the human measles virus, was considered a great scourge among domestic cattle and wildlife in Africa (Branagan and Hammond 1965, Plowright 1982) before its eradication (Anonymous 2011). The impacts of this disease on imported cattle were so severe that strenuous efforts were made to contain infections; these efforts included establishing belts of immune cattle in key habitats, and even constructing a 265-km barrier fence between Lake Tanganyika and Lake Nyassa in Africa, with a 40-km game-free strip maintained on each side of the fence by professional shooters (Plowright 1982, McCallum and Dobson 1995). This led to considerable wildlife mortality during the ongoing slaughter of animals in this game-free strip; the fence further disrupted the natural daily and seasonal migration patterns for wildlife between key habitats.

In addition to the concern for humans, rabies also may have a considerable impact on wildlife and domestic animal populations (Beran 1994). Rabies in vampire bats *(Desmodus rotundus)* occurs from tropical Mexico to northern Argentina and Chile, and the disease has led to direct losses of millions of dollars annually among cattle, as well as additional losses through reduction in production and secondary infections among livestock (Kverno and Mitchell 1976). Rabid bats also have contributed considerably to public health costs for rabies control and postbite prophylaxis of humans. The environmental and ecological effects of killing vampire bats as a means of control have not been assessed.

Avian cholera, caused by the bacterium *Pasteurella multocida,* is a serious disease among both domestic (Heddleston 1972) and many wild birds, including migratory wildfowl (Botzler 1991, Samuel et al. 2007). In North America, the first known epizootics among wildfowl occurred in Texas (Gordus 1993) and California (Rosen and Bischoff 1949, 1950) and were associated with exposure to carcasses of domestic chickens that had died from avian cholera. However, despite these historical connections and an ongoing concern that wild birds may be a source of infection for domestic birds, there is little direct evidence for consistent transmission of *Pasteurella multocida* between wild and domestic birds (Snipes et al. 1988, 1989; Christiansen et al. 1992).

Many helminth parasites and protozoa also are shared between domestic animals and wildlife (Fig. 1.2). These include lungworms and intestinal nematodes, tapeworms and flukes among mammals (Longhurst et al. 1952, Soulsby 1968, Dunn 1969, Fraser and Mays 1986), as well as a number of intestinal parasites among domestic (Soulsby 1968) and wild (Wehr 1971) birds.

Although interest in shared diseases between wildlife and domestic animals initially stemmed from veterinary concerns about the role of wildlife as sources of diseases for domestic animals, it also is important to note that wildlife can be adversely affected by diseases acquired from domestic animals. Recent epizootics in African mammals of both canine distemper (Harder et al. 1995, Roelke-Parker et al. 1996, Carpenter et al. 1998) and rabies (Cleaveland and Dye 1995) followed transmission of these viruses from domestic dog reservoirs in conjunction with increasing encroachment of human populations on wildlife reserves (Poss et al. 2002). As an added concern, some of the emerging infectious diseases in both wildlife and domestic animals also are zoonotic (Mahy and Brown 2000, Friend et al. 2001, Daszak and Cunningham 2002, Kahn 2006).

FIGURE 1.2 A sheep dying from effects of liver flukes *(Fasciola hepatica)* in habitat typical of the snail intermediate host (Courtesy of W. Frank, Universität Hohenheim, Germany).

Some diseases intersect with public health, domestic animals, and wildlife. Influenza, caused by a myxovirus, is an example. Wild ducks, geese, shorebirds, and domestic pigs are important reservoirs for these viruses and can transmit them to domestic fowl (Slemons and Brugh 1994, Acha and Szyfres 2003, Fouchier et al. 2005). Human cases usually occur after exposure to infected domestic animals such as pigs and fowl, in which viral strains have undergone a genetic recombination (Slemons and Brugh 1994, Alexander and Brown 2000); human cases of the highly pathogenic avian influenza H5N1 have been tied to domestic fowl.

Wildlife Health

While research addressing the role of wildlife diseases among humans and domestic animals has occurred over many years, a focus

FIGURE 1.3 An American coot *(Fulica americana)* dying from avian cholera *(Pasteurella multocida* infection). Note the convulsions and torticollis ("twisted neck").

on wildlife diseases with a specific concern for the wildlife themselves has developed more recently, emerging first as a formal discipline about 1951, with the founding of the Wildlife Disease Association. Initially, most available information among interested professionals was transmitted through *the Wildlife Disease Association Newsletter,* which was replaced with the *Bulletin of the Wildlife Disease Association* in 1965, which, in turn, was continued as the *Journal of Wildlife Diseases* after 1970. Several books emerged during and after the 1960s that also helped establish wildlife disease as a distinct discipline (McDiarmid 1962, 1969; Davis et al. 1970, 1971; Davis and Anderson 1971; Page 1976; von Braunschweig 1979; Davis et al. 1981; Fowler 1981; Wobeser 1981; Edwards and McDonnell 1982; Hoff and Davis 1982; Fairbrother et al. 1996; Samuel et al. 2001; Williams and Barker 2001; Majumdar et al. 2005; Wobeser 2006; Thomas et al. 2007; Atkinson et al. 2008).

Most early concerns about wildlife diseases among biologists and managers were focused on major mortality events or mortality factors affecting the management of economically important game species (e.g., waterfowl, ungulates, upland game) (Fig. 1.3). In recent years, greater emphasis has been placed on better assessing the role of parasites and diseases on general fitness (survival, fecundity, mate selection) for all wildlife species. Concern for diseases in threatened and endangered species, and in relocation and translocation programs also has increased. Many disease investigations in conservation programs continue to be focused on high-profile species that have undergone a sudden demographic crash (Munson and Karesh 2002).

Recent emerging infectious diseases of wildlife include the chytrid fungus *(Batrachochytrium dendrobatidis)* (Daszak et al. 2004), white nose syndrome *(Pseudogymnoascus [Geomyces] destructans)* (Blehert et al. 2009), and devil facial tumor disease (Hawkins et al. 2006). Emerging wildlife diseases can have a particularly severe impact on small, fragmented populations (Daszak and Cunningham 2002) and have been responsible for some local and regional extinctions (McCallum and Dobson 1995, Daszak and Cunningham 1999, Woodroffe 1999). Reasons proposed for the intensified transmission and better detection of emerging diseases include removal of geographic barriers to human and animal transport, as well as ecosystem disruption, climate change, and habitat fragmentation (Graczyk 2002).

