USGS
science for a changing world

National Wildlife Health Center

Why Bother About Wildlife Disease?

Circular 1401

U.S. Department of the Interior
U.S. Geological Survey

Cover: Some of the animals that may be involved in opportunities for the transfer of pathogens between various animal populations and between animals and humans. (USGS file photos)

Why Bother About Wildlife Disease?

By Milton Friend

National Wildlife Health Center

Circular 1401

U.S. Department of the Interior
U.S. Geological Survey

U.S. Department of the Interior
SALLY JEWELL, Secretary

U.S. Geological Survey
Suzette M. Kimball, Acting Director

U.S. Geological Survey, Reston, Virginia: 2014

Suggested citation:
Friend, Milton, 2014, Why bother about wildlife disease?: U.S. Geological Survey Circular 1401, 76 p. http://dx.doi.org/10.3133/cir1401.

ISSN 2330-5703 (online)

Preface

Curiosity during my undergraduate training in wildlife conservation caused me to enroll in an elective course on wildlife disease. The hands-on laboratory component of that course kindled my interest in this subject, and half a decade later wildlife disease became the focus for the remainder of my career. At that time I became employed by the New York State Fish and Game Department (now the Department of Environmental Conservation) to address such issues. Soon after, I realized that few of us were fortunate enough to be employed in that capacity. At that time, I also became involved in dialogue within the conservation community regarding the question—"Why bother about wildlife disease?" Although there are now substantially more of us addressing wildlife disease issues, the question "Why bother?" remains a focus for dialogue and debate. Thus, after more than a half century of personal involvement with wildlife disease issues, I feel compelled to provide some final personal thoughts on "Why bother?" as I move on to other aspects of wildlife conservation. Perhaps the perspectives provided here will serve to enhance the debate about wildlife disease in ways that will strengthen our capacity to address disease on behalf of free-ranging wildlife populations and advance wildlife conservation in the 21st century.

Milton Friend
Emeritus Scientist
USGS National Wildlife Health Center

Contents

Highlights

Highlights—Continued

Figures

Tables

Conversion Factors and Abbreviations

International System of Units to Inch/pound

Multiply	By	To obtain
	Length	
centimeter (cm)	0.3937	inch (in.)
millimeter (mm)	0.03937	inch (in.)
	Mass	
kilogram (kg)	2.205	pound avoirdupois (lb)

Temperature in degrees Celsius (°C) may be converted to degrees Fahrenheit (°F) as follows:

°F=(1.8×°C)+32

Words shown in **bold** are defined in the glossary.

(Illustration by John Evans)

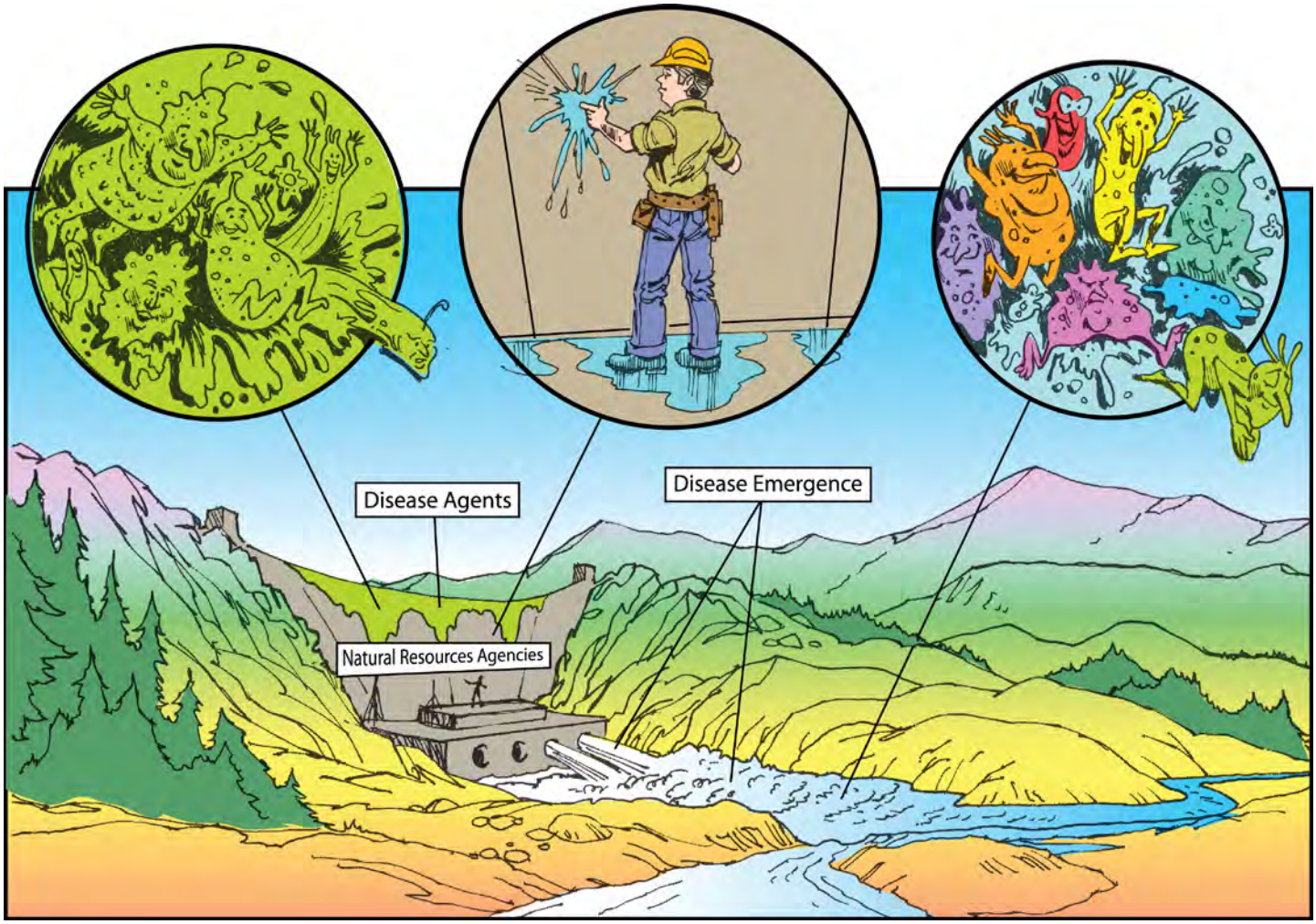
Why Bother About Wildlife Disease?

By Milton Friend

> "In nature …limiting factors are always present, and the maintenance of the numbers of a species can be regarded as a result of conflict between two opposing forces, the "population pressure" of the species, …and the equally constant destruction of individuals by physical calamities, frosts, floods and the like, predatory and parasitic animal species and infectious disease." (Burnet, 1940)

> "Infectious disease is one of the great tragedies of living things—the struggle for existence between different forms of life …
> Incessantly, the pitiless war goes on, without quarter or armistice—a nationalism of species against species." (Zinsser, 1935)

In most developed countries, the maintenance of the numbers of wildlife (see Burnet, 1940, above) is vested in the **natural resource agencies** of those countries. During earlier times, **game species** were the primary focus of natural resource agencies (Leopold, 1933; Grange, 1949); however, current wildlife conservation continues to transition towards a more holistic focus on biodiversity (Van Dyke, 2008) and environmental health (Rapport and others, 1998; Waltner-Toews, 2004). Nevertheless, that transition lags behind in addressing wildlife disease in "…the struggle for existence between different forms of life…" (see Zinsser, 1935, above). Thus, the primary objective of this presentation is to provide a pragmatic assessment of wildlife disease that is irrespective of one's orientation towards wildlife conservation. A secondary objective is to highlight the changing role of disease over time as a wildlife conservation factor. That transition is relevant to the insights provided for current and future efforts focused on sustaining global biodiversity and desired levels of wildlife populations in nature.



For decades, efforts within the wildlife conservation community to address disease on behalf of **free-ranging wildlife** populations have amounted to little more than putting a finger in one of the holes in a conceptual dam to stop the increasing flow of disease bearing down on wildlife. Since the start of the 1970s, the dam has been breached so often that greatly expanded efforts are now needed to address disease if we are to fulfill our stewardship responsibilities for sustaining wildlife populations. (Illustration by John Evans)

Auguste Rodin's sculpture, "The Thinker," highlights our consideration of the relations between wildlife conservation and wildlife disease.

Points to Ponder

Common arguments and positions against the need to address disease on behalf of wildlife include several platitudes:

1. Disease is natural, a part of nature and something to be endured.
2. Nothing meaningful can be done, so why waste limited fiscal and personnel resources on fruitless efforts?
3. Disease is a form of compensatory rather than additive mortality. That is, losses from disease are compensated by reduced losses from other causes such as predation.
4. Disease is useful because it helps to control a population's levels of nuisance and predatory wildlife species.

Those and other perspectives have generally had more support in the past than currently exists because of the dramatic increase in **infectious disease** as a cause of wildlife mortality since the later part of the 20th century (Friend, 2006a).

It is not the purpose here to address directly each of the philosophical positions just noted. Instead, it is more important to consider that wildlife and all other species of our planet (including humans) share Earth's environment. Thus, from a broad context, large-scale wildlife conservation cannot succeed as a stand-alone activity. Instead, conservation is highly dependent upon the values and actions afforded wildlife by humans. Furthermore, history has repeatedly shown the vulnerability of existing values to changing times and circumstances. The specter of wildlife disease is a cost (and sometimes seen as a benefit) that can weigh heavily in shaping public perceptions of wildlife values and associated policy decisions that may ultimately impact wildlife conservation efforts.

For context, it is relevant to move beyond wildlife and consider how human and domestic animal disease are addressed. Both situations are supported by major continuing investments that foster the development of academic venues for study and learning about disease, the establishment of associated government agencies and major scientific institutions to address health issues in affected species, the development of a wealth of advanced technologies, and the initiation of needed regulatory processes directed at combatting various diseases of concern. The profound accomplishments to date are seen in enhanced human and domestic animal health status and economic returns realized by developed countries that have chosen to invest in sound multitiered infrastructures for combatting disease. The importance of those infrastructures in controlling disease is highlighted both during their normal operations and by their occasional temporary failure during catastrophic natural events, war, and civil disturbances. Those disruptions in the effective functioning of human and domestic animal health programs are immediately felt by many.

Because many developing nations may not have an adequate infrastructure for addressing disease, their mode of operation may be primarily one of crisis response rather than preemptive disease management. That type of approach is as inadequate for wildlife as it is in providing for the well-being of humans and domestic animals. Acceptance by natural resource agencies of a crisis type of management approach for disease clearly relegates this factor to a neglected status in ways that counteract other investments being made in the conservation of free-ranging wildlife populations. Conversely, as for humans, it is reasonable to also expect high returns for wildlife from aggressively addressing disease in a preemptive manner. It is not too late to do so, as evidenced by the magnitude of hurdles overcome in the span of about a half-century in combatting infectious disease in humans.

The Many Faces of Wildlife Disease

"…we have to consider not only 'new diseases, but also changes in the activity of old ones'." (Burnet, 1940)

Disease in wildlife takes many forms, some of which are readily observed as abnormalities on body surfaces and as deformities; others may be represented by abnormal reproductive products such as deformed eggs and aborted fetuses; still others may be observed as the presence of parasitic infections (figs. *A*–*F*). Beyond the appearance of clinical **signs** of aberrant behavior of live animals and the presence of dead animals, many other diseases offer no visible clues as to cause.

Regardless of the signs you observe in impaired wildlife, you are confronted by a series of basic questions:

1. What is the cause?
2. Is the cause infectious?
3. If so, how does infection occur?
4. What other species (including humans) may also be affected?
5. Is the cause an established disease agent in the local area or something rarely seen or previously unknown in the area?

For most diseases, you will not be able to determine cause reliably based on observations alone. Thus, you must intervene in some way, which may be simply reporting the event to an appropriate source, to further the process of discovery needed to answer the questions just posed. Those answers have direct relevance for guiding further personal actions, including protection from possible infection if you or others handled the animals, wildlife management actions that may be needed, and in responding to media inquiries that generally occur in response to mass wildlife mortalities or events involving high profile species such as the bald eagle.

Examples of infectious **pathogens** and other causes of disease in wildlife.

Infectious agents	Noninfectious agents	Other causes
Viruses (rabies)	Plant toxins (castor beans)	Trauma (vehicle collision)
Bacteria (tuberculosis)	Pesticides (DDT)	Genetic (aberrant hair coat[1])
Rickettsia (epidemic typhus)	Synthetic chemicals (PCBs)	Nutritional (starvation)
Fungi (Aspergillosis)	Algal toxins (Domoic acid)	Congenital ("Andy Gump" jaw[1])
Parasites (heartworm)	Petroleum products (oiling)	Tumors[2]
Prions (Chronic wasting disease [CWD])		

[1] Nol and Friend (2006).
[2] Can be infectious or noninfectious.

The Many Faces of Wildlife Disease

A.

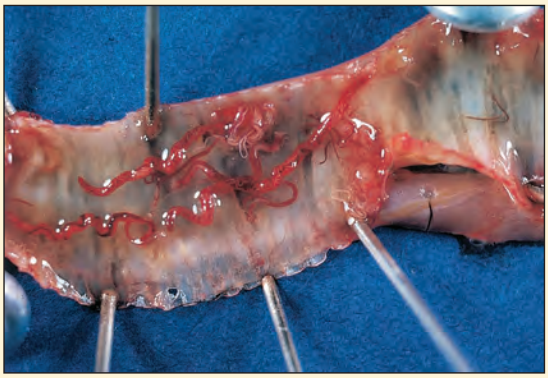
B.

C.

D.

E.

F.



Examples of disease in wildlife caused by different pathogens. *A*, The growths on the white-tailed **deer** did not directly kill the deer; death resulted from secondary bacterial infection of abraded areas of those viral-caused *fibropapillomas*. *B*, Similarly, a cursory evaluation of the hen ring-necked pheasant exhibiting clinical signs of central nervous disorder might result in assumption of a viral encephalitis; instead DDE is the actual cause, as it is for the flattened egg shell in the white-faced ibis clutch shown in *C*. *D*, Paralysis of the inner eyelid seen in this hen, green-winged teal provides a high probability that type C avian botulism is the cause. *E*, The multiple, small burns in the scales of the foot of a bald eagle are indicative of electrocution, but because of their subtle appearance they could easily be overlooked. Also, while parasites are commonly observed during carcass examinations, further evaluation is often needed to determine their significance. *F*, The *Syngamus trachea* (gapeworm) parasites shown here were responsible for a disease outbreak in **game farm** pheasants scheduled for release. (Photos *A*–*D* and *F* by Milton Friend; photo *E* by James Runningen)

The author's personal perspectives about disease as a factor for wildlife conservation have been greatly influenced by decades of personal interactions in the field with biologists such as the U.S. Fish and Wildlife Service national wildlife refuge biologist providing assistance in collecting samples from a dead tundra swan. (Photo by James Runningen)

Personal Perspectives

Initial lessons learned from my academic training and career experiences are that disease (as a process) is a normal component of biological systems; however, the types of diseases occurring (appendix 1), severity of impacts, and timing of disease events are often outcomes from human activities rather than random natural events. Thus, the words of the great comic strip philosopher "Pogo" come to mind—"We have met the enemy and he is us." I find foundation for Pogo's philosophy as it relates to wildlife disease in Aldo Leopold's treatise on "Game Management" published in 1933. I also find it reasonable to assume that if we humans are the problem, then we can also be a solution for how to address wildlife disease more adequately and effectively. At question is whether we have the will to take the actions needed. Historically, it has been difficult to motivate the majority of the conservation community to do so.

After more than five decades of involvement within the wildlife conservation field, I am still trying to resolve a discrepancy between Leopold's contention that the impacts of disease on wildlife populations are largely underestimated and the general apathy towards disease that still exists among many field level personal and administrators within the wildlife conservation field. The foundation for that apathy appears to be an extension of an often-expressed perspective that "disease is a part of nature and as such, is simply something to be endured"—that is, there is "no need to bother" for "this too will pass," and natural biological repair will follow. That perspective fails to consider three important basic facts. First, human actions are impairing wildlife habitat to the extent that, for many species, the level of wildlife population repair that might otherwise occur is severely compromised. Second, during recent decades, the collective toll from disease has undermined conservation efforts for a broad spectrum of species. Third, wildlife are the source species for the majority of **emerging infectious diseases** (EIDs) afflicting humans (Morse, 1995; Dasak and others, 2000; Cleaveland and others, 2001; Taylor and others, 2001; Wolfe and others, 2007). That third fact has resulted in many outside of the natural resource agencies becoming more focused on wildlife disease. A "One World—**One Health**" approach for addressing infectious disease has been rekindled and is at odds with a **laissez faire** approach to wildlife disease (Monath and others, 2010; Zinsstag and others, 2011). Natural resource agencies that step away from proactively addressing wildlife disease are in essence turning away from the teachings of Aldo Leopold. They are also tacitly minimizing their voice relative to approaches for addressing emerging and resurging infectious diseases with wildlife origins.

Habitat management is a basic tenet and mainstay of modern North American wildlife conservation, and it provides an important linkage for preemptively addressing wildlife disease because as noted by Leopold (1933), doctoring the environment, not the animal, is the approach needed. A focus on habitat management is also consistent with society's continually evolving focus on ecosystem health (Rapport and others, 1998). Nevertheless, perspectives about whether or not disease intervention is needed and in what form are confounded by various factors.

Disease occurrence may be considered beneficial or detrimental depending on the species involved. In addition, the outcome from intervention may result in unanticipated consequences; for example, remote-delivery oral vaccination of red foxes in Europe has been highly effective in combatting rabies (appendix 1). The soundness of that program is not in question; yet spared death from rabies, the fox population increased greatly in some areas and devastated ground-nesting bird populations. In addition, large numbers of foxes have colonized urban areas within some parts of Europe and brought with them other pathogens such as *Echinoccocus multilocularis*, a zoonotic **tapeworm** that can cause serious illness and death in humans (Deplazes and others, 2004; König, 2008).

In some instances, pathogens have been used intentionally for biological control of exotic and pest species and diseases that they carry. Because disease is a natural factor within biological systems, it is also true that some pathogens have functional roles in maintaining ecological services and biological relations among species. A key point is that we do not live in a **gnotobiotic** (germ-free) environment. Except for highly unusual circumstances, it would not be desirable to do so because most, if not all, of those living in such an environment would likely have reduced immunocompetence that would leave them highly susceptible to infectious agents that gain entry to that environment. That level of susceptibility could be akin to the rapid extinction of species eons ago attributed to the occurrence of uninhibited infectious disease events prior to the evolution of functional host immune systems (Brothwell and Sanderson, 1967).

Aldo Leopold on Disease

"The role of disease in wild-life conservation has been radically underestimated." (Leopold, 1933)

Aldo Leopold, shown here at his shack in Sauk County, Wisconsin, is considered the "father of modern wildlife management" in North America. His book of essays, "A Sand County Almanac," and his classic treatise on "Game Management" remain important contributions to wildlife conservation that are still studied by students entering this field of endeavor. (Photo used with permission from the Aldo Leopold Foundation.)

With the exception of ongoing investigations initiated near the start of the 20th century by the California Department of Fish and Game and the U.S. Department of Agriculture's Bureau of Biological Survey (BBS) into the cause of "western duck disease," wildlife disease had been afforded little attention by the conservation community when Leopold published "Game Management" in 1933. Therefore, it is meaningful that Leopold devoted a chapter of that book to the importance of disease and approaches for its control. He himself was not actively involved in disease investigations. However, during 1931 Leopold attended the Matamek Conference in Labrador, Canada (Huntington, 1931) where he interacted for 9 days with an elite, multidisciplinary group of specialists assembled to share their views about ecological relations, including causes for the dramatic cyclical oscillations in some wildlife population levels in northern areas. Disease was one of the factors that the group scrutinized (Meine, 1988).

Leopold gave disease pragmatic conservation meaning by focusing on its role in affecting wildlife populations, specifically stating, "Density limits of game populations are in many species probably set by disease." More importantly, his thoughts about addressing disease in wildlife are as foundationally sound today as they were in providing an initial path for wildlife managers to follow. For example, Leopold noted that:

"…the real determinants of disease mortality are the environment and the population, both of which are being 'doctored' daily, for better or for worse, by gun and axe and by fire and plow."

That commentary clearly identified ecological disruptions from human actions as the primary drivers for wildlife disease outbreaks. By extension then, habitat management was provided as a pathway for the conservation community to address disease.

Leopold's ecological focus on disease is also evident from his broad perspective of **game disease** being caused by living organisms (that is, microbes and parasites), chemical poisons, nutritional deficiencies, mechanical injuries, or combinations of these factors rather than focusing only on infectious disease.

"Wildlife disease control is a matter of doctoring the environment not the animal." (Leopold, 1933)

The Rabbit Bombs

"More than most subjects, the natural history of infectious disease must be set against a historical background and discussed [considered] in terms of continuing changes" (Burnet and White, 1972).

Since at least the 19th century, humans have attempted to wage biological warfare against nuisance and other unwanted wildlife. The most successful examples involve the use of myxomatosis (myxo) and rabbit hemorrhagic disease (RHD) as agents for mass population reduction of rabbits in Australia. (Illustration by John Evans)

During the early 1950s, field trials successfully resulted in the intentional establishment and spread of myxomatosis (appendix 1) as a cause for large-scale **epizootics** decimating European rabbit populations throughout Australia (Fenner and Ratcliffe, 1965). In essence, the causative virus was used as a biological weapon against the introduced European rabbit, which had become so abundant that it was causing large economic losses for agriculture. The virus is transmitted when an insect, primarily a mosquito or a flea, feeds on an infected rabbit and then takes its next blood meal from a **noninfected** rabbit.

By 1953 myxomatosis had spread to all of Australia. The disease was also intentionally introduced in Europe, Scandinavia, and South America. Control of rabbit populations has been variable (Yuill, 1970). Evolutionary factors have resulted in genetic resistance in rabbit populations and biological selection for less virulent strains of the virus following extremely high initial mortality rates (Fenner and Ratcliffe, 1965). Although myxomatosis is still an **enzootic** disease in Australia, other solutions to overabundance of rabbits continue to be pursued.

During the 1980s a new viral disease that is both highly lethal and contagious for domestic and wild rabbits appeared in Europe. Transmission of this virus is more widespread and varied than that of myxomatosis, because it occurs by multiple routes including direct contact with infected animals, indirect contact with virus-contaminated **fomites**, and **vector**-borne means. The causative **calicivirus** has since intentionally been introduced into Australia and New Zealand for biological control of European rabbit populations. Like myxomatosis, coadaptation between the calicivirus and the host appears to be taking place despite very high levels of initial rabbit mortality (Abrantes and others, 2012).

The natural attenuation of virulence for the European rabbit by two highly **pathogenic** viruses just noted does not negate the general need to address wildlife disease as a negative factor (Ewald, 1994). Personal encounters with dead and (or) moribund wildlife instantly stimulate my curiosity, because I have spent most of my professional career investigating and trying to address wildlife disease. The importance of evaluating randomly encountered field specimens has been repeatedly stressed by experiences associated with those situations. Most importantly, those experiences support the important role wildlife species play as sentinels for the early detection of novel disease events. Disease detection by sentinels facilitates the potential for wildlife managers to institute actions that can minimize the establishment and spread of infectious disease. The importance of this opportunity lies in the legacy of chronic losses and recurring epizootics that often follow the establishment of infectious diseases and by the general lack of success in eradicating those diseases following their establishment.

It is prudent to make a reasonable attempt to obtain at least a diagnosis for disease events encountered, because event diagnosis provides an initial risk assessment and requires that appropriate actions be taken to unite specimens observed with competent sources for making needed evaluations. In some instances, circumstances may limit the response to being able to provide only a concise report (including photos) of the place, date, and species involved rather than submitting specimens (fig. 1). "The Field Manual of Wildlife Diseases" (Franson and others, 2014) addresses how to handle properly such specimens, including how to safeguard oneself and others from potential hazards that may be present and key information to be reported.

The general importance of evaluating randomly encountered dead and (or) moribund wildlife is associated with the axiom "Familiarity breeds contempt." At issue is that repeated encounters with similar findings should not result in those apparently similar appearing events not being evaluated. Despite the illusion of similarity—"not all is always as it seems." Thus, personal experience has sensitized me

Figure 1. This northern pike with a large tumor was an unexpected catch during one of the author's wilderness finishing trips. The tumor was photographed and the location of the catch recorded for transmission to appropriate authorities following departure from the fly-in location. Supplemental information was included with that transmission, including the fact that no additional tumors were observed among any other of the substantial number of fish caught. (Photo by Milton Friend)

to view each wildlife mortality encounter from a perspective of—"expect the unexpected." In doing so I place great importance on wildlife as "the canaries in the coal mine," thereby, potentially providing an early warning of pending health issues. This is a reasonable expectation because faunal species are essentially present in all components of Earth's environment that support human life. Thus, the dysfunction of fauna adapted to specific ecological niches is of broad general concern, and in that context, wildlife mortality may also reflect pending disease emergence in humans or other species. A classic current example is West Nile virus (WNV; appendix 1), because in many areas, dead birds typically appear before human or horse cases and are used as sentinels to monitor for WNV presence (fig. 2).

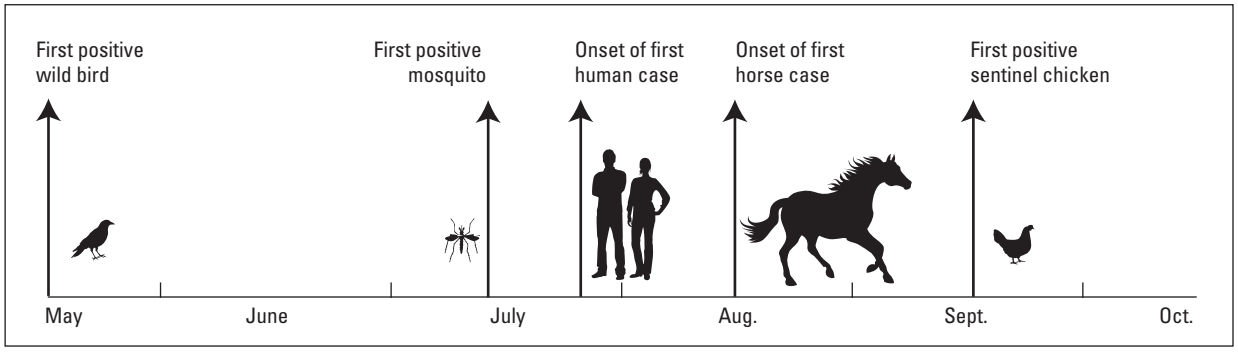


Figure 2. West Nile fever, a viral disease transmitted by mosquitoes, first appeared in the United States during 1999. The timeline for the appearance of infection during 2000 illustrates the value of wild birds as an indicator of viral activity well in advance of the appearance of this disease in other commonly infected species. The lead time provided by wild bird indicators facilitates preemptive actions by human and domestic animal health agencies.

Changing Times and Changing Needs

In a utopian world, all wildlife mortality encounters can be addressed perfectly; however, choices generally need to be made within the pragmatic circumstances of the real world. At issue is the question of what types of choices should be made. The precise answer is—"it depends." That dependency is time sensitive, subject to external factors imposed by others and (or) circumstances, and is value laden.

Before there was a need for wildlife conservation, there was no need to address wildlife disease. Conversely, once wildlife conservation became necessary to sustain various species and populations, it also became necessary to address mortality factors, including disease. However, it has not been until recently that this factor began to receive much attention by the wildlife conservation community. Today, conservation efforts are often confounded by losses from disease, yet without conservation efforts to maintain adequate population numbers, many offending infectious diseases cannot sustain themselves.

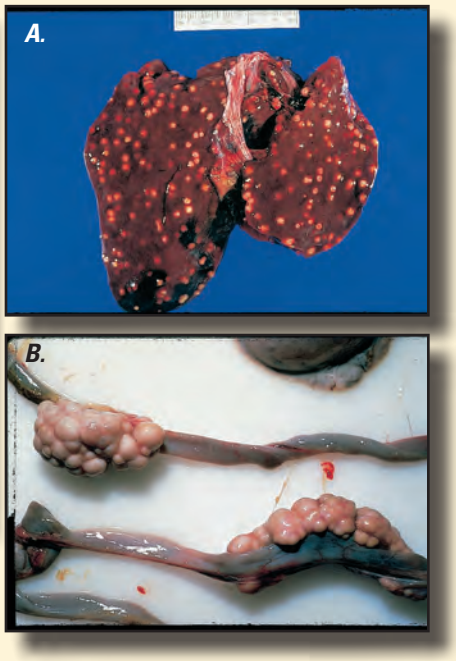
Disease has become a major factor in the retardation and failure of various wildlife conservation activities to maintain and (or) restore desired population levels. The successful propagation of species in captivity, or by the surrogate raising of young in nature, to provide for the enhancement or reestablishment of wild populations may be an operational success. Yet if disease subsequently prevents maintaining or restoring desired or needed population levels, then the species is not saved beyond being a perpetual "ward of the state" that is maintained in a confined environment or is repeatedly provided treatment in the wild to sustain its survival. Thus, successfully addressing disease is as important as successful propagation programs, and disease mitigation raises major questions relative to the costs associated with population enhancement if disease is also not addressed.

The examples here highlight the importance of considering and addressing disease in all aspects of endangered species conservation programs. The following time era highlights involving other wildlife conservation-wildlife disease relations provide additional "Points to Ponder" in considering personal choices to be made in addressing wildlife disease.

The petroglyph depicts an intimacy during earlier times between humans and other aspects of the natural world that stands in sharp contrast to the highly mobile and technologically advanced perspectives of modern society symbolized by the jetliner. These changing times are accompanied by changing needs in addressing wildlife disease. (Petroglyph photo by Scott Catron ; airplane photo by Lord of the Wings©)

Wildlife Disease and Whooping Crane Conservation

A whooping crane lies dead in a New Mexico field due to avian tuberculosis. This disease was a major factor for abandoning the 1980s Grays Lake, Idaho "foster parent" project, which attempted to establish a second flock of free-ranging whooping cranes in the wild. (Photo by Milton Friend)



A, Liver, and *B*, intestine of a Grays Lake whooping crane infected with avian tuberculosis. (Photos by James Runningen)

The whooping **crane** population in the wild had been reduced to about 21 birds in 1945 and resulted in turning to captive breeding as a means for bolstering the wild population. A captive colony was initiated at the U.S. Fish and Wildlife Service (FWS) Patuxent Wildlife Research Center during the late 1960s as part of this effort. Multiple disease and nutritional problems were encountered that initially impaired survival of whooping cranes in captivity but were eventually resolved. The first chick fledged at Patuxent in 1976, and that breakthrough event was followed by 255 eggs being produced between 1975 and 1991 for various program uses. However, additional disease events, such as a 1984 epizootic of eastern equine encephalitis (EEE; appendix 1) that killed two males and five females within the breeding flock resulted in the establishment of an additional captive colony at the International Crane Foundation (ICF) in Wisconsin. In 1989, 22 cranes from the Patuxent flock were transferred to ICF for that purpose (Ellis and others, 1992).

The first reintroduction effort for whooping cranes involved the placement of nearly 300 whooping crane eggs, from the Wood Buffalo National Park (Canada) wild flock or from the Patuxent captive colony, in greater sandhill crane nests at Grays Lake, Idaho. The objective was to create a second whooping crane population in the wild that would return to Grays Lake as their breeding grounds and winter with their foster sandhill crane parents along the Rio Grande in west-central New Mexico (Drewien and Bizea, 1978). The first eggs were placed under sandhill cranes at Grays Lake in 1975 and the last 12 of 289 of such eggs in 1988. The Grays Lake flock of whooping cranes reached a high of 33 birds in 1984–85, but by 1991 it had declined to 13 birds. After fully evaluating the situation, a 1990 decision was made to abandon this experiment and pursue other means for enhancing the whooping crane population level in the wild (Ellis and others, 1992).

Multiple factors are responsible for recruitment within the Grays Lake whooping crane flock not being able to keep pace with mortality, thereby setting that flock on an extinction path. Avian tuberculosis, *Mycobacterium avium*, is one of those factors (appendix 1). Although this disease is uncommon in wild birds within North America, it was one of the major factors in the death of whooping cranes that fledged within the Grays Lake flock. **Postmortem** evaluations by pathologists at the National Wildlife Health Center disclosed avian tuberculosis to be present in many of the cranes examined (figs. *A, B*). In contrast, earlier wild bird investigations published during 1941 and 1978 disclosed that only 0.3 percent of 3,000 **waterfowl** necropsied in the western United States (Quortrup and Shillinger, 1941) and 0.6 percent of free-flying birds in British Columbia (MacNeill and Barnard, 1978) were diagnosed with avian tuberculosis. Personal encounters with this disease in wild birds have been rare and primarily associated with **upland game birds** such as pheasants and chukar partridge reared for sporting and display purposes. Avian tuberculosis has been an important disease of domestic poultry in some geographic areas, within some aviculture and zoological collections, and within various wild bird captive-breeding programs including Wildfowl Trust facilities in England (Beer, 1988).

Lead and the California Condor

A program for breeding captive California condors has provided progeny of the endangered bird for release into its historic habitat, but lead present in the condor's animal foodbase continues to jeopardize the survival of the species in the wild. (Photo by the U.S. Fish and Wildlife Service)

A wing-tagged California condor soars over its terrain, searching for its carrion foodbase—hopefully one not contaminated by lead bullet fragments within a dead animal. (Photo by the U.S. Fish and Wildlife Service)

Lead poisoning (appendix 1) due to the ingestion of particulate lead in food items such as deer carcasses and wildlife **offal** was the major cause of mortality in California condors during the 20th century, and it resulted in the few remaining wild condors being brought into captivity during the 1980s to avoid species extinction. The California condor became extinct in the wild in 1987 when the last remaining free-ranging condor was added to a captive breeding flock of 27 other condors. The success of the breeding program resulted in condors being released into their native range since 1992 (Green and others, 2009). Despite the success obtained by that program in providing progeny for reestablishing California condors in the wild, lead remains as a persistent challenge for the survival of this species in nature.

Lead poisoning is the most frequently diagnosed cause of death among the Grand Canyon cohort of condors, despite intensive efforts to monitor condor health through annual retrapping of the birds, monitoring blood lead levels, and clinically treating those birds with elevated levels of lead (Parish and others, 2007). Captive breeding can produce a substantial number of progeny for release into the wild. However, without resolution of the lead issue, population attrition due to lead toxicosis is likely to result in species extinction from the wild despite continued clinical intervention. This costly program is primarily supported by private sector funding and may not be sustainable unless government actions are taken to reduce substantially the lead burden for condors in the wild.

The focus on the development of technology to advance human well-being has been a pursuit of humanity since primitive people appeared on Earth. Thus, "In the Beginning" that focus suppressed any needs to consider wildlife conservation or wildlife disease. (Drawing from Pixababy.com[DOS])

In The Beginning

The early history of humanity contains numerous indications of protection being afforded to various wildlife species. Tribal taboos, Mosaic Law, social customs, and government edicts are examples of actions imposed since the onset of civilization itself. Such actions were likely associated with religious beliefs, superstitions, and utilitarian needs affecting human survival. Those early "prohibitions" and "regulations of take" can loosely be considered passive forms of wildlife conservation. Disease also has been present since life began on Earth and is hypothesized to have been responsible for some early **megafauna** extinctions (MacPhee and Marx, 1997; Poinar and Poinar, 2008). Extinction is the maximum impact that can result from unbridled disease occurrence. For early humans, epizootics and species extinctions were likely disjointed occurrences beyond their understanding and were certainly beyond any capacity for human intervention on behalf of wildlife populations. Furthermore, if successful intervention during that time had been accomplished, it may have changed early evolutionary processes taking place. Even today, major differences exist relative to the types of human intervention that is beneficial or harmful in addressing disease in wildlife, especially the practice of culling.

Recent attempts to address bovine tuberculosis in the United Kingdom have resulted in further disease spread rather than disease control because of disruption in the social structure of some European badger populations (Carter and others, 2007; Weber and others, 2013). Other recent studies suggest that the manipulation of host density can impose strong selective pressures on pathogen virulence that also cause ecological changes resulting in unexpected consequences relative to disease outcomes (Bolzonia and De Leo, 2013).

The situations just cited are not argument for not addressing wildlife disease. Instead, they highlight that from a conservation perspective, wildlife disease must be considered in the context of the dynamics of evolution, environmental change, and societal support for sustaining appropriate wildlife population levels. Furthermore, every living pathogen, be it a **bacterium**, virus, rickettsia, fungus, **protozoan**, **metazoan** parasite, or other life form, is as much the product of adaptive evolution as its host (Burnet and White, 1972). The opportunities for such adaptation are often provided by human-driven ecological changes. When such changes favor the occurrence of disease, the impacts for wildlife can be far reaching. For example, from an ecological perspective, "disease can change the face of landscapes by removing keystone, abundant and endangered species, or ecosystem engineers" (Mouritsen and Poulin, 2002). Thus, the extent to which wildlife disease is or is not addressed can have major implications for wildlife stewardship.

From an evolutionary perspective, it is important to recognize that bacteria have been the dominant form of life on Earth for some 3 billion years. During early times bacteria had the Earth all to themselves and diversified to occupy every possible niche. It was not until 600 million years ago that multicellular organisms made up of eukaryotic cells emerged and then evolved to eventually form the plants and animals we know today (Crawford, 2007). Undoubtedly, many of the pathogens causing disease today have an ancestry extending back into geological time for many millions of years, and they have contributed in a marked degree to the extinction of various early higher life forms. Furthermore, in doing so, those pathogens also likely contributed to the extinction of those diseases (Brothwell and Sanderson, 1967). Thus, the continued presence of those pathogens was linked to further evolution by adaptive processes such as those facilitating host switching.

Early colonists in the New World were highly dependent upon the wildlife present to sustain and advance human well-being. Overexploitation of those once vast wildlife resources soon led to protectionist efforts to slow their demise. (Illustration from the Library of Congress)

Colonial America

The great abundance of wildlife present within much of the Americas during early colonial time buffered against any human concerns about major wildlife mortality events. For example, there is no evidence that a 1656 **pelican** die-off in the West Indies stimulated any investigations into its cause, although the event was described as being of such magnitude "…that their dead bodies covered many islands. …all of the shores of the islands of St. Alouise, St. Vincent, Becoüya, and all the Grenadines were covered with the bodies of these dead birds" (fig. 3) (Fleming, 1871). Such inaction is understandable considering that wildlife conservation did not begin to become a concern within the United States until nearly a century after initial colonial settlement. Early conservation actions were primarily focused on hunting controls, including the methods for taking animals, oriented at simply extending the longevity of populations rather than restoring population levels. Active management of factors unrelated to hunting and trapping that also were contributing to the demise of wildlife populations was not yet being considered. Furthermore, despite the magnitude of wildlife population declines, the first hunting closure for a species for a multiyear period was not initiated until 1718, for white-tailed deer in Massachusetts. By the time of the American Revolution, wildlife population declines for many species had reached such levels that 12 of the 13 colonies had closed the hunting seasons for specific species, and some colonies had enacted regulations prohibiting various methods for taking wildlife (Palmer, 1912).

Wildlife disease is infrequently documented within the accounts of animal disease in America during the first 200 years of settlement (Bierer, 1974; Fleming, 1871; Webster, 1799). In part, this may reflect a relatively low number of the infectious diseases present today having become established within New World wildlife populations. Other factors include limitations in technology for the isolation and identification of some pathogens that may have been present and ecological factors during that time that may not have been favorable for the establishment and spread of various pathogens within and between wildlife populations. Specifically, the greater general degree of separation during colonial times than today between wildlife populations and between wildlife and domestic animal populations may have served to contain infectious disease outbreaks within specific species and populations as self-limiting events. In addition, the draconian landscape changes and wildlife population declines imposed by humanity of that time may have prevented the establishment and spread of density-dependent infectious disease agents.

Rabies and sarcoptic mange in red foxes (appendix 1) appear to be the most common wildlife disease events that were recorded. Rabies was first reported in domestic dogs and presumably was brought to America from the Old World in that species. It became enzootic and an epizootic disease in America, and its movement across the continent is attributed to affected domestic dogs accompanying the westward movement of settlement and the infection along the way of wild **carnivores** by those dogs (Smithcors, 1958). Both the importation of rabies and its movement across America were facilitated by the prolonged incubation period, typically 1–3 months (seldom more than 6 months), which often occurs between infection and the appearance of clinical signs of rabies. Infectious diseases with short incubation periods would have had far greater difficulty in being introduced by humans and their domestic animals, because the slow pace of travel at the time would have resulted in most infected individuals dying along the way or recovering from their infections.

Figure 3. Wildlife mortality, such as the 1656 pelican die-off in the West Indies, was not a cause for concern during colonial time, even when it was extensive. (Photo by Milton Friend.)

The intent focus of the child on the computer screen exemplifies society's current attachment to our electronic world and reflects reported concerns of an increasing detachment of society from nature (see Louv, 2005). (Photo by Andrew Stawarz)

The Modern Era

The start of the 20th century brought with it the modern era of natural resources conservation in America. Within that century, the first 50 years were of the greatest formative importance for wildlife conservation. The last 50 years embodied similar importance for the integration of wildlife disease within the philosophy and practice of wildlife management.

The First 50 Years

The basic framework for the modern era conservation movement evolved from President Theodore Roosevelt's passion for wildlife and for nature in general. That passion was evident in his keynote address to the 1908 Conference of Governors in which he "recast conservation as a public (rather than private) and moral (rather than economic) issue…and linked conservation to the broader themes of civilization and nationalism" (Sheffield, 2010). The Roosevelt conservation doctrine for wildlife, forests, ranges, and water power recognized that:

1. these resources are renewable (if properly managed) organic components of outdoor resources that needed to be considered as one integral whole;

2. their conservation is best achieved through wise use as a public responsibility, and their private ownership as a public trust; and

3. science is a tool for discharging that responsibility (Leopold, 1933).

President Theodore "Teddy" Roosevelt, on his horse in Colorado in about 1905, exemplifies the "hands on" interactions with nature that gave birth to the modern era of wildlife conservation at the start of the 20th century. (Photo from the National Park Service)

In essence, the first component of the Roosevelt conservation doctrine encouraged an ecosystem health approach nearly a century before such a paradigm shift began to be considered as a needed approach for a healthy planet. In addition, by extension of the doctrine's public responsibility charge, stewardship of natural resources was assigned to natural resource agencies, and personnel from those agencies gained responsibility for the well-being of wildlife resources. Thus, by fiat and statutory authorities, wildlife resource agencies were embodied with responsibility for addressing challenges from disease to the sustainability of appropriate wildlife population levels.

In keeping with the above responsibility, the start of the 20th century saw disease occurrence reach a level that mandated intervention. That need was driven by catastrophic **waterbird** mortality events during 1909–13 that were of unprecedented magnitude. Reasonable estimates based on field observations and investigations placed those losses in the "millions." Of paramount importance were the epizootics of "western duck sickness" (avian botulism) in California and those on the Bear River marshes along the Great Salt Lake in Utah (fig. 4). Both situations resulted in the development of wildlife disease programs. The California investigations fostered development of the Wildlife Disease Investigations Laboratory within the California Department of Fish and Game. The Utah investigations led to the development of the Bear River Wildlife Disease Research Station as a component of the U.S. Department of Agriculture (USDA) Bureau of Biological Survey (BBS; see Cameron, 1929, and Friend, 1995, for historical accounts of the BBS).

The magnitude of those "western duck sickness" events demanded government response because of the dismal state of many of America's natural resources. Failure to address disease issues of such great scale in highly valued wildlife species would likely have directed the question "Why bother?" to the restrictions that had been imposed on harvesting those species. Thus, for the first time in American history, wildlife disease had become somewhat of a mainstream issue for the conservation of natural resources; however, that focus on disease was born out of crisis and soon waned because it had not emerged internally as an accepted need within the developing field of wildlife conservation. For example, in 1908 the *American Veterinary Review* (Lieutard and Ellis, v. 33, p. 334) reported, "again as for several seasons past, wild ducks are dying by hundreds in marshy regions bordering Lake Erie…." Nevertheless, those events failed to result in similar responses as those for "western duck sickness." Although wildlife disease investigations continued within a few conservation agencies from at least the early 20th century forward, resource allocations for their conduct were minimal due to other needs being seen as higher priorities.