In a broader sense, devising conservation strategies that are practical in the current understanding of the "state of the Earth" will require models that address disease risks

(Munson and Karesh 2002), including risks to humans, domestic animals and plants, wildlife, and whole ecosystems, and that further recognize that all of these groups have shared risks and cannot be viewed in isolation from the others. Such strategies call for a better understanding of the role of wildlife in these larger disease models, as well as call for policies and approaches to integrate human, agriculture, and wildlife disease studies, as addressed in the One Health concept (www.onehealth.com).

SELECT DEFINITIONS AND CONCEPTS

Prior to detailed discussions of wildlife disease topics, it is useful to consider some basic definitions.

Health and Disease

Two approaches commonly have been used to provide a conceptual base for health and disease in wildlife (Wylie 1970). In one, health is viewed as a concept analogous to temperature. As such, while there may be lower limits (death or absolute zero, respectively), there is no true upper limit for either health or temperature.

In such an analogy, health is the concept to be understood and measured, and one seeks to assess how far from death an organism may be. Among humans, features used to assess health can entail physical, mental, emotional, and spiritual components. Considering just physical features, states of human health have been assessed by body mass index, body fat, erythrocyte and serum enzyme measures, and urine metabolites (Berkow and Fletcher 1992). For wildlife and other nonhuman animals, far less is understood regarding what normal physiological values constitute physical health. Measures used also may include body weights, fat indices, as well as a variety of red blood cell, serum enzyme, and urine metabolite values (Malpas 1977, Warren and Kirkpatrick 1978, Seal et al. 1981, DelGuidice et al. 1990, Harder and Kirkpatrick 1994).

In contrast to viewing health on a purported linear scale, health also may be assessed as adaptability to change (Wylie 1970). Less important here is the number of parasites or problems an animal or population encounters than its response and adaptability to these stresses. One working definition of human health is the capacity to achieve socially determined goals (mental, physical, and social well-being, vigor, resilience, productivity, flourishing) within a set of socioecological constraints, only one of which is disease (Murray et al. 2002). Munson and Karesh (2002) view disease as any disorder of body functions, systems, or organs; such a disorder is not necessarily confined to one caused by an extrinsic factor such as viral or bacterial infection, infestation with parasites, or exposure to toxins (Munson and Karesh 2002). In a very broad outlook, disease has been defined simply as a reduction in fitness (Clayton and Moore 1997).

For purposes of this text, we use the definition that wildlife disease is "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). Such a definition can be applied to both individuals and populations. Our primary emphasis in this text is on infectious and, secondarily, noninfectious causes of disease.

Parasitism

Parasitism is a term used to describe one set of interspecific relationships along a broad continuum of relationships between species (Dindal 1975). Broadly, the term symbiology (literally, "living together") has been used to describe the study of any persistent relationship (symbiosis) between two different species; parasitism is one part of that larger set of relationships (Read 1970). It sometimes is argued that while parasites may elude clear definition, they generally are "known primarily when we see them" (Moore 2002).

Broadly, *parasites* are organisms living partly or completely at the expense of another

organism (its *host*). Parasitic lifestyles are regularly represented among four of the major kingdoms: Animalia, Fungi, Protista, and Monera (with a much less common occurrence among Plantae). Viruses raise an interesting problem; they have some features of living systems (e.g., genome, replication, evolution), but are not functionally active outside their host cells and generally are not considered to be living microorganisms (van Regenmortel and Mahy 2004); however, they commonly are treated as highly specialized parasites and included with other microorganisms in this text. Prions, infectious proteins lacking nucleic acids, also raise interesting problems and are addressed as a special topic in Chapter 11.

Parasitic relationships can overlap with some forms of predation (e.g., parasitoid wasps and some fly maggots that kill their hosts) as well as certain mutualistic relationships (e.g., many intestinal bacteria). Generally, parasites live in relatively long contact with the host for part of their life cycle; in contrast, predators and prey generally have a relatively short period of contact that ends in death and consumption of the prey by the predators. Further, parasites typically relinquish the role of regulating their relationship with the external environment to the host during part of their life cycle.

Endoparasites live within a host's body during at least part of their life cycle and depend completely on the host to regulate their relationship with the external environment during that time; examples of endoparasites include some helminths, fungi, protozoa, and many infectious microorganisms. Parasites living on the exterior of the host are termed *ectoparasites;* these have a partial dependence on the host to regulate their relationship with the external environment when they are present on the host; examples include parasitic arthropods such as ticks, fleas, lice, and mites. Finally, parasites generally are characterized as competing with the host for its resources, and often reduce host fitness (e.g., survival, fecundity) (Clayton and Moore 1997).

Many parasites are transmitted directly between susceptible hosts; these species have *direct life cycles* and are termed "monoxenous" (Gr. mono = one, xenous = host). However, others, including many parasitic helminths and protozoa, require two or more hosts to complete their life cycle; these have *indirect life cycles* and are termed "heteroxenous." For parasites with an indirect life cycle, the host in which the sexually mature stage of the parasite occurs is called the *definitive host*. An *intermediate host* is an additional required host for those parasites to complete their life cycle. In the intermediate host, the parasites undergo some developmental changes and may multiply, but do not reach their sexually mature stage; the intermediate host typically is a different taxonomic group from the definitive host. A less common type of host is a *paratenic (transport) host,* an organism which serves to transfer a larval stage or stages from one host to another but in which little or no development takes place (Anderson 1992). A paratenic host is not required for completion of the life cycle, but often is a prey species of the definitive host and facilitates completion of the life cycle; a paratenic host often is considered an "optional" host. The term *vector* is defined by some as any host that transmits parasites, including intermediate, definitive, and paratenic hosts (Clayton and Moore 1997); in contrast, other scholars tend to restrict the term primarily or exclusively to arthropods (Wobeser 2006).

The persistent presence of a parasite in a host is termed an *infection* (Pratt 1963); a related term, *infestation,* is used to describe the persistent presence of ectoparasites. *Prevalence* is the number of animals infected by a parasite divided by the number of animals in the population examined, and commonly is reported as a percent value (Margolis et al. 1982, Bush et al. 1997). In contrast, *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 that time interval (Margolis et al. 1982, Bush et al. 1997); incidence often is reported as number per 1,000 in the population. *Intensity* is the number

of parasites of a particular species in or on a single infected animal; *mean intensity* is the total number of parasites of a particular species found in each host, divided by the number of hosts infected with that parasite (Margolis et al. 1982, Bush et al. 1997).