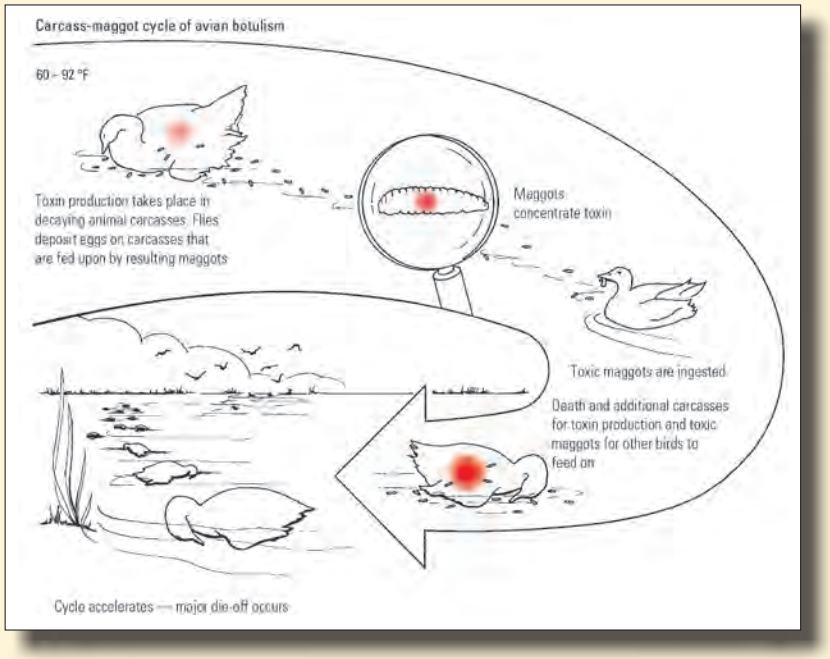
Most North American wildlife disease evaluations prior to the end of World War II were conducted by university and other non-wildlife agency personnel due to the failure of the conservation community to invest sufficient resources in disease investigations. Furthermore, as in the current time, many of the wildlife disease investigations by agencies required collaboration with other agencies and with university personnel to provide the technical expertise, specialized facilities and equipment, and fiscal resources needed. However, collaboration often resulted in a strong emphasis on basic science and research involving the ecology of infectious wildlife diseases afflicting humans and domestic animals, and most of the scientific papers about wildlife diseases published during that era reflect a focus on discovery (surveys), parasite life cycles, and species susceptibility to various pathogens. The primary wildlife conservation oriented research focused on "western duck sickness" and the role of disease in population cycles of **aquatic furbearers, grouse**, snowshoe hare, and some other wildlife species.

Lead poisoning preceded "western duck sickness" as an identifiable disease in North American wildlife, but it was not researched until early in the 20th century. Four years after publishing his findings on "western duck sickness," Alexander Wetmore published *Lead Poisoning In Waterfowl* as a 1919 USDA Bulletin (no. 793) summarizing his field and laboratory investigations of this disease. Although neither of these diseases is infectious, both were being found to cause substantial bird losses. In 1934, "western duck sickness" was shown to be a form of botulism, and this disease continues to evolve today in response to human-induced environmental changes. (See Rocke and Friend [1999], Friend [1999b], and appendix 1 in this publication.)

Figure 4. The 1910 "western duck disease" (avian botulism) in Utah's Bear River marshes (Great Salt Lake) and similar events across western North America killed millions of waterfowl and other wetland birds. (Photo by Alexander Wetmore)

Exotic Species, Food Webs, And Avian Botulism

The introduction of infectious disease agents is a general concern associated with the potential biological consequences from exotic and invasive species. Exotic species introductions may also contribute to the emergence of non-infectious diseases. Avian botulism ("western duck sickness") provides recent examples of extensive wildlife losses associated with modifications in the classical ecology for this disease by exotic species.



A, The carcass-maggot cycle of avian botulism. (From Rocke and Friend, 1999)

Type C avian botulism is the most important disease of North American waterbirds relative to numbers of birds killed. The ecology of this disease is reasonably well known and typically involves production of the causative bacterial toxin within decaying bird carcasses (fig. *A*). *Clostridium botulinum* type C exists in nature as a spore forming bacterium. Decaying bird and other animal carcasses provide an appropriate anaerobic environment and other conditions needed to stimulate the germination of spores previously ingested by the now dead birds. Botulinum toxin is produced as a byproduct of the bacterium's vegetative cell growth and multiplication, and the toxin becomes concentrated in the bodies of flesh-eating maggots that consume those carcasses. The toxin is then transferred to birds that consume the maggots (see chap. 38 Rocke and Friend, 1999).

In 1996 a type C avian botulism epizootic at California's Salton Sea killed nearly 9,000 American white pelicans (fig. *B*). More than 1,200 endangered California brown pelicans were also affected and (or) died during the epizootic (figs. *C–D*). Smaller scale epizootics occurred during subsequent years (Nol and others, 2004; Rocke and others, 2004). Both species of pelicans primarily feed on live fish that they pursue and capture by different types of feeding strategies. Brown pelicans dive from various heights above the water surface, while American white pelicans typically feed in swimming flocks that pursue schools of fish within the near surface areas, using their bills as "scoops" to capture their prey. Thus, the feeding strategies for pelicans are inconsistent with typical epizootic mortality from type C avian botulism. However, investigations by scientists from the National Wildlife Health Center disclosed, "that the live tilapia population in the Salton Sea harbors *C. botulinum* capable of producing necrotoxin within their GI tract…." Tilapia are exotic freshwater species that also established themselves in this highly saline ecosystem. Their great abundance during the era of the botulism epizootics resulted in tilapia being the primary food base for a wide variety of fish-eating bird species that became attracted to the Salton Sea because of the unusually abundant food base. Unlike other fish species, tilapia are highly susceptible to *C. botulinum* toxin. The

B, Bags of pelican carcasses about to be incinerated at the U.S. Fish and Wildlife Service Sonny Bono National Wildlife Refuge. (Photo by U.S. Fish and Wildlife Service)

neurological clinical manifestations that occur in affected tilapia result both in the likelihood that these fish will attract foraging birds and that they will have reduced avoidance response to predation.

Avian botulism outbreaks involving fish-eating birds have historically been associated with type E strains of *C. botulinum*. Such events have been the cause of periodic mortality of fish and birds within Lake Michigan since the 1960s (figs. *E–G)*. Unlike type C avian botulism, much of the ecology of type E

Exotic Species, Food Webs, And Avian Botulism

avian botulism remains to be determined. Since the start of the 21st century, type E botulism has emerged as a major disease of fish-eating birds within the Great Lakes Basin to the extent that epizootics now commonly occur in Lake Michigan. Lakes Erie and Huron experienced their first epizootics in 1998 and Lake Ontario in 2002.

The results from ongoing investigations suggest that introduced **Dreissenid mussels**, such as the zebra mussel and the quagga mussel, may enhance the growth of benthic algae and associated invertebrates. The subsequent death and decomposition of those **benthic** species may then result in conditions needed for spore germination and toxin production. Transfer of the toxin to fish-eating birds is thought to result from the round goby, an introduced exotic fish species, feeding on invertebrates and then itself being feed upon by other species of fish and (or) birds. In addition, fish that have fed on gobies may also be fed on by birds. At this time, the hypothesis just presented has considerable support but remains to be scientifically established. Nevertheless, it is likely that the introduction of exotic species into the Great Lakes food webs will be proven an important factor in avian botulism ecology within that Basin just as has occurred at California's Salton Sea.

C, Pelicans showing signs of intoxication by type C botulism. Pillowcases placed over the birds at the time of their removal from the Salton Sea are kept wet to help cool the birds in the 100 °F temperature until they reach the trucks for rapid transfer to the *D*, U.S. Fish and Wildlife Service onsite treatment and temporary holding facility. (Photos by Milton Friend)

E–*G*. The Great Lakes have become a major foci of mortality due to type E botulism in common loons and other fish-eating birds since the start of the 21st century. (Photos from Sleeping Bear Dunes, National Park Service)

By the 1930s, the captive propagation of wildlife (fig. 5) was being pursued with vigor as a wildlife conservation action. The primary purposes were to: (1) enhance population levels of important fur and game species by releasing propagated stock into the wild; and (2) provide immediate, short-term recreational opportunities by stocking animals to be hunted. That emphasis on captive propagation shifted much of the conservation community's focus on wildlife disease from disease as an abstract component of nature to a pragmatic issue of game husbandry. Thus, as for the domestic animal industry, disease would be addressed because wildlife that did not survive the captive-propagation cycle reduced the numbers of those animals that could be released to the wild, or in the case of the domestic animal industry, marketed. Therefore, the new focus on wildlife propagation provided somewhat of a response to the question of "Why bother?" In some instances, the answer may also have served to increase within-agency capabilities for wildlife disease investigations (see chap. A1 in the "Field Manual of Wildlife Diseases" (USGS, FWS, and NPS, 2014), and appendix 1, table 1–2 entries for the 1930s in this publication).

Unfortunately, the new focus failed to adequately bridge disease relations between captive-reared and free-ranging animals. Conservation agencies gave little, if any, consideration to disease prevention actions such as the establishment of health certification standards and processes for minimizing the potential for disease to accompany propagated wildlife released into the wild (fig. 6). Similarly, little, if any, consideration was given to the potential for enzootic disease within release areas to counteract the potential benefits from such releases. Both situations continue to contribute to wildlife losses from disease today.

Brucellosis (appendix 1) is an example of a current lingering wildlife disease issue that has its roots in the captive propagation of American bison under conditions that facilitated the infection of bison by cattle. Bison in the Greater Yellowstone-Jackson Hole Ecosystem became infected with *Brucella abortus* as early as 1917 and by 1932 had transmitted the disease to some elk populations in that area (Quortrup, 1945). Brucellosis was subsequently eradicated from cattle; however, cattle within the area of current bison and elk infection are subject to a continuing risk of obtaining brucellosis from those wildlife. Furthermore, the presence of brucellosis in those elk and bison prevents the use of excess animals for various conservation programs.

The first half of the 20th century also was a period of increasing interest in wildlife disease by public health and domestic animal disease investigators because of the occurrence of several reemerging infectious diseases. Plague (appendix 1), *Yersina pestis*, was introduced into California during the third plague pandemic (Modern Pandemic) that began in China in 1855 and reached San Francisco in 1900. **Commensal Old World rodents** (rats) and their fleas had primary roles in this introduction. In 1902 the causative bacterium was first found in commensal rats within the United States. It was then isolated from infected California ground squirrels in 1908 (Abbott and Rocke, 2012).

Tularemia (appendix 1) was perhaps the first mammalian infectious disease of wildlife conservation concern in America due to recurring epizootics involving aquatic furbearers of great economic importance for America's fur trade until the

Figure 5. *A*, Captive propagation of waterfowl and *B*, exotic species of upland gamebirds, such as the Hungarian partridges being released in this photo, were a major focus for early wildlife conservation efforts to restore North America's greatly diminished wildlife populations. (Photos by Milton Friend)

Figure 6. Health evaluations are an important preemptive action for minimizing the potential for disease introductions by the translocation of free-ranging wildlife to new locations and for the release of captive-reared wildlife into the wild. Such actions need to be more aggressively implemented. (Photo by Milton Friend.)

early 1960s (fig. 7). Rabies, plague, and tularemia are all examples of wildlife serving as canaries in the coal mine for those sensitive to the messages being conveyed. After becoming established in American wildlife, rabies and plague continued to extend their geographic distribution as enzootic diseases. Furthermore, the potential for disease transfer between wildlife and domestic animals extends the question "Why bother?" to encounters with dead domestic animals within and (or) on areas adjacent to wildlife habitat. This is especially relevant for shared habitat such as grazing and range lands.

The movement of infectious disease between domestic animals and wildlife (in both directions) is a major factor driving emerging infectious diseases (EIDs) and society's response to these diseases (table 1). Thus, effective monitoring for disease is needed for both components and should be considered a shared responsibility that benefits both sectors when done well. It also is important to consider that disease need not be a direct cause of mortality in the animals of either sector to require vigorous response.

The Leopold Factor

The science-based approach to wildlife conservation called for by the Roosevelt conservation doctrine was greatly advanced by Aldo Leopold, who published America's first science-based treatise on game management (Leopold, 1933). Wildlife disease was elevated in importance as a conservation issue in that treatise. Soon after publishing Game Management, the University of Wisconsin provided Leopold the opportunity to develop North America's first advanced degree wildlife ecology training program, and the first graduate students entered that program in 1939. That monumental action of providing specific science-based education for guiding wildlife management had awaited three decades after the Roosevelt conservation doctrine was issued. That same year, the U.S. Fish and Wildlife Service (FWS) was created within the U.S. Department of the Interior (DOI) to provide Federal leadership for the conservation of wildlife species shared across state borders and to provide general assistance in the restoration of Americas wildlife resources.

Figure 7. The liver from this American beaver found dead on a national wildlife refuge exhibits classic lesions from tularemia. Public access to much of the refuge was closed for fishing and other water-related activities until after the epizootic had subsided. (Photo by James Runningen)

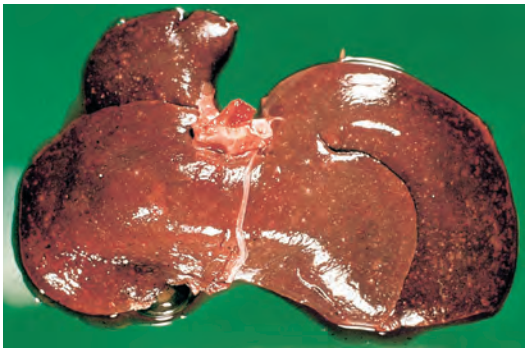


Table 1. Examples of emerging infectious diseases (EIDs) with pathogen transfers between domestic animal and wildlife hosts (and vice versa).

[77 percent of livestock pathogens and 91 percent of domestic carnivore pathogens are known to infect multiple hosts, including wildlife (Cleaveland and others, 2001)]

Disease	Pathogen	Pathogen type	Primary hosts involved		Comments
			Domestic animals	Wildlife	
Newcastle disease	Avian paramyxovirus type 1	Virus	Poultry	Cormorants	Large-scale epizootics have afflicted cormorants in North America since the late 1990s.
Canine distemper	Canine distemper virus (CDV)	Virus	Dog	Wild carnivores, marine mammals	Major wildlife conservation issues due to CDV epizootics involve threatened and endangered species.
Bovine tuberculosis	*Mycobacterium bovis*	Bacteria	Cattle	European badger, brushtail possum, deer	Bovine tuberculosis is a recent focus for conservation of free-ranging white-tailed deer in Michigan.
Brucellosis	*Brucella abortus*	Bacteria	Cattle	American bison, elk	Brucellosis is a persistent problem in the Greater Yellowstone Basin.
Echinococcosis	*Echinococcus multilocularis*	Cestode parasite	Dogs, cats	**Arvicoline rodents** (intermediate hosts), foxes (**definitive host**)	Since 1960s, echinococcosis has spread from the tundra zone of northern Canada to central regions of United States.
Mange	*Sarcoptes scabiei*	Ectoparasite	Dogs	Foxes, many other species	Mange is the most common cause of death of chamois and ibex in Europe. It has caused extinctions of isolated fox populations.
Toxoplasmosis	*Toxoplasma gondii*	Protozoan parasite	Cats, livestock	Carnivores	Sea otters recently found to be fatally infected with toxoplasmosis.

Aldo Leopold's Legacy of Professional Wildlife Management

Aldo Leopold's iconic shack in Sauk County, Wisconsin, in winter. (Photo used with permission from the Aldo Leopold Foundation)

At the start of the 20th century, little science was directly associated with the American conservation movement because academic degree programs in wildlife conservation did not yet exist. Furthermore, it would not be until 1930 that the American Game Institute (now the Wildlife Management Institute) would call for the development of a comprehensive policy for wildlife conservation. Another 7 years would also pass before the official birth of The Wildlife Society as the first professional organization focused on wildlife management (Organ, 2012).

Aldo Leopold was one of those asked to assist in the drafting of the American Game Policy but, by far, his greatest contribution to the advancement of wildlife conservation within the United States was the 1933 publication of his treatise "Game Management". It has been stated that book, "More than any other book before or since changed wildlife management from an art to a science"… and "gave birth to a profession that is today known as Wildlife Management with all the accoutrements of a viable discipline" (McCabe, 1987). Leopold clearly advanced the role of science in wildlife conservation through his publications, presentations, and by his role as Professor of wildlife ecology at the University of Wisconsin, Madison. Those contributions, and others, rightly earned Leopold recognition as the "father" of professional wildlife management in North America.

Leopold's legacy includes the development, institutionalization, and advancement of science-based wildlife conservation. His contributions have continually been illuminated through the contributions of his graduate students, many of whose careers, in keeping with Leopold's philosophy, have been highlighted "by reappraising things unnatural, tame, and confined in terms of things natural, wild, and free" (quotation is the end of Leopold's last sentence in the foreword to "A Sand County Almanac" (1949).

Leopold's views on wildlife disease also were ahead of his time and warrant current consideration, because they are as insightful and true today as they were in the 1930s. At that time, many naturalists and conservationists perceived wildlife disease to be "a part of nature" and, at worse, only having very transient impacts on wildlife populations. Thus, there was little acceptance of any need to be concerned about disease with the possible exception of "western duck disease." Despite the frequent reoccurrence of that disease, the prevailing attitude was that no meaningful intervention was possible.

Leopold contended that the impacts of disease on wildlife are largely underestimated. He also correctly noted that organized effort to understand and control wildlife disease (during the 1930s) were still in its infancy. That truism was borne out by the fact that the Wildlife Disease Association (the first professional society for this subject area) did not come into existence until 1951. His position that wildlife disease is primarily an ecological issue driven largely by human actions was decades ahead of the thinking of most others within the conservation movement of that time. Today Leopold's position is a basic tenet of the current challenges from emerging infectious diseases and a principle associated with the increasing focus on "ecosystem health" as the approach needed to protect human health.

Leopold's "seeds of wisdom" about wildlife disease have been slow in germinating and taking root within the conservation field. Fortunately, an increasing number of wildlife management professionals are now recognizing the importance of disease as a challenge for wildlife stewardship.

Aldo Leopold's Legacy of Professional Wildlife Management

Professor Leopold taking the weights and other measurements of an American woodcock harvested during the day's hunt. (Photo used with permission from the Aldo Leopold Foundation)

By the start of the second half of the 20th century, advances in technology, such as radio telemetry tracking of free-ranging wildlife by this U.S. Fish and Wildlife biologist, were advancing wildlife conservation efforts. (Photo by the U.S. Fish and Wildlife Service)

The Second 50 Years

Infectious disease began to gain importance as a mortality factor for North American wildlife with 1955 white-tailed deer die-offs in New Jersey and Michigan due to epizootic hemorrhagic disease (EHD; appendix 1; fig. 8). Subsequent die-offs closely followed in numerous other geographic areas, some of which involved pronghorn **antelope** and mule deer (Trainer and Karstad, 1970). The occurrence of EHD in highly prized game species and inadequate capability within most state wildlife agencies to address those epizootics presented a "crisis situation." The Southeastern Association of Fish and Wildlife Agencies responded with the 1957 development of what is now known as the Southeastern Cooperative Wildlife Disease Study (SCWDS) at the University of Georgia, Athens. That program was the first North American regional wildlife disease program, and it provides member states with diagnostic and other assistance for addressing wildlife disease events and issues. The increasing North American focus on wildlife disease at that time was also reflected in the 1951 origin of the Wildlife Disease Association as the first professional society devoted to this subject area. The stated goals were "to serve as a means for bringing together all investigations involved in some phase of wildlife diseases and also afford a medium for communication and discussion among such investigators." In addition, by that same time the University of Wisconsin Department of Veterinary Science graduate training program contained a broad spectrum wildlife disease focus.

During the early 1960s, the rising tide of infectious disease became obscured by perspectives that clouded public and wildlife agency vision. Scientists and the public health community proclaimed that the battle against infectious disease had been won in the developed countries of the world, and it was time to redirect human disease investigations to more pressing chronic disease issues such as heart disease and cancer. The conquests achieved in addressing major infectious diseases of domestic animals were also being touted. Within the wildlife conservation community, the 1960s and early 1970s saw a major shift in public and agency focus to that of chemical contamination of the environment as a pressing grave threat for free-ranging wildlife populations.

General awareness of environmental contaminants was enhanced by the 1962 publication of Rachel Carson's Silent Spring, numerous other books on this subject, and count-

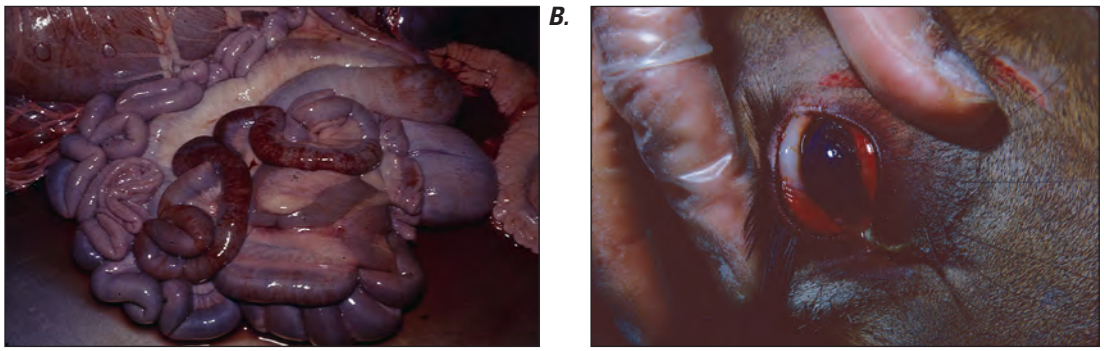


Figure 8. Hemorrhages in the tissues and organs, such as in the *A*, viscera and *B*, eye of white-tailed deer are common findings in deer and antelope that have died from epizootic hemorrhagic disease and bluetongue. (Photos by Milton Friend)

less popular media presentations targeting planetary perils associated with the "chemical plague" confronting human society (table 2). "Silent Spring" (fig. 9) quickly became a major catalyst for spurring America's environmental movement of that time. Natural resource agencies and others responded with major investments in scientific capacity, such as the staff, equipment acquisition, and facility development needed to research, document, and otherwise address the impacts of environmental contaminants on wildlife populations. I was personally fortunate to have been involved in those investigations, first as a university researcher assessing the effects of sublethal exposures of various contaminants on altering host response to infectious disease and then from a broader perspective as the FWS research project leader for pesticide-wildlife ecology studies in the western United States.

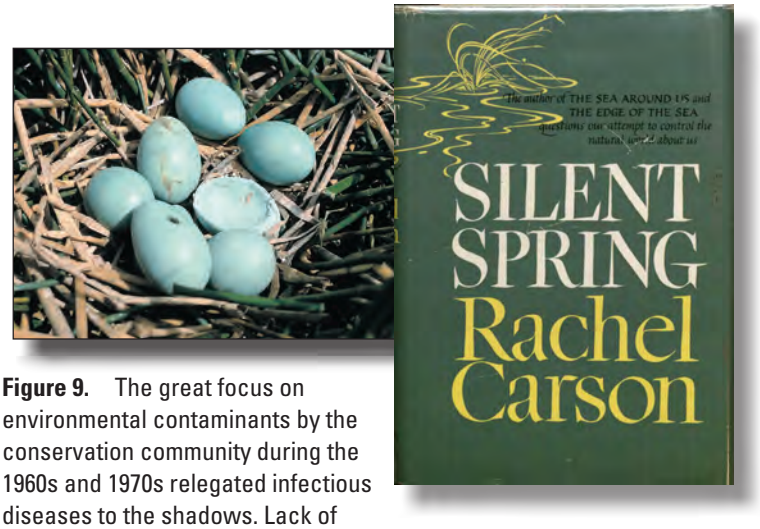


Figure 9. The great focus on environmental contaminants by the conservation community during the 1960s and 1970s relegated infectious diseases to the shadows. Lack of attention to these maladies facilitated their spread and entrenchment as diseases of concern. The collapsed egg shell in this white-faced ibis clutch exemplifies one of the outcomes associated with DDT use during the post-World War II era. (Photo by Milton Friend)

Table 2. Popular literature examples addressing the state of our environment during the 1960s–70s.

Title	Publication year	Author	Focus
Silent Spring	1962	Rachel Carson	Impacts of synthetic insecticides on the living communities of Earth.
Pesticides and the Living Landscape	1964	Robert L. Rudd	The influence of pesticides in nature and the costs borne by society for their use.
Our Plundered Planet	1968	Fairfield Osborn	Humanities' conflict with nature.
Our World in Peril: An Environment Review	1967	Sheldon Novick and Dorothy Cottrell (eds.)	The struggle and consequences of society's increasing dependency on technology.
The Politics of Pollution	1970	J. Clarence Davies III	The role of the public in advancing legislative actions to stem the impacts of pollution on our environment.
This Little Planet	1970	Michael Hamilton (ed.)	The theology of ecology and the moral issues involved in human roles in the conservation and pollution of our natural resources.

Combined findings from the plethora of environmental contaminant investigations conducted across North America and in numerous other countries provided a scientific basis for actions taken to address specific aspects of this issue. Those outcomes included bans on the use of various synthetic pesticides such as DDT (appendix 1) and demonstrated that wildlife health issues can be effectively addressed when adequate investments are made in documenting problems and gaining adequate ecological understanding of specific issues for developing and guiding appropriate corrective actions. These principles are directly responsive to the question of "why bother" and apply regardless of whether the disease agents involved are of chemical, microbial, parasitic, or of other origin. For example, current success achieved in the remote oral vaccination of wild foxes against rabies is the result of collaborative public health and natural resource agency investigations initiated at the start of the 1960s. Furthermore, the level of success achieved has resulted in a similar approach for some other wildlife species and for additional wildlife diseases.

Nevertheless, nature continued to lay down infectious disease "markers" that needed to be heeded by the conservation community but that were largely being ignored. Examples include the first appearances of avian cholera (appendix 1) in wild birds in several new geographic areas during the 1950s and 1960s and the first North American appearance of duck plague (appendix 1) during 1967 in a few wild waterfowl associated with a major epizootic in the Long Island, New York, white Pekin duck industry. Rapid and aggressive USDA actions in combatting the duck industry epizootic resulted in the official eradication of duck plague from the United States. Those events, the continuing spread of EHD, and a variety of less notable infectious disease outbreaks in wildlife were the "front wave" associated with a "rising tide" that was soon to become a "flood tide" of infectious diseases in wildlife.

Further increases in infectious disease epizootics during the 1970s included several events of great wildlife management importance. Catastrophic recurring avian cholera epizootics in Nebraska's Rainwater Basin established that geographic area as a new enzootic foci of this disease (fig. 10) and major epizootics elsewhere further established avian cholera as a disease of concern for wildlife conservation (Friend, 1999a). The Lake Andes National Wildlife Refuge in South Dakota took "center stage" in 1973 with the first major North American occurrence of duck plague in wild waterfowl. An estimated 40,000 of the 100,000 mallard ducks and small numbers of other waterfowl wintering on the refuge and the nearby Missouri River died during that epizootic (fig. 11). Just as in the early 1900s, mass mortalities from "western duck sickness" and the EHD "crisis" involving deer during the 1950s resulted in the creation of wildlife agency supported disease programs, as did duck plague. In January 1975, what is now known as the National Wildlife Health Center (NWHC) was initiated as an FWS program in response to the Lake Andes epizootic.

The 1970s also saw a chronic wasting disease (CWD; appendix 1) "marker" laid down in 1978 as the first pathological diagnosis of that disease. The clinical syndrome for CWD had been described in 1967 for captive mule deer at wildlife research facilities in Colorado, but the cause was unknown. The 1978 diagnosis, also in captive deer, established the disease as a **spongiform encephalopathy** (Williams and others, 2001). The "flood tide" of EIDs in wildlife was then joined at the start of the 1980s by a "rising tide" of EIDs involving humans and other species. Human immunodeficiency virus infection/ acquired immunodeficiency syndrome (HIV/AIDS; appendix 1) became a focal point for human disease and a bellwether "marker" for the tidal mixing of infectious diseases that would soon inundate humanity and other species following host switching by pathogens from their **reservoir** hosts to cause disease in a variety of new hosts.

By the 1980s, the increasing global surge of EIDs was reaching tsunami status that continues today (fig. 12). As previously noted, wildlife began to be affected by this advancing

Figure 10. The burning of waterfowl victims of avian cholera in Nebraska. (Photo by James Hurt, Nebraska Game and Parks Commission)

Figure 11. More than 40,000 mallards died during the 1973 outbreak of duck plague on the Lake Andes National Wildlife Refuge in South Dakota. (Photo by Milton Friend)

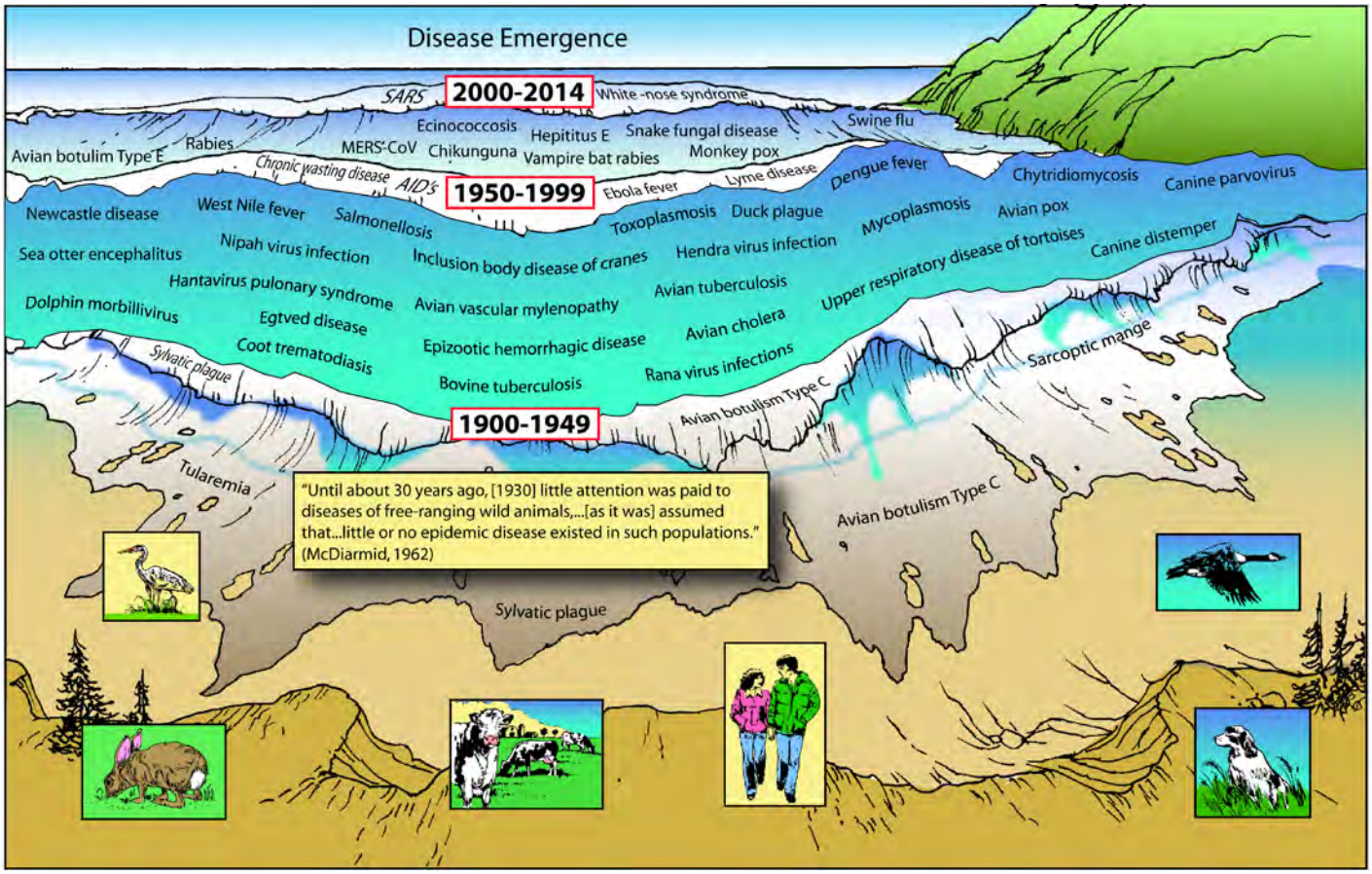


Figure 12. The period since the last half of the 20th century and continuing today has been marked by unprecedented global increases in infectious disease emergence and the resurgence of diseases once thought to have been adequately addressed. (Illustration by John Evans)

wave about a decade before humans. Even a cursory evaluation indicates that more noteworthy disease events affecting free-ranging wildlife occurred during the latter one-third of the 20th century than had been reported for all previous time within the modern era. Collectively, this situation is characterized by the worldwide scope and broad spectrum of ecosystems affected, the broad spectrum of types of pathogens involved, the relatively short frequency between major disease events, the rapidity of geographic spread, and the great magnitude of losses. Clearly, these are quite different circumstances than existed during the first 50 years of the 20th century.

Wildlife as Victims and Villains

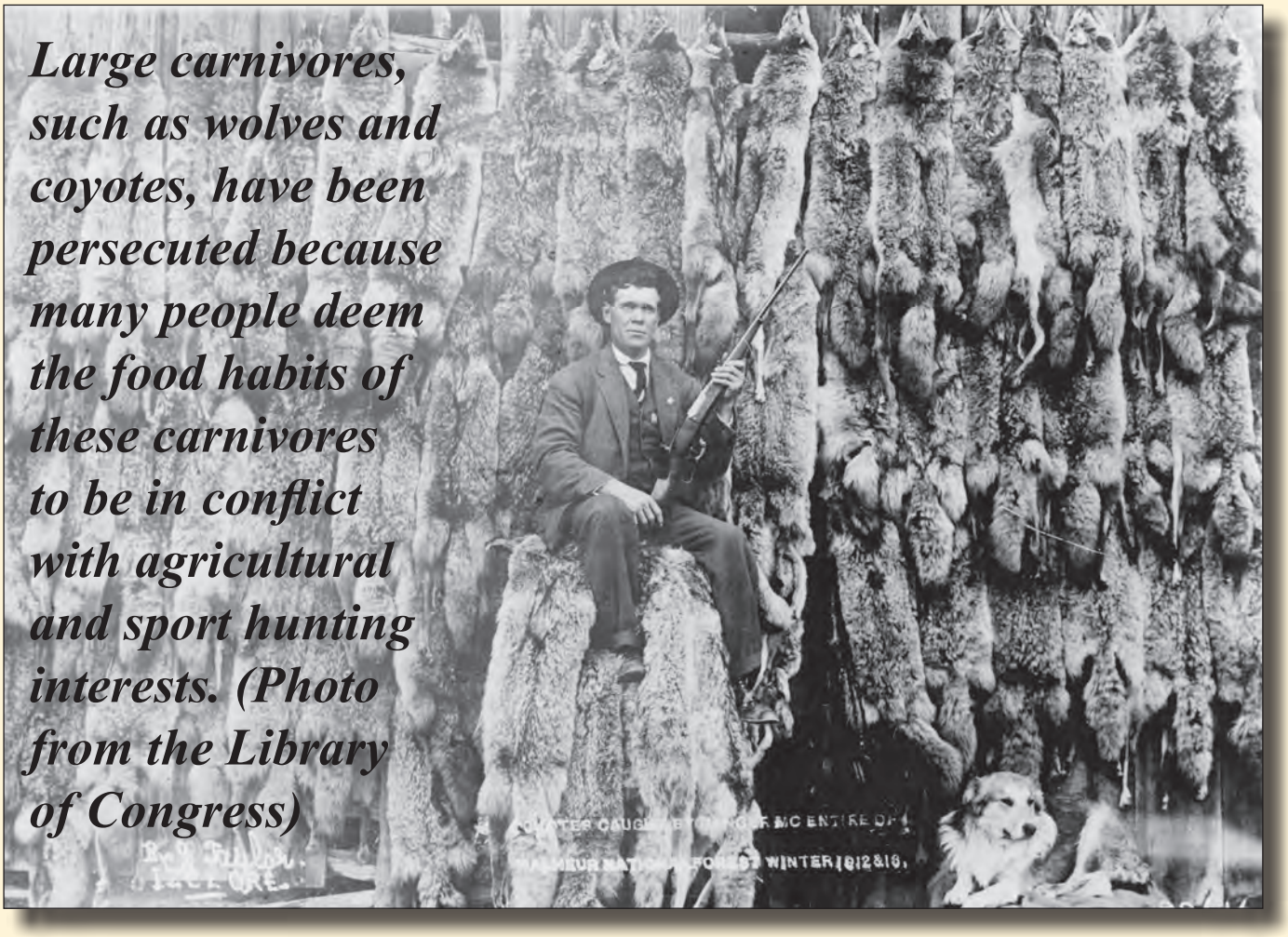
Wildlife are often in double jeopardy from disease due to the high number of infectious diseases of wildlife origin that afflict humans and (or) domestic animals. Approximately two-thirds of EIDs are of animal origin and nearly three-fourths of those have wildlife roots. Thus, irrespective of direct losses from disease, wildlife also are subjected to persecution as reservoirs and vectors of diseases affecting humans. Habitat alterations and other actions that do not involve the direct killing of wildlife, but that may have more long-term detrimental effects for the conservation of species, may also be responses to concerns about wildlife as a source of disease for other species. Thus, it is not productive for the conservation community to be nonresponsive to wildlife disease.

The dead red fox (left) was a victim of sarcoptic mange, a parasitic disease. Conversely, the live red fox that appears aggressive (right) was infected with rabies, and it would be seen by many people as a villain that threatens the health of humans and other animals. Thus, the effect of disease on wildlife is often that of "double jeopardy. (Photos by Milton Friend)

Wildlife Persecution

Late in the 20th century, I was directed to provide testimony at a "public trial" being held in a large metropolitan city on the East Coast. The "accused" in this trial was the Canada goose. The packed "court room" (a city government meeting hall) was indicative of the high passion associated with the purpose of the trial—to determine whether the resident goose flock should be executed. As I listened to the raging arguments for and against such action, I could not stop myself from wondering how it was that a species I considered during my New England youth to represent the "call of the wild" was now being cast by about half of the attendees to be "the great American pest bird." During my youth, I would be summoned by the calls of wild geese to view the migratory flocks passing over my neighborhood each fall and spring and to wonder about their journey and life in the wilderness. Returning to the situation at hand, it was evident that "my Canada geese" were not on trial, but the defendant was instead a "renegade" segment of the species that, like much of humanity, had chosen to sever its ties with the natural world and move to the city.

Separation of these different segments of Canada goose society helped me to hear better and evaluate the concerns and arguments being voiced. A basic perspective of those seeking the "death penalty" was that these birds were vermin and that, like rats, they posed disease hazards for humans. Thus, the human residents would be best served by ridding the area of Canada geese. My testimony was limited to addressing the specific disease concerns raised, all of which I challenged based on specific scientific findings of that time. Additional testimony was provided by others on various aspects of the issue. The "verdict" reached several days later by those administering the "trial" spared the geese to "poop" on lawns another day and continue with their "hooligan" ways of intimidating pets that approached their young, feeding on ornamental plantings, and in other ways being general "pests" (fig. *A*).



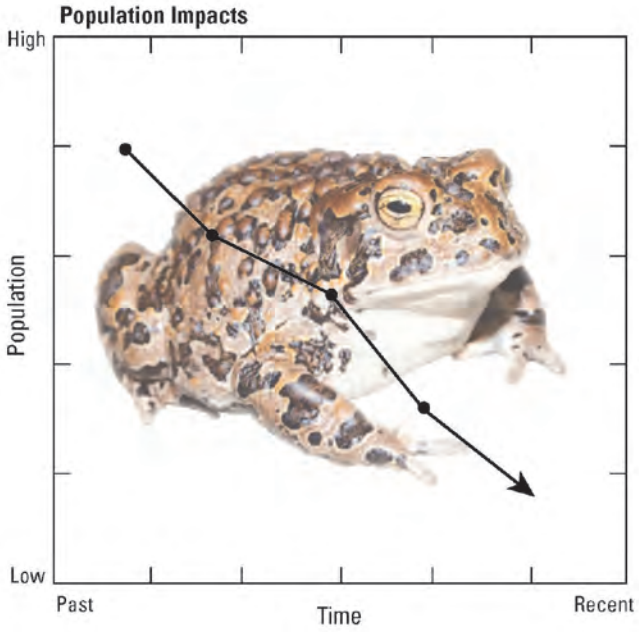
Large carnivores, such as wolves and coyotes, have been persecuted because many people deem the food habits of these carnivores to be in conflict with agricultural and sport hunting interests. (Photo from the Library of Congress)

Wildlife Persecution

If I were testifying today, my testimony would highlight disease transmission between Canada geese and humans that had not yet been documented at the time of my earlier testimony (fig. *B*). Although most humans are not known to be at much risk from these pathogens, emerging infectious diseases have made their way into some urban goose populations and need to be considered.

The destruction of wildlife to protect humans from zoonotic diseases is far less common in the United States today than in the past. Earlier in my career, the common response to rabies outbreaks was to implement lethal removal of wildlife rabies vectors (for example, foxes, skunks, raccoons) in the disease area. That practice is not commonly utilized today because of the development of effective **remote-vaccination programs** for foxes and raccoons. However, the vaccination of free-ranging wildlife is not currently applicable for most zoonotic diseases. Thus, deer are killed by government-hired sharpshooters in an attempt to reduce the risk of Lyme disease (appendix 1) in some urban areas. In addition, in other situations, wildlife may be removed to protect domestic animal production from diseases shared with wildlife.

A, Canada geese "playing through" as golfers approach to tee off. The long-standing conflicts between golfers and geese have now expanded to *B*, human health concerns, such as those highlighted in the journal articles shown. (Photo by Milton Friend)



The declining population levels of many wildlife species are due to increasing losses from disease during recent decades. (Photo by Paul Maier)

Population Impacts

Numerous factors, including disease, can affect species population levels. Conversely, species population levels can affect the ability of infectious disease to become established and spread. For example, a hypothetical disease X might cause an epizootic that kills a large percentage of some wildlife species within a geographic area, or population, or both. However, disease X may then fail to become established because the number of susceptible hosts available for additional infection falls below the threshold number needed to sustain the pathogen. That number is typically calculated as the "R_o" value, or basic reproductive rate of the disease. Specifically, it is "the average number of secondary infections attributable to a single infectious case introduced into a fully susceptible population" (Fine and others, 1982; see also Anderson and May, 1982; Wobeser, 1994). The environmental persistence of the pathogen and other factors governing its existence within the affected geographic area and populations also contribute to the dynamics of the disease.

Thus, from a wildlife conservation perspective, disease needs to be addressed within the context of wildlife management efforts to sustain desirable population levels and species biodiversity. In that context, factors to be considered include the indirect and direct immediate impacts from disease occurrence, the ability of the affected population to recover to predisease levels, and the general importance of the species involved relative to biological, social, and economic considerations.

Direct losses from disease that may foil the success of wildlife management and conservation range from highly visible, cataclysmic mortality events to small-scale mortality events involving a substantial percentage of the population involved. In addition, somewhat hidden diseases of attrition can inflict large-scale tolls that exceed the annual losses from high visibility diseases. Avian botulism and lead poisoning illustrate this point. As recently as the summer of 1997, an estimated 1 million waterfowl and other waterbirds died from avian botulism during outbreaks on wetlands in Saskatchewan. An additional 514,000 died on the marshes of the Great Lake area in Utah during that same summer-early fall period.

In contrast to the highly visible outbreak disease such as of avian botulism, lead poisoning of waterfowl is an example of the somewhat hidden diseases of attrition for which epizootic mortality events are the exception rather than the rule. Nevertheless, the annual death toll from these types of diseases may equal or exceed that of highly visible diseases. Some popular media reports and articles have referred to lead as "the invisible disease" and as "the silent killer." During the highly charged conflict period of the 1980s, opponents of efforts to address lead poisoning repeatedly rejected conservative science-based estimates of an annual loss of 1.6 to 2.4 million waterfowl. Instead, they continually showed their lack of understanding of this disease by voicing, "Show me the bodies."