Mortality refers to the death of a host and *morbidity* is the condition of having an illness, weakness, or other disability. In contrast, *necrosis* refers to a localized area of death in a tissue or organ rather than the death of a whole organism.

Virulence is a measure of the impact of parasites upon their host. Broadly, virulence can be defined as any collective effects on host fitness, including mortality, morbidity, and reduced fecundity (Clayton and Moore 1997). Virulence also has been described more precisely as a complex property comprising three characteristics: infectivity, invasiveness, and pathogenicity (Frobisher 1962, Pratt 1963).

This latter definition has been applied primarily to microorganisms and is considered here in further detail. *Infectivity* is defined as the ability to initiate and maintain an infection in the host (Pratt 1963). This trait is dependent on the capacity of a parasite to establish a persistent presence by evading or overcoming local defense mechanisms of the host. For example, bacteria causing plague *(Yersinia pestis),* typhoid fever *(Salmonella typhi),* or shigellosis (*Shigella* spp.) do not have high infectivity to laboratory workers under normal circumstances (Pratt 1963). In contrast, *Francisella tularensis,* the cause of tularemia, is a highly infectious bacterium readily transferred to workers within the laboratory (Pratt 1963, Hopla and Hopla 1994).

Invasiveness is defined as the ability to progress further into the host from the initial site of infection (Pratt 1963). For example, many bacteria, including *Pasteurella multocida,* the cause of avian cholera, commonly invade the blood stream, causing a *septicemia* (presence of pathogenic bacteria or their toxins in the blood) among infected wildfowl.

Pathogenicity is the ability to injure a host (Pratt 1963) by damaging host tissues. For larger parasites such as helminths and arthropods, physical damage and blood loss are common causes of morbidity and mortality. Among bacteria, toxins affecting the nervous system, heart, or kidney are more typical causes of pathogenicity (Pratt 1963). In the laboratory, pathogenicity in living animals commonly has been measured by an LD_{50} test; an LD_{50} is the dose of an infective agent or toxin lethal for 50% of a test population (Fig. 1.4) (Reed and Muench 1938).

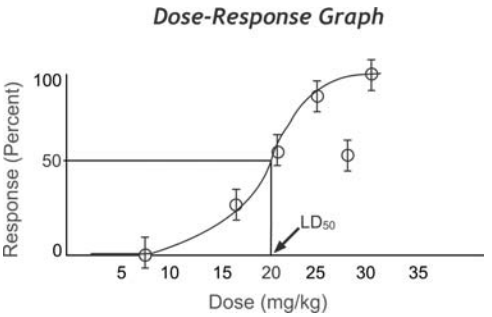

FIGURE 1.4 Sigmoid curve typical of an LD_{50} test (Courtesy of National Library of Medicine. Based on original from http://aquaticpath.umd.edu/appliedtox/images=toxtutor/chart-5.gif. Drawn by Patient Education Institute).

The LD_{50} test has been highly regarded as a measure of pathogenicity because of its consistency and its requirement for relatively few test animals. However, the LD_{50} test also is limited to assessing mortality and cannot assess more subtle influences such as morbidity, reduced reproductive success, or increased susceptibility to predation; further, it also is useful only for single, isolated mortality factors and is not readily adapted to assessing synergystic relationships between two or more factors influencing mortality. In recent years, the LD_{50} (and the consequent killing of laboratory animals) increasingly has been replaced by alternative tests to measure pathogenicity, such as assessment of cellular pathology through the use of tissue cultures. However, it still is used in toxicology, where every registered pesticide must have at least three avian LD_{50} tests (A. Fairbrother, pers. comm.).

In summary, for a microorganism to be considered virulent, it must simultaneously be infective, invasive, and pathogenic for a given host

(Pratt 1963). For example, *Mycobacterium tuberculosis,* a cause of human tuberculosis, is very invasive and highly pathogenic to guinea pigs *(Cavia porcellus)* in the laboratory (Wämoscher and Stöcklin 1927, Dörr and Gold 1932, Wilson and Miles 1964); yet there are no evident reports of its occurrence among guinea pigs in their natural habitats (Shope 1927, Wilson and Miles 1964, Williams 2001). Thus, while very invasive and pathogenic, an apparent absence of infectivity would result in the bacteria not being considered virulent for guinea pigs. Likewise, although *Pasteurella multocida,* the cause of avian cholera, is virulent to at least 180 species of birds (Samuel et al. 2007), there is no evidence that *P. multocida* can invade the blood stream (cause a septicemia) or cause a clinical disease among turkey vultures *(Cathartes aura)* (Botzler 1991, Samuel et al. 2007); thus by definition *P. multocida* is not invasive, and consequently not virulent, for turkey vultures.

Historically, microparasite virulence was viewed as a sign of recent association between a host and parasite, and it was argued that subsequent host–parasite co-evolution would lead to a reduction of virulence and even the development of commensalism or mutualism (Burnet and White 1972). A more recent hypothesis is that virulence also can be maintained by natural selection and may increase or decrease in evolutionary response to environmental conditions or the density and behavior of hosts (Levin 1996). Thus the level of virulence expressed by parasites may result from the strategy developed by the infective agent for optimal transmission and survival (Ewald 1994). Alternatively, it also has been proposed that the virulence of microparasites is coincidental to parasite-expressed characters that evolved for other functions, or emerged as the product of short-sighted evolution in infected hosts (Levin 1996). All of these factors may play a role in different circumstances.

Diseases in Populations

Several key terms are used in describing diseases in populations. The term *epidemic* (*epi:* upon; *dem:* people) refers to a disease affecting many people within an area at one time, at a significantly greater occurrence than expected. Examples include outbreaks of bubonic plague and human influenza. The term *epizootic* has been used to refer to epidemics within nonhuman animals and the term *epornitic* occasionally is used to refer to epidemics among avian populations. The term *pandemic* refers to a worldwide epidemic (among humans, since most other species don't have the same broad distribution). For example, human influenza is estimated to have caused the death of >20 million humans worldwide during the pandemic of 1918–19 (Slemons and Brugh 1994). Bubonic plague also has caused numerous pandemics among humans (McNeill 1977).