West Nile virus (WNV; appendix 1) entered the New York City area in 1999 and aided by bird migration spread across the United States, north to Canada, and south into areas of Latin America by 2004. Tens of thousands of birds ranging from **hummingbirds** to wild turkeys died from WNV during the year 2000 alone (McLean, 2002). The broad host range for WNV includes 294 species of birds in 57 families and 24 orders along with 25 species of mammals from bats to reindeer and horses (McLean and Ubico, 2007). Other than humans and horses, the first mammalian species found infected in the United States were the striped skunk, eastern gray squirrel, eastern chipmunk, big brown **bat**, and little brown bat (all during the year 2000). **Owls**, **hawks**, and members of the **crow family** are especially vulnerable to this disease.

Carcass visibility, or lack thereof, should not be confused with the magnitude of losses occurring. Diseases of chronic attrition, such as lead poisoning, seldom result in numerous carcasses seen in the field, because predators and scavengers are generally able to keep pace with removing many of the sick before they die as well as consuming carcasses. In contrast, diseases that spread and kill rapidly, such as avian cholera, overwhelm any ability of predators and scavengers to consume the hundreds of affected birds that often die in a single day.

Diseases associated with environmental contaminants such as pesticides, lead poisoning, petroleum toxicosis (appendix 1), and other products with human technology can often be resolved through regulation once there are sufficient scientific findings to support cause-and-effect relations. For example, the first science-based evaluation of the effectiveness of the 1991 United States ban on the use of lead shot for waterfowl hunting (followed by a Canadian phase-in of nontoxic shot use during 1990–99) indicates a saving of 1.4 million waterfowl during the 1997 hunting season (Anderson and others, 2000). Findings from Spain also illustrate the initial effectiveness of nonleaded ammunition use in reducing waterfowl exposure to lead. Those findings indicate hunter killed ducks with only embedded lead shot in their tissues declined from 26.9 percent to less than 2 percent during the three hunting seasons following an enforced ban on lead shot use in the Ebro delta. During that same period, lead shot ingestion by mallard ducks decreased from a preban level of 30.2 percent to 15.5 percent (Mateo and others, 2014).

Avian reproductive failure associated with thin eggshells and other exposure impacts from synthetic pesticides and other chemical contaminants following their broad-scale post-WWII use is another example of diseases of attrition taking a heavy wildlife toll. The California brown pelican, peregrine falcon, and bald eagle were all being critically impacted to the extent that each became listed as a federally threatened and endangered species. The 1971 United States ban on the use of DDT was followed by a general global ban that also prohibited the use of DDT and a number of other harmful pesticides and other environmental chemical contaminates. Those actions facilitated species recoveries and removed from the endangered species list.

The direct impacts of disease on wildlife are also confounded by indirect factors. Depending on the disease involved, outdoor recreation activities have been curtailed by the **zoonotic potential** of specific pathogens. For example, plague and tularemia epizootics involving wild rodents on national park, national wildlife refuge, and other public lands have resulted in temporary closures of areas to protect human health. In other situations, warnings about specific zoonotic diseases have been posted rather than implementing area closures. Regardless of which approach is taken, both have the potential to stimulate negative human perceptions about wildlife.

The golden toad, from Costa Rica, was officially declared extinct in the wild in 2008 and is but one of a number of amphibian species whose populations are being decimated by the fungal disease chytridiomycosis (appendix 1). (Photo from the U.S. Fish and Wildlife Service)

Disease and Extinctions

The ultimate negative disease impact from a wildlife conservation perspective is the extinction of species. Beyond scientific evidence and evaluations that disease has been the cause for many early species extinctions before humans appeared on earth (Brothwell and Sanderson, 1967) and the hypothesis that disease was a factor in some megafauna extinctions during ancient times (MacPhee and Marx, 1997; Poiner and Poiner, 2008), there are a substantial number of species and subpopulations for which disease has already caused extinction in the modern era (primarily amphibians) or is currently a major threat for their continued existence (table 3). The extinction endpoint may result from chronic and (or) epizootic losses from disease suppression of population numbers below the minimum threshold level needed for species recovery; disease may constrain population recovery to the extent that the rate of recovery is so slow that other factors (including fiscal and other resource costs) result in the eventual extinctions of the species from the wild; and (or) disease may serve as a selective mortality factor directly leading to the species extinction.

Despite the conceptual potential for disease to cause species extinctions, hard evidence for such outcomes is difficult to obtain and the paucity of such evidence fuels the continuing debate associated with evaluations based on modeling outcomes (de Castro and Bolker, 2005). From a wildlife conservation perspective, extinction is a biodiversity issue beyond that of being a pragmatic conservation issue for sustaining populations at levels consistent with the general interests of society (nonconsumptive and consumptive uses). Nevertheless, greater acceptance of disease as an extinction factor may also support greater efforts to address disease as a means for mitigating against population declines.

Table 3. Examples of pending species extinctions.

Species	Pathogen or disease	Geographic area
Amphibians (multiple)	Chytrid fungus	Worldwide.
California condor	Lead poisoning	Western United States.
Bighorn sheep	Pneumonia complex	Western United States.
Harbor seal	Phocine distemper	United Kingdom.
Tasmanian devil	Viral cancer	Australia.
European red squirrel	Pox virus	United Kingdom.
Iberian lynx	Bovine tuberculosis	Spain.
Black-footed ferret	Plague	Western United States.
Desert tortoise	Respiratory disease	Western United States.
Ethiopian wolf	Rabies	The Web Valley, Bale Mountains National Park, Ethiopia.
Little brown bat	White nose syndrome	Eastern and midwestern United States.
Koala	Koala retrovirus and chlamydiosis	United States; Australia.

A vivid current example of pending extinction is the Tasmanian devil, the largest **extant marsupial** carnivore. In 1996 infection by a transmissible tumor was first reported for this species. The resulting disease was named devil facial tumor disease (DFTD; appendix 1), and within a decade it had spread across most of the species range, killing up to 90 percent or more of older age animals. DFTD transmission appears to be dependent on frequency (number of contacts) rather than dependent on density (number of additional susceptible hosts). In addition, there is no threshold density for disease maintenance (that is, R_o does not apply). Given the rate of geographic spread, the rate of population decline, and other aspects of DFTD, the near-term extinction of Tasmanian devils from the wild should be considered likely (McCallum and others, 2007).

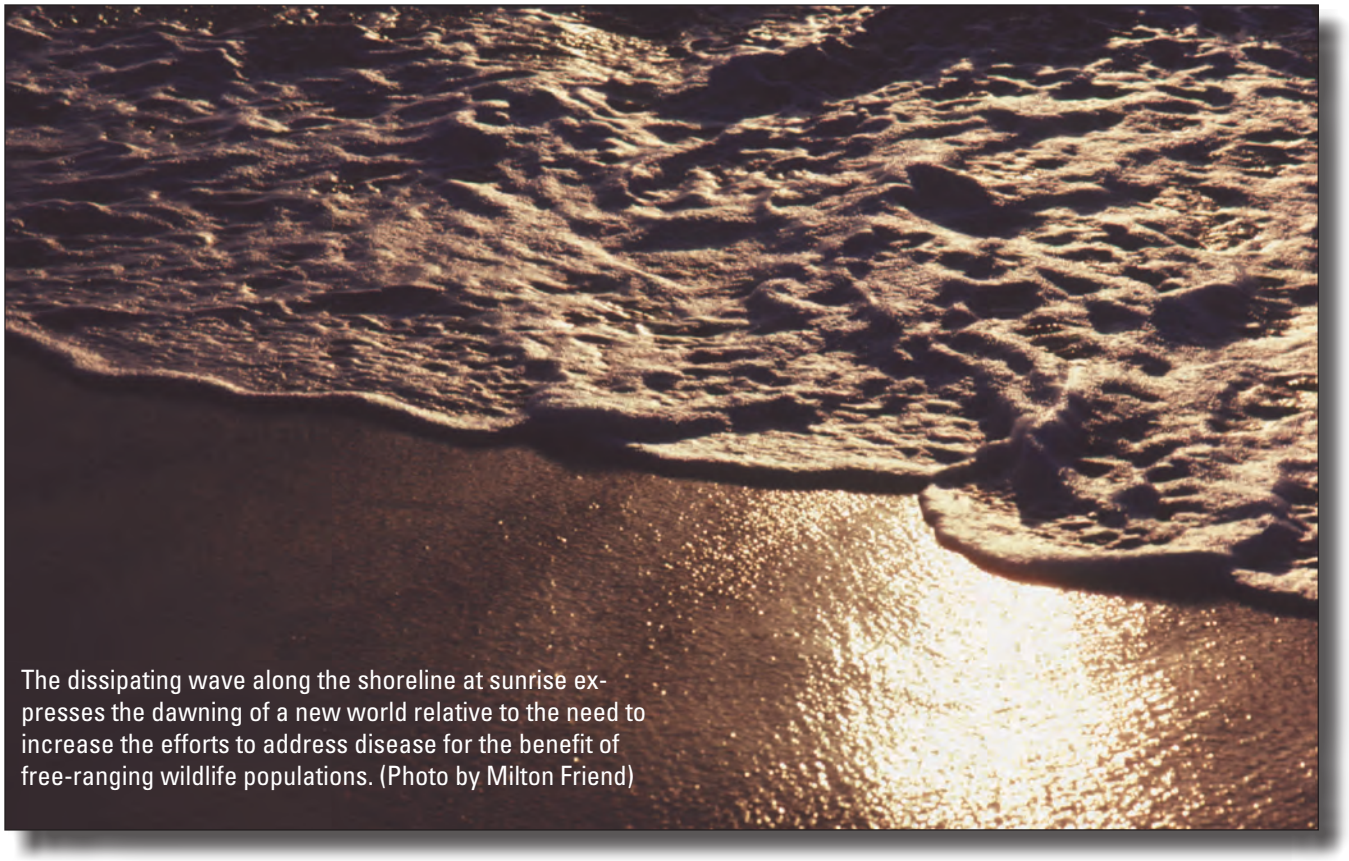
Noninfectious disease agents can also drive species to extinction. As previously noted, the California condor has been temporarily rescued from the brink of extinction by removing the few remaining condors from the wild during the 1980s to serve as a captive breeding population. However, the success of reintroduction continues to be jeopardized by lead poisoning, the reason California condors were originally removed from the wild.

For context, consider that,

> "Although host-pathogen interactions have been a subject of interest in conservation biology for some time, the possibility that disease might actually drive extinctions in certain contexts has rarely been considered. … Nevertheless, we believe that understanding the role of disease in provoking endangerment and extinction is critically important to the education of conservation professionals, if only because the contribution of disease to declines and outright extinction has likely been underestimated. What we do not understand, or ignore, may be what hurts us most." (MacPhee and Greenwood, 2013)

A recent evaluation of global extinctions since 1900 based on the International Union for Conservation of Nature (ICUN) Red List resulted in 30 animal extinctions and in 209 animals being listed as critically endangered, and disease is but one of the factors leading to that endpoint. The authors of that evaluation note that much of the supporting information is anecdotal, and one must seek more rigorous standards for determining causes of species endangerment and extinctions due to disease (Smith and others, 2006). While there is scientific merit in their evaluation, the question of disease and species extinctions is somewhat of a chicken and egg issue. That is, did the species go extinct because losses from disease reduced its population to levels that facilitated extinction by other forces (collectively or individually)? Alternatively, did other forces reduce the population to levels where disease was the final factor (that is, the proverbial "straw that broke the camels' back")? What matters most is that disease should be considered a major threat for species with low population numbers. Increased disease surveillance and monitoring along with timely intervention to suppress disease risks should be emphasized for these species and populations (fig. 13).

Figure 13. Disease has become a formidable current challenge for the preservation of global biodiversity and is likely to remain so for the foreseeable future. Corrective actions are needed now, for "endangered means there is still time." Conversely, "extinction is forever." (Photo sources: desert tortoise by Ken Nussear, U.S. Geological Survey; black-footed ferret by Ryan Hagerty, U.S. Fish and Wildlife Service; salt marsh harvest mouse by M. Bias, U.S. Department of the Interior; greater sage grouse by Stephen Ting, U.S. Fish and Wildlife Service; European red squirrel by Peter Trimming; Iberian lynx by Program Ex-Situ Conservation; Yosemite toad by Paul Maier; Hawaiian crow by U.S. Fish and Wildlife Service; black-faced honeycreeper by Paul E. Baker, U.S. Fish and Wildlife Service; Wyoming toad by Sara Armstrong, U.S. Fish and Wildlife Service)



The dissipating wave along the shoreline at sunrise expresses the dawning of a new world relative to the need to increase the efforts to address disease for the benefit of free-ranging wildlife populations. (Photo by Milton Friend)

A New World Has Dawned

Clearly, the new dawn of our ever-changing world has removed disease from the edges and background shadows as a wildlife conservation issue, and wildlife disease is now under the full illumination of center stage. European investigators have credited the mass media with stimulating much of the public's newfound interest in this subject area (Gortazar and others, 2007). Wildlife management must respond accordingly to what is indeed a "new world" and move beyond past inaction if we are to be successful in sustaining free-ranging wildlife resources for the next generation. Yet one of humanity's greatest strengths is also one of the largest hurdles to overcome in moving forward. That hurdle is defined by a single word—TRADITION!

Early judgments and perspectives about the role of wildlife disease among those concerned with the conservation of wildlife resources were primarily rooted in the philosophy and teachings of the noted naturalists and ecologists of that time. For example, at the start of the 1930s, famed ecologist, Charles Elton (fig. 14) noted that, although disease is widespread in wildlife, animals are normally much healthier than humans are. Furthermore, predation eliminates the weak from the population (thereby, minimizing disease spread). The context of Elton's considerations of disease was that:

> "disease is in fact, a perfectly natural phenomenon, and it forms one of the commonest periodic checks upon the numbers of wild animals…. Such epidemic diseases are normally associated with overcrowding in the population.… In consequence of this, there is usually a rather well-marked fluctuation in the numbers of the population, great density being followed by great scarcity, and this by a period of gradual increase up to another maximum, which is then followed by another epidemic" (Elton, 1931).

Elton's comments have often been interpreted and used as argument against any need to address disease because many proposed that:

1. disease is a necessary means for nature to keep wildlife numbers in check (that is, "a necessary evil—something to be endured");

2. disease events are self-limiting within wildlife populations and are followed by population recovery (that is, "this too will pass" and will be without any permanent harm); and

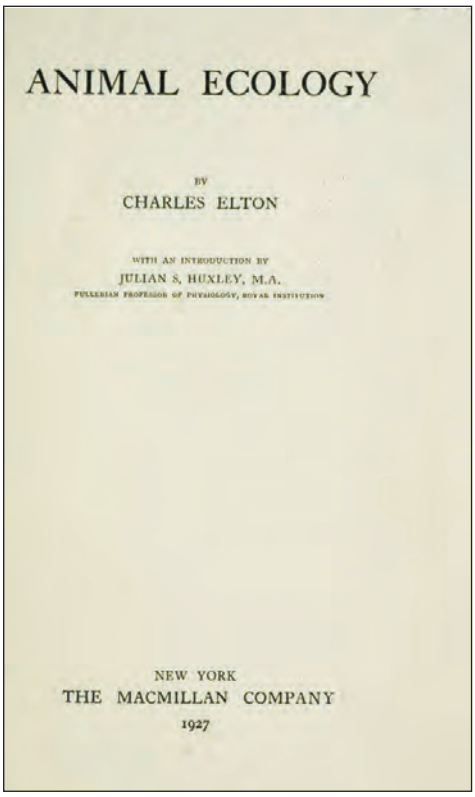
3. losses from disease are compensatory with predation rather than additive and as such need not be addressed; that is, in the absence of disease predation will account for wildlife loses that otherwise would have occurred from disease. In the presence of disease, predators selectively remove diseased wildlife thereby sparing healthier wildlife from predation. Thus, this equilibrium-like situation precludes the need to address disease.

Ecologist David Lack's viewpoints also greatly influenced the perspectives of wildlife conservationists and others. Lack (fig. 15) was focused on evaluating the natural regulation of animal numbers and championed food availability as the primary factor involved. He argued that disease was seldom more than temporarily important in controlling wildlife numbers and rarely could it be considered as a serious factor in the natural regulation of the numbers of most species in the wild. Further, he supported the concept "that a pathogen tends to evolve in such a way as to become less harmful to its host with time, since it has a better chance of survival if it does not destroy its habitat" (Lack, 1954).

> "Few ideas have been so ingrained in the literature of medicine and parasitology as the idea that parasites [all infectious microorganisms] should evolve toward benign coexistence with their hosts. Few ideas in science have been so widely accepted with so little evidence…In recent years both theoretical and empirical studies have led to a rejection of obligate evolution to benignness…yet it is still presented in well-respected journals and medical texts as the foundation upon which evolutionary arguments are built…" (Ewald, 1994)

The evaluations by Lack and Elton were focused on the "unencumbered natural world" but were being interpreted as guidance for considering disease within the pragmatic world of wildlife management, which needs to sustain harvested wildlife populations within reasonably "stable" levels. Thus, the issue is not whether disease is a primary or secondary cause of wildlife mortality, but whether it is a factor that needs to be addressed. For example, Lack cites an estimated loss of 1 million waterbirds from avian botulism at a lake in Oregon during 1925, a 1–3 million bird loss at the Great Salt Lake in Utah in 1929, and another loss of 250,000 birds at that location in 1932. He concludes that those and many other heavy losses at other locations suggest "…that disease, coupled with crowding and perhaps food shortage, might be important in controlling the numbers of North American ducks" (Lack, 1954). Clearly, the magnitude of each of the losses just noted substantially impacted the numbers of birds available for hunting. They also resulted in public outcry demanding actions to address the situation.

For many decades, the above perspectives and others collectively supported a nonintervention tradition by many relative to wildlife disease, despite the efforts of Aldo Leopold, others of his era, and those that followed. Thus, the evolution of wildlife disease from being considered primarily as tangential crisis events by the wildlife conservation community to becoming an integrated component of wildlife management has been a recent achievement. This transition will continue to evolve along with other transitions associated with the struggle to conserve global diversity in a world of unprecedented human population growth and competing demands between human, domestic animal, and wildlife use of Earth's land and waters.


ANIMAL ECOLOGY

BY
CHARLES ELTON

WITH AN INTRODUCTION BY
JULIAN S. HUXLEY, M.A.
FULLERIAN PROFESSOR OF PHYSIOLOGY, ROYAL INSTITUTION

NEW YORK
THE MACMILLAN COMPANY
1927

Figure 14. Charles Sutherland Elton (1900–91), famed English zoologist and animal ecologist, is noted for his fundamental roles in the establishment of modern population and community ecology. "Animal Ecology" was a classic publication in its time.

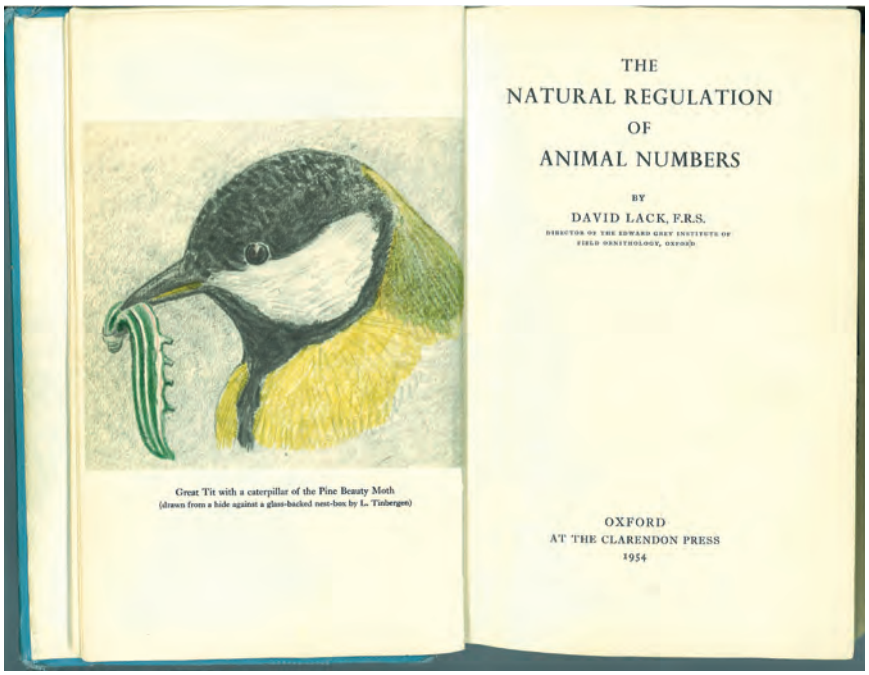
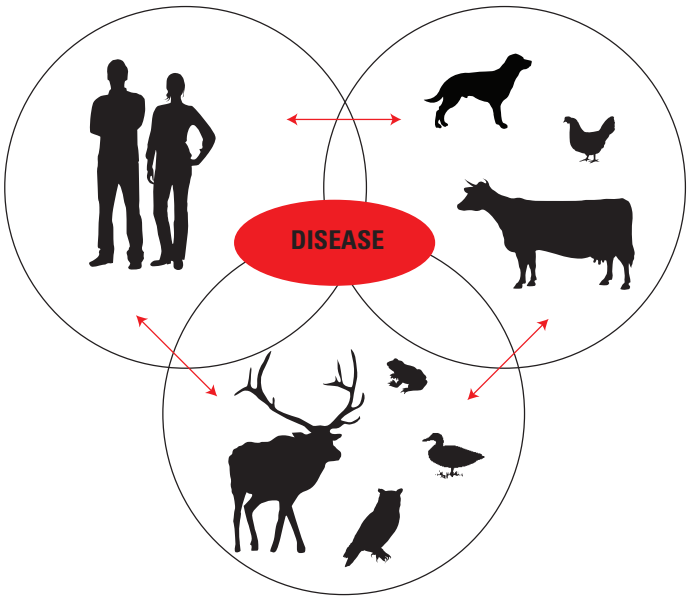


Figure 15. British evolutionary biologist David Lambert Lack (1900–73) authored "The Natural Regulation of Animal Numbers," one among many of his contributions to the advancement of ornithology, ecology, and ethology.

The "Rebirth" of Zoonoses



"... emerging zoonotic diseases are among the most important public health threats facing society."

(Mahy and Brown, 2000)

The rebirth of zoonoses has become one of the most pressing issues facing society in our "New World." The movement of these infectious diseases can be from humans to domestic animals or wildlife and vice versa.