In contrast, the term *endemic* refers to a parasite or disease with a low incidence, but one that is regularly present in a host population. *Enzootic* is a similar term that has been used in reference to diseases characteristic of nonhumans. For example, *Yersinia pestis,* the cause of bubonic plague, is maintained among some rodent populations such as the California vole *(Microtus californicus)* and the deer mouse *(Peromyscus maniculatus)* in western North America; *Y. pestis* is considered enzootic in these species (Poland et al. 1994). One potentially confusing aspect of the term endemic is that in ecological literature it commonly refers to a species that evolved solely in a limited area or region, as on certain islands (Van Dyke 2003); use of the alternative term "enzootic" in disease literature can help reduce that potential confusion.

The terms endemic and enzootic also have been used to signify a parasite or disease characteristic of a geographic region (rather than a particular host species). Thus, plague also can be characterized as enzootic to dry grasslands, mountain meadows, and some deserts of western North American and other regions of the world. As another example, avian cholera, caused by *Pasteurella multocida,* regularly causes epornitics among wildfowl of North America; while found in all North American flyways, it generally is considered enzootic to

northern California, Nebraska's Rainwater Basin, and the Texas playa lakes (Friend 1999).

The term *reservoir* has been used by authors in several ways. Simply stated, it can be defined as the sum of all sources of infection—the natural habitat of the parasite (Pratt 1963). More specifically, a reservoir of infection has been defined as an ecologic system in which an infectious agent survives indefinitely (Ashford 1997); such an ecologic system would encompass all of the vertebrate and invertebrate host populations and encompass the pertinent environmental factors as well as any quantitative factors, such as critical community size, needed to maintain an infectious agent indefinitely (Ashford 2003). For our use, we generally refer to Ashford's modified 2003 definition of a reservoir.

Examples of reservoirs range from red foxes *(Vulpes vulpes)* for rabies viruses in Europe (Rupprecht et al. 2001), to soil, mud, or water as reservoirs for the bacterium *Listeria monocytogenes* (Bille et al. 1999). However, the notion of reservoir for a parasite or disease may vary with geographic scale, such as that occurring within a specific watershed versus a more general assessment of the reservoir on a worldwide basis. Further, in describing reservoirs, researchers only address a limited number of aspects of the environment and thus may miss key criteria in their descriptions. The term *reservoir species* has been used in a specialized sense to refer to an introduced host that has artificially raised the size of the collective host populations or densities, consequently allowing pathogen transmission even when the endemic host population had been reduced below the density at which a pathogen is able to maintain effective transmission (Daszak and Cunningham 2002).

Time scale is another important factor. Arthropod-borne agents of vertebrates that survive in temperate regions often survive during a period of time (e.g., cold, dry) when arthropod survival is low. In some cases the infective agent survives primarily in the vertebrate population and the vertebrates could be considered the primary reservoir. However, our use of reservoir (e.g., in Chapters 9 and 10) will generally take an annual or multi-year perspective, and thus we will refer to arthropod–vertebrate reservoirs in these chapters, even where some members (e.g., arthropods) play a smaller role during some parts of the year.

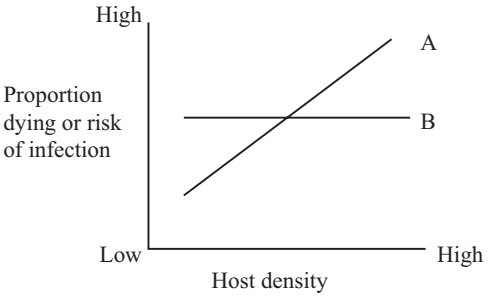

FIGURE 1.5 Contrast of density dependent (A) and density-independent (B) disease impacts.

Density and Disease

Parasites and diseases whose risk of infection or impact varies consistently with the density (number per unit area) of their host populations are termed *density-dependent,* whereas those whose risk or impact do not change in response to differing densities are called *density-independent*. Thus, for a density-dependent disease, the rate of transmission of a disease through a population, or risk of infection for the susceptible individual, varies consistently with the density of the host population (Fig. 1.5). Generally, parasites transmitted directly between hosts of a susceptible species, without the need for intermediate hosts or vectors, cause density-dependent diseases (Scott 1988).

Rabies often is cited as a classical density-dependent disease (Macdonald 1980, Bacon 1985). One would expect that a rabid fox would have a higher probability of encountering an uninfected, susceptible fox in a high-density fox population, compared to a low-density fox population. Thus the risk of rabies transmission would be higher in a high-density than in a low-density population of susceptible animals.

In contrast, among density-independent diseases, the rate of transmission of the causative agent through a population, or risk of infection for the host, is independent of the density of the susceptible host population. Pesticides and environmental toxins, infectious diseases with reservoirs in soil or water, severe weather, and accidents tend to have density-independent impacts. For example, the risk to an individual bird of flying into a power line during a migration would not be expected to change in a consistent fashion if the flock size increased or decreased. Also, one of the devastating impacts of some pesticides is that their lethal effects are unabated even as the host populations reach very low levels (Hickey and Anderson 1968, Risebrough 1978, Peek 1986).

It is important to note that the significant distinguishing feature between density-dependent and density-independent diseases is the proportion (rather than the actual number) of a susceptible population affected. For example, if the hosts in a susceptible population have a 5% risk of mortality (in a density-independent situation), one would expect approximately 5 hosts in a population 100, or approximately 50 hosts in a population 1,000, to die. Thus, while the total numbers of animals dying increases in larger populations, the actual proportion of animals affected (5%) is unchanged when transmission is density independent.

Density dependence becomes more complicated with parasites undergoing indirect life cycles. For example, among parasitic helminths, the intestinal tapeworm *Echinococcus granulosus* involves large canids (e.g., wolves, *Canis lupus;* coyotes, *Canis latrans*) as definitive hosts and ungulates (e.g., moose, *Alces alces;* deer, *Odocoileus* spp.) as intermediate hosts. For a given season, the risk of infection to wolves by ingesting the tapeworm infective stage (hydatid cysts) is more directly dependent on the density of hydatid-infected moose than on the density of the other infected wolves (who are shedding eggs infective to moose). Likewise, in a given season the risk of moose becoming parasitized by ingesting tapeworm eggs is more directly affected by the density of wolves shedding eggs than by the density of other hydatid-infected moose. Thus, over a shorter term, such as a single season, risks of acquiring indirect life cycle parasites tend to be density independent. However, over several seasons, one would expect that increases in wolves (shedding eggs) would lead to increases in infected moose, which, in turn, would lead to increases in the prevalence of infection among the wolves (eating infected moose); thus, over a longer term, risks of acquiring indirect life cycle parasites can become density dependent.

There is some similarity for arthropod-borne parasites (e.g., West Nile virus) to the relationship described above for indirect life cycle parasites. However, arthropod-borne parasite life cycles are complicated by additional factors such as the repeated feeding by some arthropods in a season, with their consequent increased likelihood of acquiring parasites. In such cases, the risk of infection to a susceptible vertebrate host is potentially dependent on both the density of infected vectors and the density of the other infected vertebrates. Likewise, the risk of infection to a vector is certainly influenced by the density of infected vertebrates, but also can be influenced by the density of other infected vectors that may infect vertebrates and make them available later in a season to the uninfected vectors. It is more difficult to break the cycle in this case as both vertebrate hosts and vectors can contribute to a condition more closely representing that of density dependence. While over a shorter term, such as a single season, arthropod-borne diseases often tend to be density independent, the influences of multiple feeding each season may lead to an additional density-dependent influence as well, over several seasons.

Disease Models

We present two models to use as foundations for conceptualizing key relationships in the field of wildlife diseases. One entails

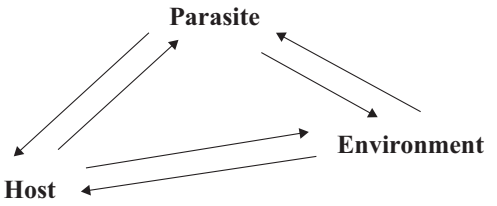

FIGURE 1.6 Parasites, hosts, and their environments are closely intertwined—each affecting the other two in varying degrees.

an understanding of three general components: *parasites* and other causative agents of diseases, the affected vertebrate and invertebrate *hosts,* and the *environment* in which these occur. In this model each component interacts with the other two, and must be described and understood in relation to the other two (Fig. 1.6). Any change in one can drastically alter the balance of a resulting disease.

An earlier and more complex disease model, proposed in 1939 by Soviet biologist Evgeny N. Pavlovsky, first was used to describe arthropod-borne diseases (Pavlovski 1966). It variously has been termed the Natural Nidality Doctrine of Transmissible Diseases, the Landscape Theory of Epidemiology, the Landscape Theory of Zoonotic Diseases (Pollitzer and Meyer 1961, Pavlovski 1966), and the Natural Nidality Theory (Nelson 1980).

Pavlovski believed that most transmissible diseases exist in nature as discrete *foci* or *nidi* (sing. *nidus,* "hearth," "home"). A nidus is defined as that portion of a region with a definite geographic character, and would be similar in usage to that of the terms biotope, ecosystem, or habitat type. A nidus can be a small local area or a broad geographic region (Pavlovski 1966). Thus, rabies in the range of infected red foxes *(Vulpes vulpes)* in Europe would be an example of a broad nidus; likewise, plague would be seen as having nidi among dry grasslands (steppes), mountain meadows, and some desert habitats in western North America and Asia. A nidus also can be dynamic, as in the cases of a rabies nidus among red foxes moving across Europe (Macdonald 1980), or a plague nidus shifting in North America (Barnes 1982). However, most nidi are more permanently associated with specific regions and habitat types.

Within each nidus is an ecological association termed a *biocoenosis,* composed of the infective agent, the wild vertebrate reservoir species, intermediate hosts or vectors (typically arthropods or other invertebrates), and any wild vertebrate amplifying hosts. All of these organisms are limited in their geographic and ecological distribution by the environmental determinants of the habitat. These biocoenoses allow circulation of the parasites indefinitely and can be viewed as integrated wholes (Pavlovski 1966). Microscale disease foci are influenced by qualities of the entire ecosystem. Focal diseases in discrete sites may spread out from the nidi to cause epizootics among susceptible hosts. Susceptible wildlife, humans, or other domestic animals might become involved if they invade an active nidus. This holistic approach to the study of disease ecology has been used as a foundation for the study of plague in North America (Nelson 1980) as well as a number of other diseases (Pavlovski 1966).

CAUSES OF DISEASE

Diseases can be caused both by infective agents and by noninfectious causes including toxins, tumors, nutritional and metabolic problems traumas, and many others. Most diseases we address are caused by infective agents. Infective agents most commonly are living organisms and typically are classified among five major kingdoms (Whittaker 1969). Viruses and prions are additional agents that, while not considered living entities, also are infective agents (Büchen-Osmond 2003).

Living organisms with a parasitic lifestyle comprise the majority of species in the world; by one estimate parasites outnumber free-living species in a four to one ratio (Zimmer 2000). Among living organisms, an initial division typically is made between eukaryotic and prokaryotic organisms. Eukaryotic organisms all have double-stranded DNA enclosed within a nuclear membrane, an endoplasmic reticulum,

mitochondria, Golgi apparatuses, lysosomes, and other cellular organelles; these organisms divide by mitosis or meiosis. Traditionally they have been distributed among the Kingdoms Protista (single-celled eukaryotes), Plantae, Animalia, and Fungi (Whittaker 1969) (Appendix 1).

In contrast, prokaryotic organisms lack true nuclei and have single-stranded DNA; they also lack such organelles as mitochondria, endoplasmic reticula, Golgi apparatuses, and lysosomes (Murray et al. 1999). Prokaryotes divide by fission rather than by mitosis or meiosis. Prokaryotes include both the Archaebacteria and Eubacteria ("true bacteria") (Murray et al. 1999) and are classified in the Kingdom Monera (Whittaker 1969) (Appendix 1).

Viruses lie at the boundary between life and inert matter and are not typically included in classifications of living organisms, even though they regularly replicate, mutate, evolve, and serve as significant influences on the evolution of their hosts (Villarreal 2004). Classification schemes have emerged for viruses based on their proposed evolutionary relationships (Büchen-Osmond 2003, van Regenmortel and Mahy 2004). Recently, considerable interest has emerged in the role of infectious proteins ("prions"), which have no nucleic acids (Büchen-Osmond 2003) but are important contributors to some wildlife diseases.

Views about the evolutionary relationships within and between various groups have been changing (Doolittle 1999). We note some of the recent changes in proposed classifications (e.g., Adl et al. 2005); however, because of its long and well-established history, we base our discussions on the traditional five-kingdom system (Monera, Protista, Fungi, Animalia, and Plantae) (Whittaker 1969) in this text (Appendix 1). Viruses and prions are treated as addenda to the five-kingdom system. For each major group we provide a general definition of the group, a brief description of some of its distinctive features, and a summary of any recent taxonomic changes.