What are zoonoses? The common dictionary, and often repeated, definition in scientific journals and media coverage of zoonotic disease conveys the limited concept of infectious disease transmissible from animals to humans. However, that perspective is inadequate. Zoonotic disease is multidimensional and ecologically complex, as are many of the pathogens involved. Like those pathogens, the definition of zoonosis has also followed an evolutionary path (table 4) (Hubálek, 2003). Here, it is sufficient to recognize that zoonoses are infectious diseases transmissible *between vertebrate* animals and humans *and vice versa.* In addition, the animal component has an essential role in maintaining the pathogen in nature for diseases transmitted to humans; for example, foxes and rabies. Humans serve that same role for diseases being transmitted to lower vertebrates; for example, measles (appendix 1) and **great apes**. These revelations have direct ramifications for wildlife conservation.

The general importance of zoonoses for humanity has "waxed and waned" over time in concert with changing conditions including changes in the number of human cases and (or) exposures associated with enzootic areas, such as chronic disease presence and activity levels, for specific zoonoses. The occurrence of major epizootics or epidemics involving the expansion of established geographic range for specific diseases and (or) the appearance of "new" zoonoses within a geographic area is also of great concern. Rabies is a well-established zoonosis and, except for anthrax

Table 4. The redefinition and expansion of zoonoses.

[Adapted from Stedman's Medical Dictionary (2006)]

Term	Definition
Zoonosis	An infection or an infestation shared in nature by humans and other animals.
Amphixenoses	A zoonosis maintained in nature by humans and lower animals; for example, certain streptococcoses and staphylococcoses.
Anthropozoonosis	A zoonosis maintained in nature by animals and transmissible to humans; for example, rabies, brucellosis.
Cyclozoonosis	A zoonosis that requires more than one vertebrate host (but no invertebrate) for completion of the life cycle; for example, various taenioid cestodes such as *Taenia saginata* and *T. solium* in which humans are an obligatory host; hydatid disease, a cyclozoonosis in which humans are not an obligatory host.
Direct zoonosis	A zoonosis transmitted between humans and other animals from an infected to a susceptible host by contact, by airborne droplets or droplet nuclei, or by some vehicle of transmission; the agent requires a single vertebrate host for completion of its life cycle and does not develop or show significant change during transmission; may include anthropozoonoses (rabies), zooanthroponoses (amebiasis), and amphixenoses (certain staphylococcoses).
Metazoonosis	A zoonosis that requires a vertebrate and an invertebrate host for completion of its life cycle; for example, the arbovirus infections of humans and other vertebrates.
Saprozoonosis	A zoonosis, the agent of which requires both a vertebrate host and a nonanimal (food, soil, plant) reservoir or developmental site for completion of its life cycle. Combination terms may be used, such as saprometazoonoses for fluke infections, when metacercariae encyst on plants, or saprocyclozoonoses for tick infestations, the agents of which complete part of their life cycles in soil.
Zooanthroponosis	A zoonosis normally maintained by humans but that can be transmitted to other vertebrates; for example, amebiasis to dogs, tuberculosis.

(appendix 1), perhaps the next earliest zoonosis to confront humans. The first recorded description of **canine rabies** (appendix 1) dates back to about 500 B.C. (Steele, 1975). Rabies is an important zoonosis in much of the world, because death is the outcome once clinical signs appear. Human deaths from rabies are rare in the United States, but the disease is diagnosed annually in wildlife and other animals where it continues to cause periodic epizootics. A recent major rabies epizootic that occurred among raccoons in the mid-Atlantic and northeastern United States (fig. 16) illustrates that even a zoonosis of antiquity can reassert its prominence in the modern era as a challenge for humans and wildlife alike.

The human, fiscal and other costs to society from zoonotic diseases are such that, "emerging zoonotic diseases [are] among the most important public health threats facing society" (Mahy and Brown, 2000).

"Because of their distribution and abundance, particularly in urban areas, raccoons are expected to play a major role in the spread of this epizootic (rabies) to new areas for years to come." (Centers for Disease Control and Prevention, 1994)

Figure 16. Rabies in raccoons was an infrequent occurrence in the Middle Atlantic and New England states prior to the late 1979 origin of a major rabies epizootic that followed the translocation of infected raccoons from an enzootic area within the southeastern United States. Today, raccoons are the major wildlife rabies reservoir throughout the eastern United States. (Map modified from Fishbein and Robinson, 1993; photo by Milton Friend)

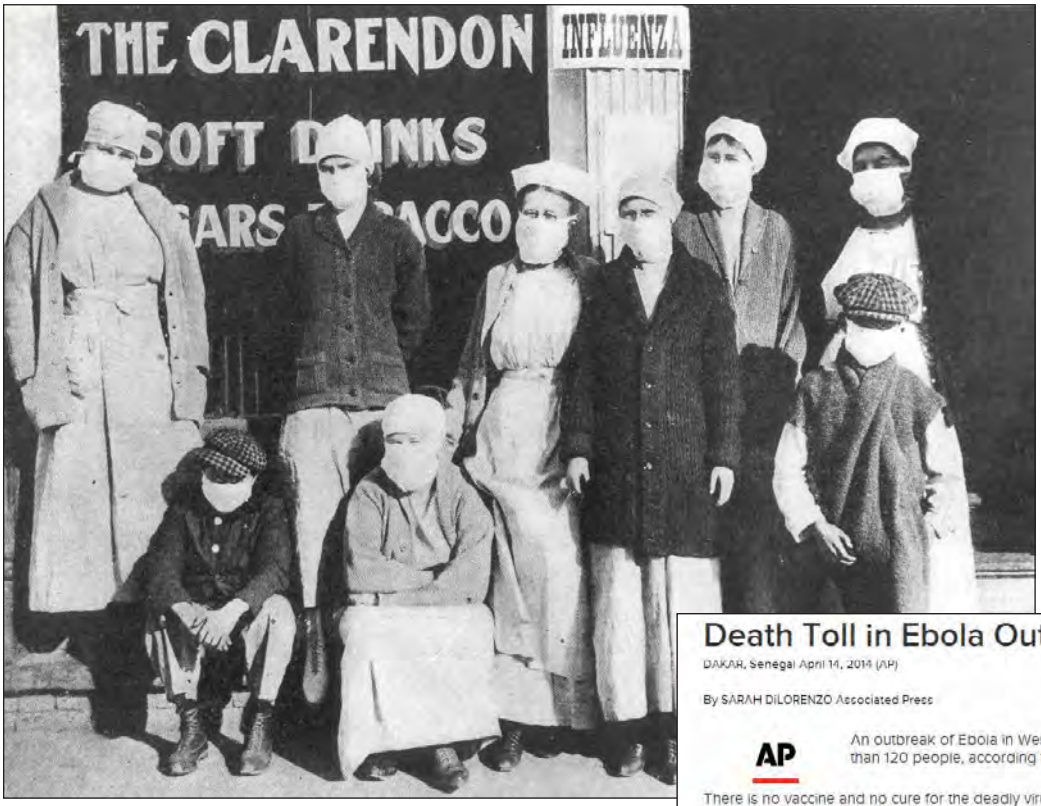
- Approximately 75 percent of emerging zoonoses worldwide have wildlife origins (Kahn and others, 2012).
- Diseases caused by zoonotic pathogens are twice as likely as strictly human pathogens to be classified as emerging or reemerging (Taylor and others, 2001).
- Over two-thirds of pathogens classified as zoonoses infect multiple nonhuman vertebrate species. (Brisson and others, 2011).
- At least 61 percent of all human pathogens are zoonotic (Taylor and others, 2001).

Throughout history, zoonoses also have been the cause of great "plagues" that have challenged the very existence of humanity at local, regional, and global levels. Globally, an estimated 200–500 million people were sickened during the 1917–19 H_1N_1 influenza virus "Spanish flu" pandemic (appendix 1), more than 20 million of whom died (Kohn, 1995). The specter of that pandemic (fig. 17) contributed greatly to the unprecedented global response following the 1997 diagnoses of highly pathogenic H_5N_1 avian influenza virus in Asia and the subsequent spread of that virus throughout much of Asia and Europe.

The emergence of highly pathogenic H_5N_1 is just one of a number of recent emerging infectious diseases that have wildlife roots, including numerous diseases that have caused epizootics of great concern for society (table 5).

The World Health Organization (WHO) reported that in 2006, 39.5 million people were currently infected with HIV/AIDS worldwide and that for the next year alone (2007), 18 billion dollars would be needed to prevent future HIV transmission and provide care for those already infected (Pedersen and Davies, 2010). A myriad of other emerging zoonoses followed HIV/AIDS to the headlines of major newspapers as well as serving as subject matter for major media venues of all types. These diseases have also become a major focus for scientific investigations and the development of specialized programs and facilities to address them.

Some emerging zoonoses cause major economic impacts for agriculture because of their presence in food production species such as poultry (H_5N_1) and swine (Nipah virus; appendix 1 and table 5). For example, the highly pathogenic H_5N_1 avian influenza virus that appeared in Asia during 1997 and reached 51 countries by early 2010 caused billions of dollars in losses for the poultry industries of those countries (Pappaioanou, 2010). West Nile virus (WNV) also stands out because of its geographic spread across the United States and within North America following the 1999 New York City index cases involving human fatalities, fatal cases in horses, and the thousands and thousands of wild birds killed.



Death Toll in Ebola Outbreak Rises to 121

DAKAR, Senegal April 14, 2014 (AP)

By SARAH DiLORENZO Associated Press

AP

An outbreak of Ebola in West Africa has been linked to the deaths of more than 120 people, according to the latest World Health Organization count.

There is no vaccine and no cure for the deadly virus, and its appearance in West Africa, far from its usual sites in Central and East Africa, has caused some panic.

Health workers are trying to contain its spread, tracking down anyone with whom the sick have had contact. Mali announced Tuesday that samples from all its suspected cases had tested negative for the disease.

Malian Health Minister Ousmane Kone said that the country had sent out 10 samples for testing at labs in the United States and Senegal, and all were declared negative for Ebola. There are no other known suspected cases in the country.

As of Monday, the U.N. health agency said it had recorded a total of 200 suspected or confirmed cases of Ebola, the majority of which are in Guinea. That figure includes some of the Mali cases that the government now says are negative. The organization said the deaths of 121 people in Guinea and Liberia have been linked to the disease.

Officials have said the current outbreak could last months.

By MARIA CHENG, Oct. 14, 2014 11:54 AM EDT

LONDON (AP) — West Africa could face up to 10,000 new Ebola cases a week within two months, the World Health Organization warned Tuesday, adding that the death rate in the current outbreak has risen to 70 percent....

WHO raised its Ebola death toll tally Tuesday to 4,447 people, nearly all of them in West Africa, out of more than 8,900 believed to be infected. Aylward said calculating the death rate means tracking the outcomes of all possible patients — a complicated process since the numbers of cases are substantially underreported and much patient data is missing.

Figure 17. The "Spanish flu" pandemic of 1917–19 serves as a not too distant reminder of the potential for emerging infectious diseases to inflict catastrophic loss of human life and heightens concern about other emerging highly pathogenic influenza viruses, the current (2014) outbreak of Ebola fever in Africa, and a variety of other infectious diseases. (Spanish flu photo from the Centers for Disease Control and Prevention; newspaper headlines from the Associated Press)

Table 5. Examples of recently emerged (post-1990) high-profile zoonotic diseases.

[High-profile diseases are those previously unknown in geographic areas listed, and human mortality is associated with initial events]

Disease	Pathogen	Year of emergence	Location	Primary means of transmission from animals to humans	Animal species showing observable signs and (or) death		Comments
					Wildlife	Domestic	
Hantavirus pulmonary syndrome (HPS)	Sin Nombre virus	1993	United States	Fine-particle aerosols of rodent excreta	No clinical disease	No clinical disease	Deer mice are the primary reservoir.
Hendra	Hendra virus (Paramyxo-virus)	1994	Australia	Contact with infected horses	No clinical disease	Horse	The fruit bat (**flying fox**) is the reservoir host.
Avian influenza	Highly pathogenic H_5N_1 virus	1997	Asia, Europe	Aerosol and contact with infected birds	Waterfowl	Poultry	Low pathogenic strains from waterfowl mutated to highly pathogen strain in poultry.
Nipah	Nipah virus (Paramyxo-virus)	1998	Malaysia	Aerosol; close contact respiratory droplets	No clinical disease	Pigs	Bats are the natural reservoir; infection of pigs was the source for human cases.
Severe Acute Respiratory Syndrome (SARS)	Coronavirus (SARS-CoV)	2003	China and then to other areas globally	Aerosol; close contact respiratory droplets	No clinical disease	No clinical disease	Bats are the natural reservoir; **civet cats** are the intermediate host responsible for most human infections during the initial outbreak.
West Nile fever	West Nile virus	1999	United States	Mosquito bite	Wild birds	Horse	A large number of wild animals died. Corvids (**crows** and related birds) are highly susceptible to WNV. American robins are a major amplification host. The disease has broad range of host animals including birds, mammals, and American alligators.

As previously noted, threats from EIDs are unlikely to decrease, because the ever-changing relations between humans and the environment are a major factor driving disease emergence. Dr. Carlyle Guerra De Macedo, Director, Pan American Health Organization 1987, noted that because of the human-animal interface that is inherent for zoonoses, "These diseases thus illustrate, perhaps better than any similar problem, the close relationship between public health, the environment, and socioeconomic well-being." (Referring to developing countries; Guerra de Macedo, 1987).

The separation between the relevance of zoonoses to wildlife management and conservation and to public health issues has rigidly existed in the past but has been greatly eroded by the current wave of EIDs, many of which are zoonoses. Further, the great costs of zoonoses for society demand that these diseases be aggressively dealt with. For example, of the 868 zoonoses identified at the start of the 21st century, a review of 56 of them revealed approximately 2.5 billion cases of human illness and 2.7 million human deaths worldwide per year (MacMillan, 2012).

The prominence of wildlife as components of zoonotic diseases invites wildlife persecution by some segments of society. The 1997 outbreak of highly pathogenic H_5N_1 avian influenza in Asia serves as a recent example. The finding of wild birds infected with H_5N_1 resulted in fears that wild birds would cause large-scale spread of the virus, prompting policymakers in some countries to call for the elimination of wild birds. The initiation of wild bird culling that followed prompted the United Nations (U.N.) to warn against taking such measures (Pappaioanou, 2010). Because many wild birds are migratory, losses incurred across their total range affect their population status. Thus, wildlife conservation interests need to be fully engaged in addressing emerging zoonoses that have a wildlife component.

A key point is that wildlife management and the conservation of wildlife species cannot function in a vacuum. Without adequate public support for wildlife, other interests of society will be given priority. Clearly, the aggressive management of EIDs will continue to be demanded by society, especially for emerging zoonoses. Because wildlife are an important component of such diseases, wildlife conservation agencies and the conservation community at large have shared responsibility with other segments of society for addressing EIDs through the management of the wildlife species involved. For example, Ebola virus, the cause of a dreaded, usually fatal disease of humans in Africa also poses a major threat for nonhuman primates.

Awareness of the elevated risks for exposure to zoonoses by those involved with the handling of live wildlife or in the collection and (or) processing of wildlife carcasses also is important. Such individuals should have a basic appreciation of zoonoses so they can take reasonable precautions for minimizing their own potential risks for exposure. Failure to do so can have personal consequences, even for those with formal training in animal diseases.

Ebola hemorrhagic fever first emerged as an infectious disease in 1976 in Zaire (Democratic Republic of the Congo). The excessive mortality rate during that outbreak (88 percent of 318 reported human cases) instantly brought this disease to worldwide attention. The 2014 major epidemic of Ebola fever in humans was preceded by an August 2012 tally by the Special Pathogens Branch of the Centers for Disease Control (CDC) that listed 30 outbreaks (including the original) involving 1 of the 4 strains of the causative virus. Nonhuman primates are highly susceptible to this virus (especially gorillas and chimpanzees), and massive declines in ape numbers have been associated with Ebola virus outbreaks. In the Lossi Sanctuary in the Democratic Republic of the Congo, more than 90 percent (5,000) of the gorillas were reported victims of this disease (Ball, 2012).

Because most emerging zoonoses have wildlife roots, human exposure to these diseases are likely to first be encountered from wildlife. Such was the situation in New York State when a fungal disease associated with a hunter-killed white-tailed deer infected 4 of the 10 people who handled that deer. All four developed lesions on their hands 2–7 days after their contacts with the deer. Two of the affected people were involved with the shooting and field dressing of the deer, and the other two were conservation department employees who examined the animal at a deer check station and then transported it to the laboratory where it was evaluated. A fungal disease, streptothricosis (appendix 1), was determined to be the cause of infection, and the infected deer was the first wildlife case of streptothricosis documented in North America. This disease has rarely been reported in domesticated animals in the United States. Furthermore, this was the first time the pathogen involved, *Dermatophilus* (sp.) had ever been isolated

Zoonoses and You—Lessons Learned the Hard Way

Removal of the organs and viscera (field dressing) of harvested deer and other large wildlife species is commonly done without any personal protective clothing, such as rubber gloves and aprons. The resulting contacts have on occasion resulted in the transfer of disease agents from those wildlife to humans. (Photo by Milton Friend)

Plague is another zoonotic disease that can be transmitted to a human by inhaling infectious droplets from an infected animal. The first documented wild carnivore-associated case of primary pneumonic plague in a human was in 2007 in a wildlife biologist at Grand Canyon National Park, Arizona. Tragically, the outcome was the death of the biologist.

The biologist had a close association with the mountain lion that infected him. His employment activities included trapping and radio collaring mountain lions. The biologist had tracked the animal involved for 6 months before he found it dead. He then carried the carcass about 1 kilometer to his vehicle and transported it to his garage, where he removed the hide and conducted a necropsy.

Despite established agency protocols, the biologist apparently did not use protective clothing and other personal protection equipment while processing the carcass. Also, based on records found, the biologist was misled by his observations to believe that the animal had been attacked by another mountain lion and had died from the injuries associated from that attack. Those investigating this tragedy reached several important conclusions from the evidence available. Their published report should be read (Wong and others, 2009) and their commentary considered in the larger context of one's own wildlife contacts. Specifically, those investigators found:

1. exposure resulting in infection of the biologist was via inhalation of aerosols generated while handling the infected mountain lion;

2. the necropsy involved several procedures likely to generate aerosols and was most likely the primary cause for the biologist's exposure to the pathogen; and

3. the biologist did not seriously consider plague as a possible cause of the mountain lion's death, or for his own illness that followed. Nevertheless, his work experiences should have sensitized him to the potential for plague in the area where he was working.

from humans, "…thus adding yet another disease to the Zoonoses or the diseases of animals transmitted to man" (Dean, 1961; Dean and others, 1961). During the following deer hunting season, no additional infected deer were found among the 2,352 hunter-killed deer I and others examined at deer check stations within that region of the state.

More recently, the CDC confirmed infections with parapoxvirus (appendix 1) in two deer hunters in the eastern United States. One of the hunters was a wildlife biologist. Both hunters became infected when they nicked their fingers while field dressing apparently healthy deer they had shot and then transported to deer check stations. This case also appears to involve a novel deer-associated infection (Roess and others, 2010).

Fortunately, the pathogens involved in the deer-related zoonotic events did not pose life-threatening or serious disease threats, and the events were isolated. Nevertheless, those cases highlight that even uncommon wildlife-associated disease events can jeopardize the well-being of people who handle wildlife. Thus, basic precautions should always be taken.

Wildlife Zoonoses 101

The current era of emerging infectious diseases (EIDs), especially the zoonoses among them, has greatly increased the challenges we face from infectious disease. These challenges are significant, and despite the "quantum leaps" in advanced technologies for addressing disease issues, our individual actions weigh heavily in this issue. Human-animal relations are major factors driving the occurrence of EIDs and in many instances are also barriers to change. Thus, there is increasing need to adjust basic human-animal relations in ways that minimize the potential for humans to be victimized by the pathogens that abound in our environment. This should begin with personal actions that can be taken by all, such as those recommended for petting zoos and within the more detailed, "Compendium of Measures to Prevent Disease Associated with Animals in Public Settings, 2005" (Centers for Disease Control and Prevention, 2005). The analogy with petting zoos is a statement that even in controlled environments zoonotic disease risks are present and need to be addressed. *Escherichia coli* O157: H7, *Cryptosporidium* sp., *Salmonella* spp., orf virus, *Coxeilla burnetti* and *Giardia duodenalias* are examples of the pathogens documented from petting zoo encounters (appendix 1) (Weese and others, 2007), all of which might also be encountered in nature.

1-2-3 Approach

Perhaps a useful starting point is to consider excursions into nature (and even to petting zoos) as if one were visiting a foreign land. If you are not personally familiar with that geographic area, a basic question often raised is "what diseases, if any, should I be concerned about." Typically, the CDC will have posted information about recommended immunizations and other protective needs, such as, malaria pills, foods to avoid, etc., that travelers should consider. Traveler inquiries are generally motivated by perceptions of potential but unknown health hazards that may exist in the areas to be visited. Lack of motivation to do so is most likely associated with the "specter of the unknown" being replaced by a pragmatic "comfort level" relative to personal knowledge of general conditions within one's own country.

In contrast to human-based infectious disease situations, wildlife zoonoses are more closely represented by uncertainty and unknowns that might be encountered for disease risks. Therefore, it is useful to consider a 1-2-3 approach if you are not personally familiar with situations that might be encountered during visits to "nature."

1. What types of serious infectious diseases are present in wildlife within the area to be visited?

2. Which of those diseases are transmissible to humans?

3. How can I best protect myself from exposure to those diseases?

A basic consideration relative to question 3 is knowledge of the primary routes of pathogen transmission for any diseases of concern. Although the major focus should be on diseases (including parasites) of animals you are likely to have direct contact with while hunting, trapping, or field research, for example, it is also important to consider pathogens that may be deposited in surface waters by other species using the area, such as **voles** and tularemia, raccoons and leptospirosis, American beaver and giardia, and those transmitted by invertebrate vectors, such as ticks and Lyme disease. In making these types of disease risk evaluations, consider small rodents such as **mice** as well as the more charismatic wildlife. For example, during 2012 three deaths from nine human cases of infection by hantavirus occurred as an unprecedented tightly focused geographic cluster within Yosemite National Park. The causative virus is shed in mouse urine and feces and is typically transmitted to humans via fine-particle aerosolized dry fecal matter.

Fortunately, events such as the hantavirus outbreak in Yosemite are currently rare; however, they are increasing in number and diversity of pathogens involved. The human response should not be to avoid visits to nature or appropriate physical contacts with wildlife. Instead, minimal self-education serves the adage "forewarned is forearmed." Education provides for both components of that equation. "Common sense" provides the guidance needed to apply that knowledge in ways that sustain pleasurable visits to nature and with its "citizenry."

Preemptive activity
(Centers for Disease Control and Prevention, 2005)

- Provide visitors with educational materials about zoonotic disease risks.
- Train staff about zoonotic disease.
- Prevent animal-human contacts in food service areas.
- Provide areas in the petting zoo where animals are not allowed.
- Restrict food and beverages to areas free of animals.
- Provide handwashing facilities.
- Prohibit hand-to-mouth activities (that is, eating, drinking, smoking, and carrying toys and pacifiers) in areas where humans come into contact with animals.

Humans and animals interact or meet in many ways within urban environments; children especially may *A*, engage with domestic animals at petting zoos and animal fairs and *B*, with freeranging wild and feral animals in city parks and other places. (Photos by Milton Friend)

Zoonoses seem to present fewer hazards when one is knowingly involved with activities associated with highly pathogenic disease agents and the diseases they cause than during general situations. This may be because the first instance involves known circumstances while the second involves the abstract. Nevertheless, that difference should have no bearing, because we have a continuing need to take preventive steps for protecting ourselves from infectious diseases. Beyond the scope of specific infectious disease investigations lies the reality of the world we share with more than 1,400 species of infectious organisms known to be pathogenic to humans, including approximately 870 that are zoonotic (Taylor and others, 2001). Despite those potential threats, society goes about its business without being intimidated by the specter of infectious disease for it has no option. However, because too little preemptive thought and too few associated actions are taken for combatting those more than 1,400 species of infectious organisms, infectious disease is the leading cause of human mortality.

The adaptive advantage most infectious agents have over humans is an important factor in their endless struggle for survival and for our need to deal with the unexpected. The unexpected is in part reflected in the increasing array of pathogens crossing what previously were believed to be species barriers, which results in unanticipated appearances of known pathogens in hosts they had previously not infected. If all or much of the above seems to have little relevance for wildlife management and (or) conservation consider the "Next Big One."

The Next Big One

Leading experts involved in the investigation of EIDs are in general agreement that the "Next Big One" (highly virulent pandemic that sweeps across the world) "is not only possible but probable. …it will almost certainly be a zoonotic disease…that emerges from wildlife and will most likely be a virus" (Quammen, 2012).

Beyond the importance of zoonoses as threats for human health is that of human diseases being introduced into free-ranging wildlife populations. There is growing concern among the wildlife conservation community about ecotourists and others transferring human diseases into wildlife populations. Outbreaks of human tuberculosis among banded mongooses and suricates (appendix 1) in Botswana have been attributed to humans as the source for infection (Alexander and others, 2002; Randerson, 2002). An undiagnosed 1988 epizootic among the endangered mountain gorilla in Rwanda is thought to have been measles of human origin (Ferber, 2000). In addition, new intestinal parasites have been found in the feces of mountain gorillas since tourists began visiting their habitat in large numbers (Alexander and others, 2002; Randerson, 2002). Those findings and others have led to an increased focus on disease as a necessity for enhancing the conservation of various African wildlife species (Goldberg and others, 2007; Goldberg and others, 2012). This pathway for disease transfer is likely to have increased use by pathogens due to increasing global contacts between humans and wildlife, especially in urban areas. Thus, it is prudent for the wildlife conservation community to take preemptive actions to prevent this pathway from becoming a highway for disease introductions into free-ranging wildlife populations.

Zoonotic Pathogens as Weapons

Zoonotic pathogens are potential candidates for use as bioweapons (chap. 6 in Friend, 2006a). This is not a new concept, as biological warfare was attempted in various ways centuries before the germ theory for disease was proposed in 1530 (Geissler and Courtland Moon, 1999; Wheelis, 2002). For example, during the 1346 Siege of Caffa (or Kaffa, which is now Feodosija, Ukraine) the Mongol army catapulted plague-infected cadavers into that besieged city. It was reported that, "mountains of dead" were thrown into the city, infecting the inhabitants and causing many deaths from the **Black Death** (Wheelis, 1999; 2002). Because this incident happened well before the germ theory had even been proposed, failure to understand and take precautions against the cause of plague and its transmission also resulted in many deaths among the Mongol army.

The United States, Russia, and several other countries have had longstanding biological weapons programs. Development of these weapons was intensified during World War II and continued to varying degrees during the immediate postwar era. In 1972, the "Convention on the Prohibition of the Development, Production and Stockpiling of Bacteriological (Biological) and Toxin Weapons and on their Distribution" (BWC) was signed by more than 100 nations, was ratified, and went into effect in 1975. Here in the United States, Nobel Laureate (1958) Dr. Joshua Lederberg provided a powerful

"Detection of disease in lower animals may be essential to detecting a bioterrorism event because most of the bioterrorism threat agents are zoonotic disease agents" (Ashford and others, 2003)

1970 statement to the Conference of the U.N. Committee on Disarmament (Congressional Record, September 11, 1970, p. E–8123–8124) in which he addressed various threats such weapons pose for humanity. In doing so, Dr. Lederberg stated that:

> "We now begin to realize that the intentional release of an infectious particle, be it a virus or bacterium, from the confines of the laboratory or of medical practice must be formally condemned as an irresponsible threat against the whole human community."

One of the actions he proposed to provide as a basis for detecting nefarious activities would be constantly expanding participation of nations in international health (human) programs. Disease monitoring may be helpful in detecting biological weapons research at early stages when intervention can be most beneficial. Because of the transfer of zoonotic pathogens between animals and humans, it seems prudent and responsible for wildlife disease surveillance to be a component of such efforts.

Nobel Laureate (1960) Dr. Frank Burnet, when considering "New Disease and the Outlook for the Future" in the first edition of his book, "Biological Aspects of Infectious Disease" (1940), stated that:

> "If the worst happens and another world war develops, it is quite certain that attempts at artificial dissemination of disease will be made. …If a bacteriological weapon were developed, its attack would be invisible and unknown, and death [of humans] would be delayed for days."

In the second edition (1953) of that book, Burnett discussed the increasing potential for this threat becoming reality due to major technological advances taking place in microbiology and other fields, and provided the following sober commentary:

> "Somehow the use for human domination of nuclear reactions, artificial virulent and artificially disseminated microorganisms and control of thought and behavior methods, whether pharmacological or psychological, must be prevented. Unless this can be done the whole biological and social background to the human species is in danger of complete disorganization of a type so apart from any previous experience that nothing less than a new process of evolution will be needed before a healthy civilization again becomes possible" [emphasis added].

Approximately a decade later (1962), in the third edition of that publication, Burnet noted that:

> "…the use of biological warfare is an absolute novelty, a complete break in the tradition of war as an extension of personal combat. …But as long as the possibility of the irresponsible use of power persists, we cannot escape the conviction that the steady advance of experimental microbiology will progressively increase the likelihood that effective biological weapons of mass destruction, sabotage and assassination can be produced."
>
> "This is a situation that must be faced. While war is possible, the development of microbiological weapons will go on and if they are perfected they will be used as seems expedient when war occurs."

Wildlife can be incorporated as vehicles for transmission for many such agents and may serve as an early detection system following the release of such agents. The current era of EIDs is providing a new array of highly fatal pathogens that can cause devastating losses of human life and (or) food production animals. Furthermore, the nefarious uses of pathogens of more established vintage, such as anthrax (appendix 1), within urban environments have been perpetrated in Tokyo, and Washington, D.C.

Constant vigilance to facilitate early detection and reporting that leads to actions for minimizing impacts is a national interest, and protocols are in place for initial reporting (*www.selectagents.gov*). Areas that have high concentrations of wildlife and high numbers of visitors—such as refuges and parks (fig. 18), including those in urban areas—are "soft targets." Therefore, unusual animal mortality, especially that involving species not commonly found on the areas at that time of year (or ever), should be promptly evaluated by professional disease investigators.

Figure 18. Wildlife can serve as vehicles for the intentional dissemination of pathogens to other species such as livestock, poultry, and humans. Wildlife refuges, national parks, and other locations where animals and humans are in close proximity to one another are vulnerable "soft targets" that require extra vigilance for the early detection of disease events. (Photo by Jim Peaco)

EDITORIAL

Use caution, sense to avoid plague in city

A relatively rare outbreak of the Bubonic plague is sweeping through City Park in Denver. People should avoid contact with rodents in the area.

The Denver Post Editorial Board

POSTED: 05/11/2011 01:00:00 AM MDT

In Colorado, we're accustomed to hearing about the Bubonic plague. Despite its scary name and grim history of killing millions, the disease largely has been confined to the foothills or far-flung parts of the state, killing a few dozen rodents and pets every year.

But this year's outbreak is sweeping through Denver's City Park, which on any warm weekend day is host to hundreds of youth soccer players, people walking dogs and picnickers.

The carriers this time are squirrels, and City Park is chock full of them. The take- home message for anyone who visits City Park, or any of Denver's many parks and adjacent neighborhoods, is to stay away from squirrels, dead or alive. And keep your pets away from them, too.

A dozen infected animals have been found in City Park, which is home to a large number of squirrels that tend to approach people without hesitation, hoping for a handout. There's never justification for feeding wildlife, but the plague is an especially good reason for avoiding it.

Workers at the Denver Zoo, adjacent to the park, are carefully looking for signs of the disease in their animals. In April, they had found four dead squirrels and a rabbit on zoo grounds. All tested positive for the plague. "At this point, we're just continuing to monitor," said Ana Bowie, zoo

linkedin.com...

Bubonic plague: Four Corners health alert as man sickened, prairie dogs dying

BY NATE CARLISLE
THE SALT LAKE TRIBUNE
PUBLISHED OCTOBER 7, 2008 12:03 AM

This is an archived article that was published on sltrib.com in 2008, and information in the article may be outdated. It is provided only for personal research purposes and may not be reprinted

A northeast Arizona man has contracted bubonic plague and health agencies are urging Four Corners residents to help prevent the spread of the disease.

The Apache County, Ariz., man who contracted the disease began showing symptoms in late September, the Navajo Nation said, including a 103-degree fever, chills, diarrhea and groin tenderness.

The man was responding to treatment and doing well Monday afternoon, said Jenny Notah, a spokeswoman for Navajo Area Indian Health Service.

The Navajo Nation also said plague likely killed a number of prairie dogs east of Flagstaff, Ariz. There have been no recent reports of plague cases on the Utah side of the Four Corners.

Humans can contract plague by touching bodily fluids of infected animals or after being bitten by fleas that have contracted it from infected rodents, according to the U.S. Department of Health and Human Services.

In 2006, the disease was found in rodents in Mesa Verde National Park and

The increasing urbanization of society and the associated interfaces with long-standing wildlife habitat are facilitating zoonotic disease emergence such as the recent outbreaks of sylvatic plague highlighted in these newspaper articles. (From the Denver Post, May 11 ,2011, and the Salt Lake Tribune, October 7, 2008)

Urban Environments and Disease Emergence

An additional consideration relative to the question "Why bother?" (to address wildlife disease) is associated with the dramatic shift in human demography and associated impacts of human population growth on wildlife habitat. Wild and rural lands that have been the mainstay of habitat for most terrestrial wildlife are increasingly being converted to urban and suburban environments, wetlands continue to be drained and, mountaintops and rain forests destroyed by mining and logging activities. In many instances the wildlife associated with those areas are forced to relocate to more marginal habitat that is less productive in sustaining desired population levels. From a disease perspective, the relevancy of this habitat loss is reduced resiliency of those wildlife populations to overcome major losses from disease. Also, just as the human population has primarily become urbanized, numerous wildlife species have also moved to the cities and suburbs where they have increased potential to interact with other free-ranging wildlife species as well as humans and a wide variety of domestic, feral, and exotic animals (fig. 19). Thus, the collective urbanization of humans and wildlife provides conditions for the "perfect storm" in an unchartered sea of zoonotic and other infectious disease potential.

Figure 19. City parks and other aspects of urban and suburban environments are increasingly providing enhanced opportunities for the transfer of pathogens between various animal populations and between animals and humans. Thus, there is an increasing need to replace ad-hoc animal disease surveillance within these environments with structured programs oriented from a "One Health" perspective. (Photo by Milton Friend)

The density of human environments facilitates the maintenance and transmission of what are commonly referred to as "crowd diseases" and the rapid emergence and spread of newly acquired infectious pathogens. (Photo by Milton Friend)

The Human Factor

Urbanization has been referred to as the "Third Frontier" for American society following exploration and settlement as the "First Frontier" and agricultural expansion as the "Second Frontier." Human population levels in cities greatly exceed those in rural communities and are growing at an increasing rate, and it is estimated that by 2050 about three-fourths of the human population will live in cities and their suburbs (Cohen, 2003). These large population numbers are favorable for explosive epidemics and, in association with other aspects of modern society, for the maintenance of **pandemics** reminiscent of centuries past. It is thus important to consider that the dynamics of most urban environments involves the constant movement of goods, including various animal species, into and through its cities. In addition, the human interface with wildlife in urban environments almost exclusively involves nonconsumptive activities (except for fishing) such as bird and wildlife watching and feeding. The "2011 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation" (FWS, 2012) found that 52.8 million people fed wildlife around the home.

During the 1960s and 1970s, some professional wildlife biologists began to focus their attention on urban habitats to increase the consideration of such habitat for active wildlife management. Especially relevant today is a 1966 presentation by Professor Raymond Dasmann, a noted wildlife biologist of the mid-20th century, who in discussing "new conservation" stated that more wildlife biologists should "get out of the woods and into the cities…to make the cities and metropolitan regions, the places where people live, into environments where each person's everyday life will be enriched to the maximum extent possible by contact with living things and natural beauty" (Adams, 1994). Dasmann's rather romantic perspec-

tive about human-wildlife relations has since been overtaken by social change. “In the space of a century, the American experience of nature has gone from direct utilitarianism to romantic attachment to electronic detachment” (Louv, 2005).

The Animal Factor

Urban fauna are highly diverse and include “human-sponsored” animals in zoological collections and scientific investigations, companion animals, agricultural animals, as well as free-ranging wildlife, including various fish and other **coldblooded** species. Substantial numbers of animals are present in cities, and, in most instances, are far more diverse in species and greater in number than those in rural areas (table 6). Today’s increasing focus on urban habitats is primarily on the necessity of wildlife conservation associated with the increasing importance of urban habitat for sustaining global biodiversity. Despite its large-scale human presence, suburbia has been noted to be the largest unmanaged ecosystem in America by one wildlife biologist (cited by Louv, 2005). Although some urban and suburban environments are being actively managed, wildlife themselves are the dominant force of action and are primarily acting independently in their use of urban environments as transient visitors and as areas for colonization. In essence, urban environments are the New Frontier, and wildlife are the settlers colonizing new areas to sustain their own existence because their native habitat is being degraded and lost due to human-based actions.

This New Frontier is being expanded further by the FWS through its “Conservation in the City” program, which involves an expanded initiative for urban wildlife refuges. That initiative is an action plan associated with the FWS “Conserving the Future” focus (FWS, 2011) and is in response to a recognized growing need “to reconnect and restore conservation relevance with the growing urban population.” Ten demographically and geographically varied cities are to have new national wildlife refuges established within their borders by 2015 as part of this effort (O’Brion, 2012). This program is further endorsement of an increasing number of media reports and other presentations of the past decade proclaiming, “Cities are not just for people anymore” (Reed, 2012). Those media commentaries have primarily been focused on wildlife as pest species due to their excess numbers and on reporting human-wildlife conflicts. Thus, it is noteworthy that New York City’s Central Park was originally seen as a necessary aid to both civic consciousness and public health and is the first professionally designed urban park in the United States (Louv, 2005). Paradoxically, Central Park has been a recent focus for rabies and other wildlife disease investigations.

The interfaces between humans and the various animal species present in urban environments facilitate the transfer of pathogens between humans and animals. (Photo by Milton Friend)

Table 6. Examples of animals commonly present in the urban environment.

Captive animals	
Livestock	Cattle, horses
Poultry	Chickens, domestic waterfowl
Companion animals	Dogs, cats
Research animals	Mice, rats
Human-sponsored wildlife	
Zoological collections	Zoo animals
Aviculture flocks	Waterfowl, pigeons
Exotic and other wildlife pets	Snakes, ferrets, birds
Research animals	Primates, many other species
Free-ranging wildlife	
Feral animals	Dogs, cats, ducks
Indigenous wildlife species	Raccoons, skunks, deer
Colonizing wildlife species	Red fox, Canada goose
Migratory wildlife species	**Songbirds**, waterfowl

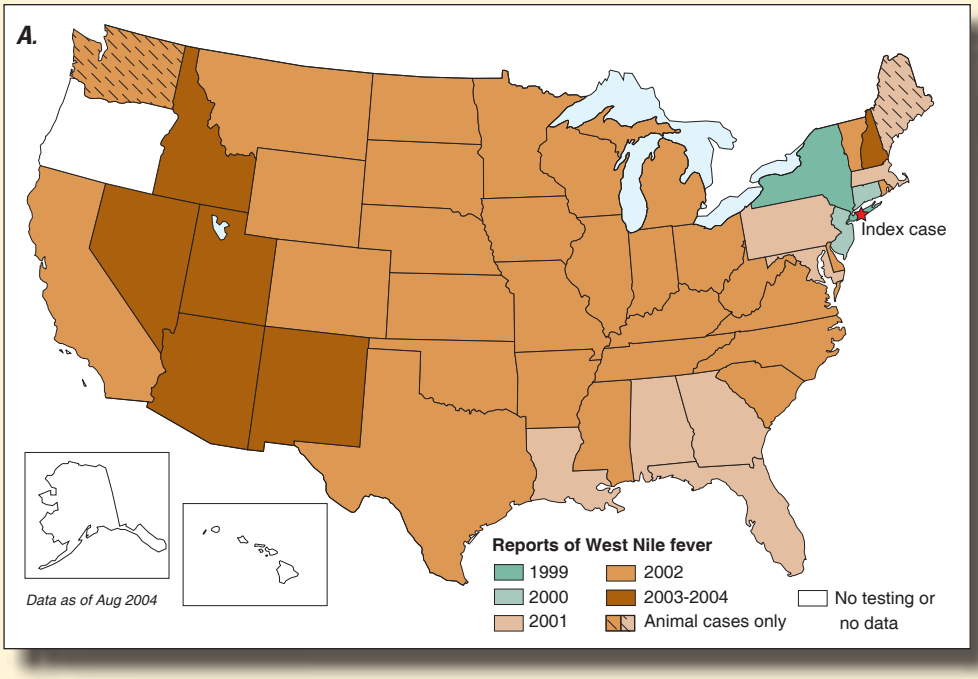
Urban Fauna and Disease

The animal life of urban environments varies greatly from one geographic area to another and with season of the year. Nevertheless, it is useful to consider the general composition of urban fauna (table 6) and their disease dynamics. For free-ranging wild birds, there is generally considerable interfacing between resident and transient populations during seasonal migration periods. Spring dispersal of wild mammals may also result in some infusion of dispersed young into and through urban areas. This interfacing of previously disparate cohorts of the same and other wildlife species provides fresh opportunities for pathogen transfers resulting in disease events. Furthermore, the infection of transient cohorts by their resident urban cohorts can facilitate disease transfer to other areas as those migrants continue their journey. The spread of house finch conjunctivitis and West Nile virus (WNV, fig. *A*) are graphic examples of the ability of birds to expand the enzootic range of infectious disease great distances.

As noted elsewhere in this publication, duck plague first appeared in North America in 1967 as the cause for a major epizootic in the Long Island, New York white Pekin duck industry. The subsequent eradication of duck plague from the commercial duck industry of the United States has been followed by numerous duck plague epizootics in urban, migratory, and other waterfowl flocks across the Nation. In addition, there have been two large-scale epizootics involving migratory waterfowl. A catastrophic 1973 epizootic appeared in waterfowl (primarily mallard ducks) wintering at the Lake Andes National Wildlife Refuge (fig. *B*) was followed more than 20 years later (1994) by a large-scale epizootic in the Finger Lakes region of New York State (primarily American black ducks).

Aggressive actions taken to combat urban waterfowl duck plague epizootics may have contributed to the rare documentation of duck plague in migratory waterfowl populations despite recurring outbreaks in a variety of urban and suburban captive and free-ranging wildlife populations (fig. *C*). However, in some instances the culling of urban waterfowl collections infected by duck plague has been vigorously opposed by various segments of society. That opposition highlights one of the difficulties associated with wildlife disease management within urban environments; companion animal status conferred upon these waterfowl by segments of the public may interfere with needed disease control actions and facilitate disease establishment and spread when eradication was possible.

Another disease dynamic of increased importance within urban environments is the transfer of pathogens between wildlife and companion animals (that is, dogs and cats). A recent study of urban areas in California and Colorado has disclosed that "domestic cats, wild bobcats and pumas [mountain lions] that live in the same area share the same diseases" (National Science Foundation, 2012). The passage of those pathogens from wildlife to domestic cats provides a vehicle for bringing those diseases into the home, thereby bridging an "infection gap" between people and wildlife. Rabies, plague, and tularemia are among the diseases of wildlife that cats and dogs have brought into the home. There is also potential for companion animals to transmit their pathogens to free-ranging wildlife. The establishment of parvovirus and heartworm infections in wolves and wildlife rabies in the United States are examples of disease transfers from infected dogs to wild mammals (appendix 1).



A, Reports of West Nile fever in the United States, 1999–2003. (Friend, 2006a)

Urban Fauna and Disease

Pathogen-laden feces are a common means for disease transmission. Infection of the southern sea otter with toxoplasmosis (appendix 1) is an example of the transfer of an infectious pathogen from the domestic cat to a marine mammal (via runoff into the nearshore environment with contaminated cat feces). In addition, during 2008 an *Escherichia coli* outbreak among a cluster of children was traced to elk droppings on football fields near Denver, Colorado and resulted in a decision to cancel midget football games on fields close to where elk graze (Scanlon, 2008). Urban waterfowl commonly litter park areas and golf courses with their feces. That type of contamination periodically results in public health agencies closing public swimming areas because of *E. coli* from waterfowl feces. The feces of the raccoon, another wildlife resident of urban areas (fig. *D*), often contain eggs of the zoonotic parasite *Baylisascaris procyonis*. Human contact with those feces can result in infection involving larvae of these roundworms migrating in ocular and other tissues. A far more hazardous feces shed parasite is *Echinococcus multilocularis*, a cestode (tapeworm) of foxes. People who accidentally ingest the eggs of this parasite may develop **alveolar hydatid disease**. Because dogs and cats can also become infected and serve as definitive hosts, usually by feeding on infected small rodents (intermediate hosts), they can bring the parasite to one's home as well as to public areas where companion animals are walked or allowed to roam.