Although our main focus is on diseases caused by infective agents, there also is a wide variety of noninfectious diseases to which wildlife are subject (Fairbrother et al. 1996). Two that we will address include cancers and toxins.

ROLE OF DISEASES IN WILDLIFE POPULATIONS

Disease agents function by reducing the fitness of their hosts in a variety of ways (Scott 1988). In wildlife management, factors that directly reduce wildlife numbers have been termed *decimating factors,* and diseases are one of many different decimating factors (Leopold 1933). In contrast, *welfare factors* are non-lethal factors such as a shortage of food, water, or cover that reduce wildlife reproductive success (Leopold 1933) or make wildlife more susceptible to other mortality factors such as predation, accidents, and so on; diseases also can function as welfare factors.

Historically, a major focus of wildlife managers was on the role of diseases as decimating factors, especially among economically important wildlife such as ungulates, waterfowl, and upland game. These kinds of diseases often are exemplified by microparasites that undergo multiplication within their hosts. Such diseases commonly produce epizootics where waves of infection pass through populations, alternating with periods in which the pathogen disappears following a loss of susceptible hosts as they die or survive and become immune. Examples include avian cholera in wildfowl (Botzler 1991, Samuel et al. 2007), hemorrhagic diseases of deer and other ungulates (Howerth et al. 2001), or tularemia in rabbits (Mörner and Addison 2001). The 1988 epizootic of phocine distemper virus in the North Sea population of harbor seals *(Phoca vitulina)* is a particularly well-documented example of a decimating disease (Hudson et al. 2003). Here the parasite appeared in a series of harbor seal populations around the coasts of northern Europe, and then disappeared following a lack of new susceptible animals (Hudson et al. 2003).

Disease also can serve as a welfare factor by reducing the reproductive success of susceptible animals (Gulland 1995). Among bacteria,

Salmonella pullorum reduces the egg-laying capacity of ring-necked pheasants *(Phasianus colchicus)* by 75% or more, and hatched birds often are stunted and less fit (Biester and Schwarte 1965). *Brucella abortus* infects and causes abortions in bison *(Bison bison),* elk *(Cervus elaphus),* and other ungulates (Thorne 2001). Infections by many viruses, including members of the families Parvoviridae, Herpesviridae, Paramyxoviridae, and Orbiviridae, can result in abortion or neonatal death. Because neonatal mortality or reproductive failure resulting from infectious agents may be difficult to discern, host population size may be modulated by virus infection in the absence of measurable adult mortality (Poss et al. 2002).

Diseases also can reduce the energy resources available for host immunity and lead to greater susceptibility to other parasites. Such parasites benefit when poor nutrition or other environmental conditions reduce the efficiency of the immune system, making their hosts more vulnerable (Chandra and Newberne 1977, Gershwin et al. 1985). For example, normally quiescent but opportunistic bacteria carried in the intestinal tract (e.g., *Salmonella* spp.) or respiratory tract (e.g., *Pasteurella* spp.) can cause overt disease in the presence of a compromised immune system. Also, some species experiencing diseases are more susceptible to other stresses such as cold or food shortage (Sheppe and Adams 1957), thus contributing to diminished well-being of individuals and populations. There also are interactions with malnourishment, infections, and environmental chemicals on growth and reproduction (Porter et al. 1984)

Also, macroparasites commonly occur as enzootic infections, more commonly causing host morbidity than mortality. Sick animals may be less cautious and have slower reflexes than healthy animals (Poulin 1994). Such behavioral changes in animals may lead to greater susceptibility to predation or accidents. Likewise, lead poisoning (plumbism) and botulism intoxication may make waterfowl more susceptible to predation. Neurological diseases, such as canine distemper or rabies, may enhance the likelihood of some terrestrial mammals dying from highway mortality or other accidents.

Sexual selection also may be influenced by parasites and diseases. For example, secondary sexual characteristics of male birds, including brightness or color and vocalizations, may signal a male's overall well-being and freedom from parasites. Males resistant to parasites within a species may be more attractive to breeding females due to their brighter plumage, more vigorous songs, or other superior mating behaviors, compared to infected males (Hamilton and Zuk 1982, Loye and Zuk 1991, MØller 1991, Zuk 1991). Linked to these findings is evidence that parasitism may be more common among individual animals affected by developmental asymmetry of secondary sexual characteristics (MØller 1996, MØller and Swaddle 1997, Thornhill and MØller 1997). Conversely, higher parasite levels may contribute to greater asymmetry of secondary characteristics. For example, parasite-infected reindeer *(Rangifer tarandus)* have less symmetrical antlers (Folstad et al. 1996), and mite-infected barn swallows *(Hirundo rustica)* have higher levels of asymmetry in wing length and tail feathers compared to uninfected members of their respective species (MØller 1992). In turn, such levels of asymmetry could influence females seeking males for mating (MØller and Swaddle 1997) and thereby affect mating success and fitness.

The cost in fitness from an infectious bacterium or virus that kills an animal or weakens it to the point where it is susceptible to predation or starvation is self-evident. The fitness costs from arthropods, intestinal nematodes, and some microparasites often are far more subtle (Hart 1997). For example, a light parasite load that may not noticeably impact a healthy, well-fed adult bird may severely affect it in times of nutritional or socially related stress, or in conjunction with the physiological demands of laying and incubating eggs, provisioning nestlings, escaping from a predator, or fighting with conspecifics

(Hart 1997). In cases where male offspring grow larger and more quickly than female offspring, parasitism can impede the ability of avian mothers to raise males, shifting the sex ratio and affecting population viability; removing their parasites allows the mothers to forage longer and rear more sons (Reed et al. 2008). Also, among polygynous species, pathogens are dispersed by infected females after the resident male dies, and the effects of pathogen-mediated dispersal increases as the harem size (number of females) increases (Nunn et al. 2008).

CAN DISEASES REGULATE WILD POPULATIONS?

Although the mortality from a disease can be dramatic, there often is little relationship between observed mortality and the effectiveness of a disease in limiting or regulating a host population. For example, avian cholera can be an explosive local disease, killing thousands of birds on a given site (Friend 1999). Approximately 37,000 birds died in one California epornitic, yet it was estimated that even such severe mortality affected only about 0.5% of the California waterfowl populations and that these losses could be recovered readily on the breeding grounds (Rosen 1972). Among waterfowl, avian cholera generally is less important than habitat destruction or hunting in limiting populations.

However, there are cases where diseases can substantially influence wildlife populations, especially on initial introduction to a population. Some microorganisms can suppress wild host populations through reduced survival, reduced fecundity, or both (Scott 1988, Tompkins and Begon 1999, Hudson et al. 2003). In a classic case, myxoma virus, a poxvirus, has caused a long-term depression of European rabbit *(Oryctolagus cuniculus)* populations in Australia (Fenner and Ratcliffe 1965) and Europe (Ross 1982). Rabies also can temporarily suppress affected host populations (Bacon 1985). Canine distemper, a viral disease, has caused severe declines of some African lion *(Panthera leo)* populations (Morell 1994) as well as near extinction of black-footed ferrets *(Mustela nigripes)* (Thorne and Williams 1988).

Among macroparasites, there are a number of studies with evidence for helminth and arthropod parasites effectively controlling wild animal populations through reduced survival or fecundity of the hosts (Tompkins and Begon 1999). The parasites involved included two species of fleas, four species of mites, two species of bugs, one species of fly, and three species of nematodes; the affected hosts included eight species of birds and three of mammals. One of the best documented cases involves *Trichostrongylus tenuis,* an intestinal nematode, that helps drive population cycles of red grouse *(Lagopus lagopus)* in Scotland (Potts et al. 1984; Hudson et al. 1985; Hudson and Dobson 1989; Dobson and Hudson 1992; Hudson et al. 1992, 1998, 2003).

Among toxins, there is strong evidence that during their regular use, dichlorodiphenyltrichloroethane (DDT) and other environmental toxins suppressed populations of raptors and fish-eating birds (Hickey and Anderson 1968). For example, use of DDT depressed peregrine falcon *(Falco peregrinus)* populations by reducing eggshell thickness, interfering with calcium carbonate deposition in eggshells, and altering reproductive behaviors (Enderson and Berger 1970). Significant recovery of several raptorial and other bird species occurred after banning many persistent and bioaccumulative pesticides in the United States (Anderson et al. 1975, Spitzer et al. 1978, Grier 1982, Grue et al. 1983, Bolen and Robinson 2003).

Pathogens infecting a broad range of host species can cause serious problems for endangered populations (McCallum and Dobson 1995), and species-wide extinctions have been linked to diseases. For example, there is good evidence that avian malaria *(Plasmodium relictum capistranoae)* and avian pox (Poxviridae) have caused some population suppressions, local extirpations, and even species extinctions among native Hawaiian birds. These losses involved some complex interactions among the native hosts, introduced species of hosts, parasites, and vectors, as well as well as habitat

(Warner 1968, van Riper et al. 1986). Interestingly, there also is recent evidence for limited species recovery among some native Hawaiian birds that did not become extinct (Woodworth et al. 2005). While it is highly likely that disease caused at least some of these extinctions, the evidence still is indirect. The first known definitive report of a parasite causing species extinction is the loss of a land snail *(Pardula turgida)* brought about by a microsporidian parasite (*Steinhausia* spp.) (Cunningham and Daszak 1998).

Bighorn sheep *(Ovis canadensis)* introduced into Lava Beds National Monument, California, were locally extirpated from effects of *Pasteurella multocida* pneumonia in July 1980 following their apparent contact with domestic sheep on adjacent grazing leases (Foreyt and Jessup 1982). The response of managers to prevent this loss was complicated by political conflicts among the several federal and state agencies and ranchers with responsibilities for the animals or land. There also is evidence that local populations of prairie dogs (*Cynomys* spp.) can be extirpated by bubonic plague in short-grass prairies (Kartman et al. 1962, Barnes 1982).

Rinderpest, a morbillivirus infection, historically caused substantial reductions among wild ungulate populations in Africa, including local extirpations of some species and significant changes in the species composition of African ungulates in many regions (Talbot and Talbot 1963, Holmes 1982, Plowright 1982, McCallum and Dobson 1995). This introduced pathogen swept through southern Africa between 1890 and 1899 and killed up to 90% of the populations of some native wild species (Plowright 1982). Rinderpest is benign in its ancient cattle host (McCallum and Dobson 1995), but highly virulent to the wildebeest *(Connochaetes taurinus)* and cape buffalo *(Syncerus caffer),* as well as introduced cattle recently exposed to this morbillivirus (Plowright 1982). Rinderpest exemplifies a disease in populations lacking past exposure or innate immunity; the causative virus infected a large proportion of the susceptible populations and mortality was high. Wild ungulates were blamed as reservoir hosts for susceptible breeds of European cattle and were slaughtered in areas around cattle ranches. However, control of rinderpest in Tanganyika wildlife through use of a vaccine in cattle in the 1950s provided evidence that cattle, rather than wildlife, played a central role as rinderpest reservoirs (Branagan and Hammond 1965). Plowright later concluded that even large populations, in excess of 100,000, of susceptible wild African ungulates were unable to sustain rinderpest infections in the absence of cattle (Plowright 1982). Following a worldwide cattle vaccination campaign to combat the disease, rinderpest was declared to be only the second disease to be eradicated on a worldwide basis, following smallpox (Anonymous 2011).

Mathematical models of microparasitic diseases were developed to assess expected impacts of these diseases on their hosts (McCallum and Dobson 1995). Some generalizations that emerged are that most pathogens do not depress host population equilibria far below their disease-free carrying capacity (Anderson 1979), and that parasites highly pathogenic for individuals usually have only a minor effect on host populations. Often, if a disease is detectable at high prevalence, it probably is mild and unlikely to be a major problem to an endangered species. Also some parasites highly pathogenic in the laboratory are unlikely to cause problems in low-density populations because infected animals die before the disease can be spread.

These conclusions are subject to two major qualifications. First, they apply to single-host species models, and many pathogens implicated in extinctions of one host have other reservoir hosts in which they are relatively benign (van Riper et al. 1986, Thorne and Williams 1988). Thus if a pathogen is a generalist and an endangered species is susceptible, the pathogen can cause the endangered species to decline if it has a sympatric host species (reservoir species). Second, the mathematical models assume the disease primarily increases host mortality. If the disease decreases fecundity, then diseases at high prevalence may have a significant impact on host populations without causing increased deaths (McCallum 1994); DDT had

such effects (Enderson and Berger 1970). Similar generalizations have been drawn from models of helminth and other macroparasitic infections (Anderson 1980).

Where diseases affect hosts differentially, occurrence of sympatric populations of vertebrate hosts with a shared parasite can result in one host benefiting by a greater impact of the parasite on the other (Hudson and Greenman 1998). Parasite-mediated competition can act when an invading species introduces a parasite to a vulnerable resident species. One example is the likely significant impact on the native red squirrel *(Sciurus vulgaris)* of the introduction of a parapox virus by the introduced eastern gray squirrel *(S. carolinensis)* (Tompkins et al. 2002). Similarly, diseases introduced with domestic dogs have exerted significant impact on rarer and endangered indigenous species as Ethiopian wolves *(Canis simensis)* (Laurenson et al. 1998) and wild dogs *(Lycaon picta)* (Kat et al. 1995). Likewise, bighorn sheep are more susceptible to the effects of *Pasteurella multocida* than are domestic sheep, and the pasteurellae caused a likely extirpation of bighorns from Lava Beds National Monument (Foreyt and Jessup 1982, Foreyt 1989). Parasite-mediated competition also has been proposed as a mode of action among white-tailed deer *(Odocoileus virginianus)* in gaining competitive advantage over moose *(Alces alces)* in areas of the eastern United States and Canada (Kearney and Gilbert 1976). The parasite *Parelaphostrongylus tenuis* is a meningeal nematode with little or no impact on white-tailed deer; in contrast, other ungulate species are far more susceptible (Lankester 2001). However, this purported role of regulating moose populations by the parasite has been controversial (Nudds 1990). In this context, it has been proposed that where a parasite species infects more than one host species, the pathogen will be least pathogenic to the host with the larger range and more pathogenic to the species with limited range; such a relationship has potentially serious impacts for rare and endangered species with limited distributions (Price et al. 1988).

However, it must be recognized that parasite infections or toxins are only one of several elements affecting host population numbers over time (Scott 1988). It often is difficult to clearly distinguish the specific role of diseases as decimating factors. It is even more difficult to document their roles as welfare factors in interactions with nutrition, stress, genetic problems, predator–prey interactions, accidents, climate, or other ancillary factors.

Diseases may exert selective pressures on various social behaviors. For example, mating behaviors, social avoidance, group size, and group isolation may have been affected by selection pressures to reduce transmission of pathogens (Loehle 1995).

A final, positive note is that while parasites can be detrimental to host fitness in one environment, they can be beneficial to it in another. There is some evidence that parasitized individuals may enjoy a selective advantage over unparasitized conspecific hosts in some circumstances (Thomas et al. 2000).

OVERVIEW AND SUMMARY

Overall, wildlife diseases can be serious decimating factors to affected host populations. They can suppress and regulate these populations, cause local extirpations, and have been associated with species extinction. Most of the emphasis in this book will focus on the role of diseases as decimating factors. Additionally, diseases can serve as welfare factors and may reduce reproductive success or increase the likelihood of death from other causes. Diseases can influence sexual selection among hosts. Further, diseases may interact with other extrinsic factors such as nutrition and stress in their hosts, and even have been proposed as a means by which their hosts can gain an advantage over a competing host species. With this brief introduction, we are ready to begin a more detailed look into the fascinating world of wildlife diseases.

We begin (Chapter 2) with a summary of the tools and strategies wildlife hosts can use in protecting themselves from the effects of various diseases they encounter, so as to be able to clearly address these defense mechanisms in

later discussion of these diseases. In many ways, the relationships between hosts and parasites can be described as an evolutionary "dance," a constant competition with each side seeking to respond appropriately to the moves of the other to optimize their own success and avoid a loss of fitness. Considering disease in an evolutionary context, there never are ideal phenotypes (Ewald 1994). Hosts and parasites each are full of compromises, and each is under considerable selective pressure as it evolves an optimal level of success. Many of the discomforts felt by organisms experiencing a disease (e.g., fever, diarrhea, allergies, anxiety) are connected with contemporary defense mechanisms (Ewald 1994).

We then assess the major macroparasites by taxonomic group in Chapters 3 through 5. For purposes of this text, we provide the taxonomic information appropriate to a level that is most practical for an introduction to wildlife diseases; the actual taxonomic levels addressed among various parasite and host groups are not consistent. Next we address eukaryotic single-celled organisms by basic taxonomic group in Chapters 6 and 7.

Then our emphasis is upon prokaryotic and other microparasites, including bacteria and viruses, in Chapters 8 through 10. Understanding the basic life cycles and life history strategies of each parasite group as their selective pressures work to optimize their biological success, the problems they encounter, and how they overcome them, underlie an understanding of how these diseases ultimately can be managed.

In Chapter 11 we address a few special topics such as noninfectious diseases, including toxins, cancers, prion diseases, and the global amphibian decline. While of more recent interest, these issues increasingly are recognized as having considerable importance for wildlife.

Finally, in Chapter 12 we address specific applications and special topics, including emergent diseases, special problems, and a look at future wildlife disease studies, management, and conservation.

Our goal is to provide a broader understanding of wildlife diseases from an ecological and evolutionary approach of key taxonomic groups, rather than emphasizing a clinical and pathological perspective. Hosts and parasites are constantly interacting with each other in a dynamic fashion. Each species tries to optimize its own success, and the degree of sophistication that has evolved in that process is remarkable. Methods of transmission and types of host defenses are among the more striking examples. It is a sense of this larger picture we wish to emphasize.

In approaching a study of wildlife diseases, we remind readers that we work from the perspective of contemporary Western science. This approach has led to enormous advancements in human understanding of the natural world, including significant insights into the understanding of the health and diseases of wildlife. It also is a system with some limitations and often is characterized as being hierarchical, elitist, and dualistic in its approach; many argue that no one cultural worldview is privileged (Klukhohn and Leighton 1946/1962, Feyerabend 1987, Abram 1996, Nakashima 1998, Berkes 1999), and we acknowledge that Western science is only one of a number a ways of viewing an understanding of wildlife health and diseases. Of course, we also recognize the many benefits and values of using Western science as a foundation for understanding the world.

Finally, the major emphasis is on disease in wildlife populations rather than a focus on individuals. The effects of wildlife diseases on individual animals is covered well in veterinary references focusing on captive wildlife (Fraser and Mays 1986, Fowler et al. 2003) as well as more general veterinary texts (Fraser and Mays 1986). There also are some valuable references covering more specialized topics (Murphy et al. 1999, Mullen and Durden 2002, Stockham and Scott 2002).

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