B, More than 40,000 mallards died during the 1973 outbreak of duck plague on the Lake Andes National Wildlife Refuge in South Dakota. (Photo by Milton Friend.)

C, The lagoon and area of the Palace of Fine Arts in San Francisco was the site for two of the earliest duck plague outbreaks in the United States. (Photo by Carol M. Highsmith, from the Library of Congress)

Arthropod vectors are yet another common means for disease transmission within urban environments. For example, WNV is not transmitted from bird to bird or other vertebrates. Instead infected birds infect mosquitos that feed upon them, and those mosquitoes transmit the virus to the next susceptible host they feed upon (birds, mammals, humans). Similarly, Lyme disease is transmitted by the bite of infected ticks, not from contact with deer or deer mice, both of which have roles in Lyme disease ecology.

Clearly, the animal diversity and interfaces between different species within urban environments provide multiple opportunities for pathogen transfer between species, not all of which have been noted here. For example, an outbreak of tularemia that forced temporary closure of an urban zoo likely entered the zoo through water contaminated by small rodents or by the entry of infected small rodents themselves. Human epidemics of this disease in urban areas of Europe have also been traced to the contamination of public water supplies by infected small rodents. Aerosol transmission of ornithosis (appendix 1) has caused considerable losses of wild birds in aviaries and closed their use by humans.

The "bottom line" is that urban environments are important wildlife habitat and need to be managed in ways that benefit free-ranging wildlife. Furthermore, human attitudes towards wildlife will increasingly be shaped by human experiences in urban environments, because this "New Frontier" is where most within urbanized society now interface with wildlife. Thus, it is imperative that wildlife disease be adequately addressed in these environments so that wildlife continue to be cherished.

D, The bare-footed mother and child shown feeding a raccoon were engaged in common—and risky— human behavior that is repeated countless times in urban parks and other areas throughout the world. Not only could they have been bitten by the raccoon, they might have become infected by the parasites in the feces if they had come into contact with the feces. (Photo by Milton Friend)

The Disease Factor

Urban environments are largely uncharted wilderness relative to disease management in free-ranging wildlife populations. These environments now represent the mainstream for most of society and are also an evolving spawning ground for EIDs with consequences for human and wildlife health alike. Human contributions, particularly in urban areas, to the EID potential include:

1. population density that favors so-called "crowd diseases";
2. the multidimensional components of urban areas that bring a wide array of animal species into those environments for various human purpose, such as research, zoos, country fairs, companion animals, etc.;
3. the constant movement and interactions of people and goods at local, regional, national, and international levels;
4. a substantial cohort of people with suppressed immunocompetence because of various medical conditions and (or) age-related susceptibility to infection (the very young and those with advanced age); and
5. human behaviors and actions relative to life style and other functional interactions with their environment.

The immunocompromised human population has greater risk for acquiring novel infectious diseases and for sustaining transmission of established infectious diseases. However, urban environments generally also have a well-developed medical infrastructure and technical capabilities for sustaining human health through education and other disease prevention programs, (such as, vaccination) rapid diagnostic services, and disease response. Nevertheless, many emerging zoonoses, such as WNV and severe acute respiratory syndrome (SARS),

Infected garden feeders and bird baths blamed for deaths of one fifth of Britain's greenfinches

By DAVID DERBYSHIRE
UPDATED: 04:40 EST, 8 April 2010

Share Tweet Pin it +1 Share 22 View comments

For millions of cold and hungry birds, feeders hanging in gardens are a lifeline during the hard days of winter.

But some wildlife experts are warning that they can also be a deathtrap.

They say dirty feeders and infected bird baths are spreading a disease that has killed a fifth of Britain's greenfinches in recent years.

They are urging nature lovers to clean up their bird tables, and temporarily stop feeding birds if they are signs of infection in their garden.

Around 20 million people in Britain regularly leave out food for birds.

They spend £250million a year on specialist seeds and the figure is rising.

Charities and conservation groups say garden feeders are an essential resource for birds, especially during the winter.

But a conference in London next month will hear evidence that they have contributed to the spread of at least two diseases in last decade.

In Britain, around one in five greenfinches has been killed by the disease Trichomoniasis, or 'trich', since

One fifth of Britain's greenfinches have been killed by dirty feeders and infected bird baths

Wildlife feeding is a popular human activity within urban environments and is responsible for the deaths of 20 percent of European greenfinches in the United Kingdom from disease due to pathogen transmission at pathogen-contaminated feeders and birdbaths. (From the Mail Online, London)

> "The two key factors which affect the spread of infectious diseases in the human community other than the nature of the infectious agent are human ecology and behavior" (Roizman and Hughes, 1995).

are novel diseases within the area of first eruption. Because of their novelty, it is unlikely the medical infrastructure could have predicted and adequately prepared for those specific diseases without aggressive wildlife disease surveillance and monitoring programs to provide earlier indications of disease activity. Thus, human factors that may enhance the general eruption and spread of novel diseases need to be preemptively considered, as do the wide variety of infectious agents of wildlife that are generally of low pathogenicity.

Wildlife in urban environments essentially live in the human community and are similarly affected by those human factors. Thus, human interests and needs within urban landscapes result in the presence of a large and varied assembly of domestic and wild animal species in relatively close proximity to one another and the human populations of those areas. More species of animals and greater numbers of those animals are now living in close proximity with humans than ever before. Many in the large complex of urban animals may not have direct contact with one another because they live in zoos and research facilities. However, existing barriers may be breached by indirect transmission of pathogens via arthropod vectors, contaminated water, and other processes. Depending on the geographic location, species associated with the exotic pet trade may also be a component of urban fauna. An infectious disease threat associated with exotic pets is the introduction of exotic or novel pathogens into new geographic areas. The occurrence of monkeypox (appendix 1), a disease of the African continent, in the United States serves as a recent example of the materialization of this threat (see chap. 3, The Wildlife Factor, *in* Friend, 2006a).

The Reston strain of Ebola virus was brought into the United States in a 1989 shipment of primates from the Philippines to a research facility. Fortunately, this strain of Ebola has reduced ability to produce disease in primates and is not known to cause clinical disease in humans. It was again imported into the Unites States from the Philippines in different primate species in 1990 and 1996 and into Europe in 1992. The crossover of this virus to pigs was first detected in the Philippines in association with swine mortality events during 2007 and 2008 (Food and Agriculture Organization of the United Nations, 2009).

The development of urban and suburban areas as focal points for disease in free-ranging wildlife populations should be a major concern for wildlife conservation agencies. For example, it is unlikely that many of the people who feed birds also adequately clean those feeding stations as a disease prevention measure. As a result, disease at birdfeeding stations continues to result periodically in substantial epizootics involving various bird species, such as salmonellosis in passerines and trichomoniasis in **doves**. Given the number of urbanites who feed birds and the great importance of urban environments as habitat for many species of songbirds, enhanced public education about minimizing disease risks while enjoying this activity seems warranted. Various birdseed companies and other organizations such as the Audubon Society periodically provide information about birdfeeder sanitation as a means of reducing disease risks along with providing guidance for reporting illness in birds. Failure to address this problem can result in the major decline in some species as has occurred for "*garden birds*" in England due to salmonellosis and other diseases (Kirkwood, 2008; Kirkwood and MacGregor, 1998; *see also* the European greenfinch example in "The Disease Factor").

Wildlife-associated disease events in urban environments are not limited to birdfeeding stations (fig. 20). In essence, they are reflective of the full spectrum of disease events encountered in nature. For example, type C avian botulism (a noninfectious disease) has killed waterfowl at the Reflecting Pool on the Mall in the Nation's capital and at a lagoon at Chicago O'Hare National Airport. Sylvatic plague has recently killed small rodents within city parks in Denver, Colorado and Flagstaff, Arizona. Rabies has recently become an increasing problem within northern Virginia urban areas. Duck plague has been a recurring problem in urban waterfowl within various states. Distemper (appendix 1) is a common disease of urban raccoons, and a variety of diseases, including West Nile fever, occur in urban bird populations.

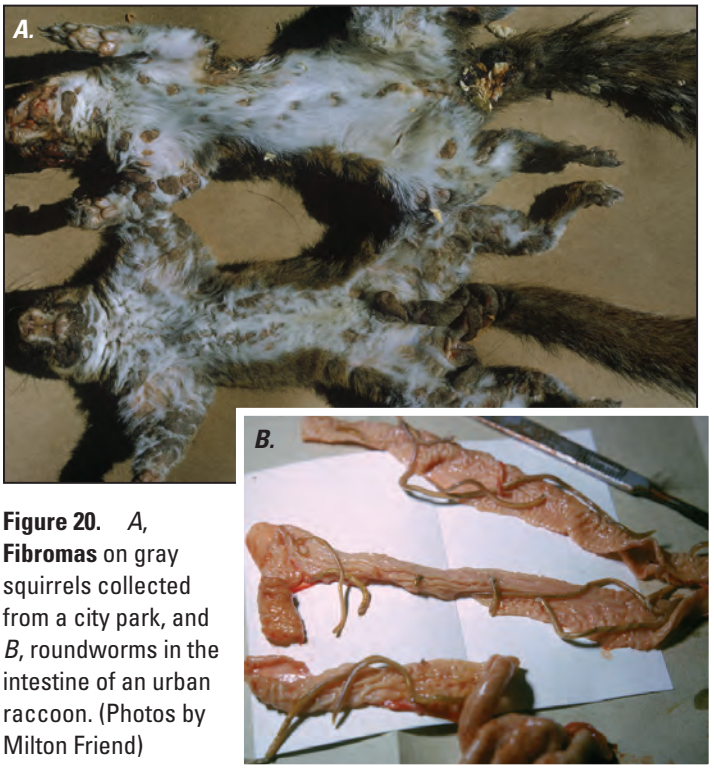
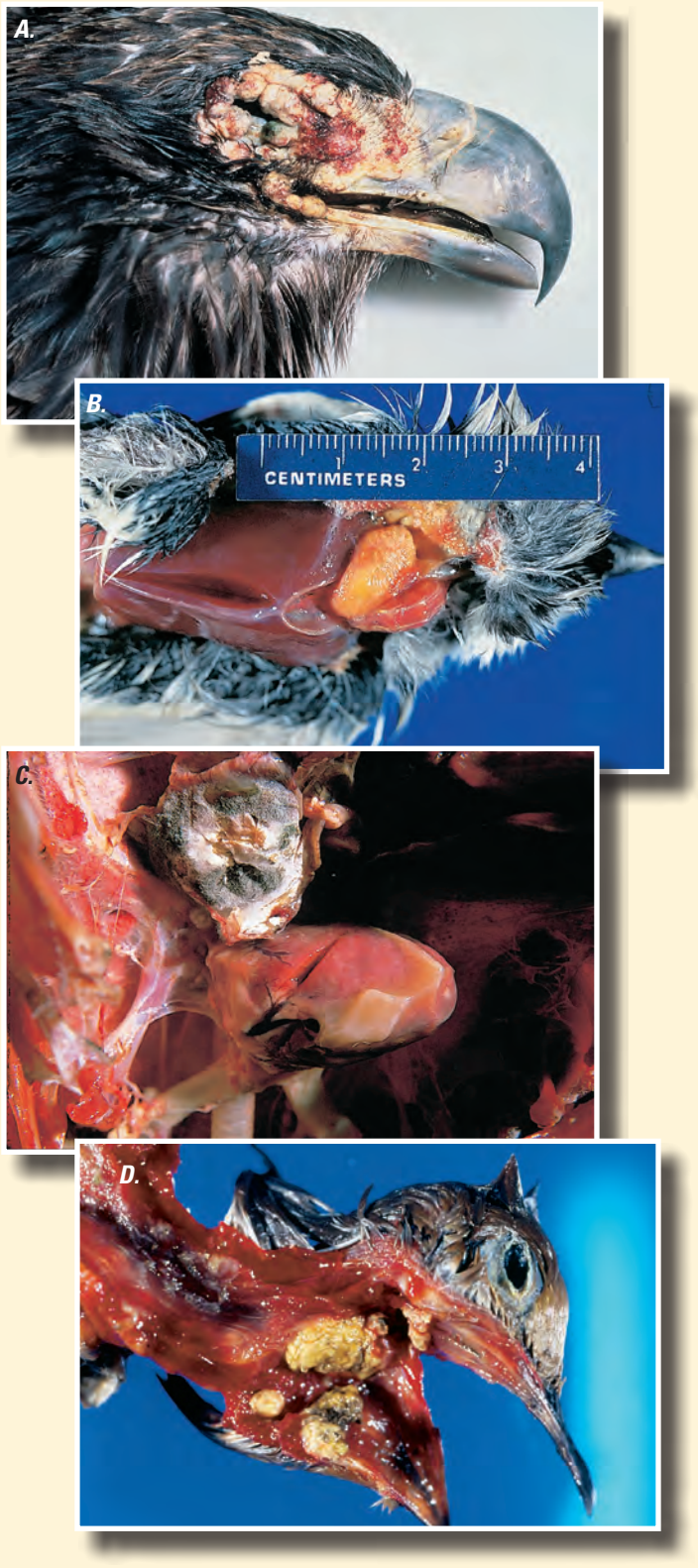


Figure 20. *A*, **Fibromas** on gray squirrels collected from a city park, and *B*, roundworms in the intestine of an urban raccoon. (Photos by Milton Friend)

Birdfeeding and Disease

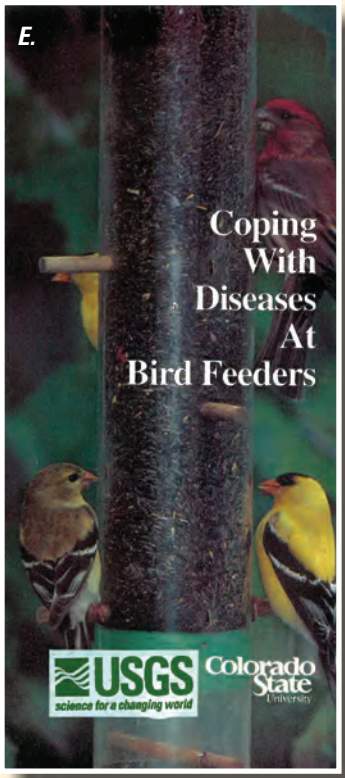
Despite its popularity as an activity and the human pleasures derived from viewing the species of birds attracted to feeding sites, birdfeeding is associated with major bird losses from disease. In various ways, birdfeeding stations provide "equal opportunity" for different classes of pathogens to "feed" at the same "cafeterias" being enjoyed by the birds. Thus, avian pox (viral, fig. *A*), salmonellosis (bacterial, fig. *B*), aspergillosis (fungal, fig. *C*), and trichomoniasis (parasitic, fig. *D*) have been frequent visitors along with other less common diseases. Also, in keeping with the "cafeteria style venue," secondary infections such as trichomoniasis often occur in **raptors** that feed upon the "songbirds" dining at those feeding stations. Birdfeeding is so prevalent in urban areas that some raptor populations have adapted their hunting strategies to the extent that they have established feeding patterns that are clearly associated with the distribution of bird feeders within an area. On several occasions I have been privileged to view a dove or some other species being taken from my urban backyard feeder by a swiftly maneuvering Cooper's or sharp-shinned hawk.

Given current and projected patterns of human demographics, it is highly likely that birdfeeding will continue to increase in popularity within urban and suburban areas. The potential for this activity to enhance bird losses from disease within these environments needs to be preemptively addressed through public education and outreach efforts that sensitize people to the need for maintaining clean feeders and feeding areas (fig. *E*). Because novel disease events for **land birds** may first be encountered at feeding stations (for example, **mycoplasmal conjunctivitis** in house finches), early detection coupled with appropriate timely intervention are important for preventing disease establishment and spread. Thus, diagnostic assessments of dead and visibly "diseased-like" birds within urban environments need to become a priority for combatting wildlife disease within these environments. The rapid spread of mycoplasma conjunctivitis across the entire eastern range for this species (fig. *F*) provides testimony to the need for a greater focus on disease in nongame species within urban environments.



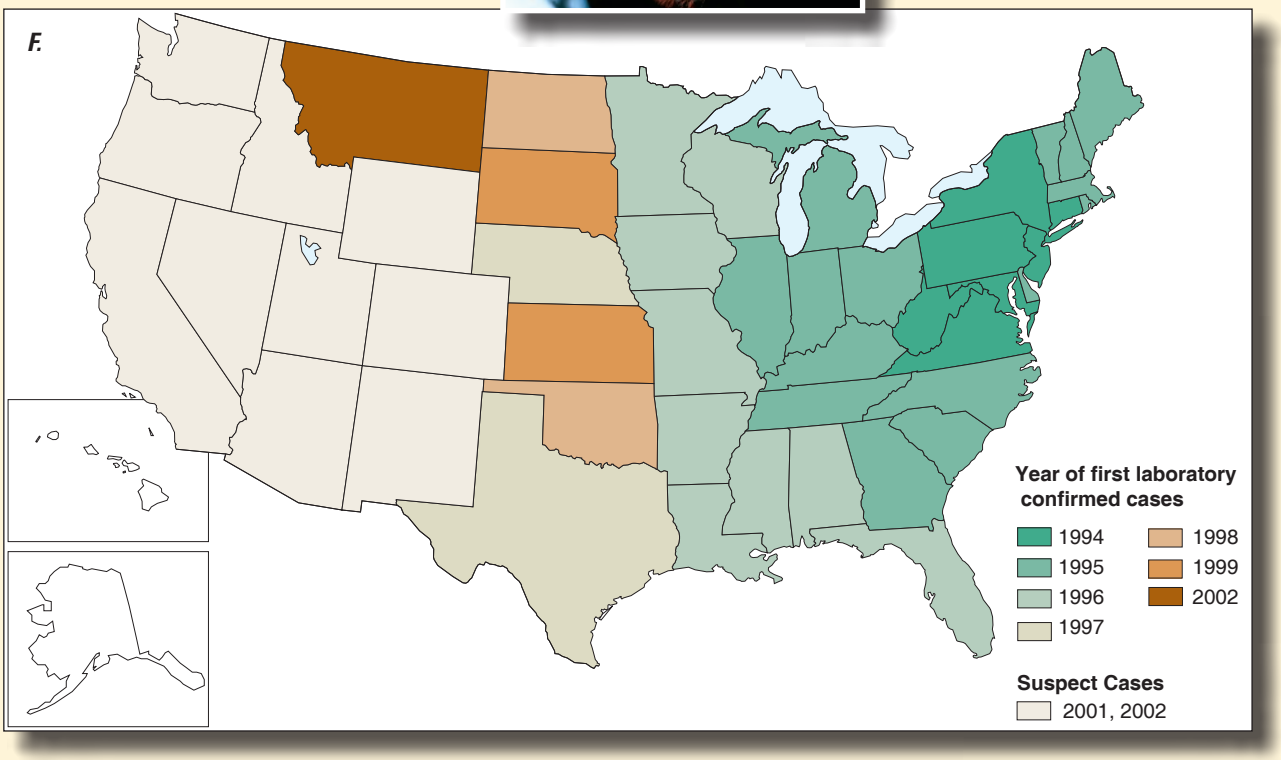
Examples of diseases seen in birds that feed at birdfeeders and in other birds that feed on such birds. *A*, Avian pox lesions, such as those on the face of this bald eagle, can impair breathing, sight, and feeding so much that a bird may die. *B*, The large, yellow, cheesy area in the esophagus of this English sparrow was caused by infection with salmonellosis. *C*, The cheesy fungal plaques and "bread mold" fungal mat in this bird's air sacs are due to chronic aspergillosis. *D*, Trichomoniasis is the cause of the yellow, cheesy growths in the esophagus of this mourning dove. (Photos by: *A*, James Runningen; *B*, J. Christian Franson; *C*, Milton Friend; *D*, J. Christian Franson)

Birdfeeding and Disease



E, Educational publications of this type are useful for addressing disease in the backyard environment.

F, Reported geographic spread of house finch inner eyelid inflammation (conjunctivitis) since the initial 1964 *Mycoplasma gallisepticum* observation. (Friend, 2006a; photo by Terry Creekmore)



From a wildlife conservation orientation, it is inconsistent and counterproductive to pursue the development of urban habitat for sustaining global biodiversity and not aggressively address wildlife disease within that habitat. It is also counterproductive to pursue the development of "natural areas" and other habitat for the enhancement of wildlife presence within urban areas but not have an aggressive disease monitoring and surveillance system focused on zoonotic disease. A paradox is that the presence of various wildlife species in urban areas fosters human connectivity with nature, an important need for supporting wildlife conservation in our increasingly electronically oriented society. Further, the presence of various free-ranging wildlife species within urban environments is generally beneficial for human health and a positive contribution to the quality of life associated with specific urban areas.

Addressing disease in free-ranging wildlife in urban environments has far greater need for preplanning and the establishment of operational and response protocols than any other situations other than the diagnosed appearance of a highly pathogenic zoonosis or a suspected act of bioterrorism. That preplanning is needed to assure the opportunity for time-sensitive field assessments and responses involving free-ranging wildlife in multiple jurisdictions. The need for time-sensitive assessments and responses is associated with the great diversity of potential host species available for infection and disease spread, high-density populations of some species that facilitates disease maintenance, and the fluidity of potential host species and goods within and through the areas that may facilitate disease spread to other areas.

Concluding Commentary

The tsunami of infectious disease impacting free-ranging wildlife populations since the last half of the 20th century is unprecedented in recorded history and shows no signs of abating during the 21st century. The commentary, examples and illustrations provided within this document collectively argue that the wildlife conservation community needs to invest itself more fully in combatting disease for the well-being of free-ranging wildlife populations and the preservation of global biodiversity. Our changing world and the associated pressures on biological resources have greatly elevated disease as a factor that must be dealt with (fig. 21). As a result, the question "Why bother?" has now been transformed to that of "how can we do better?"

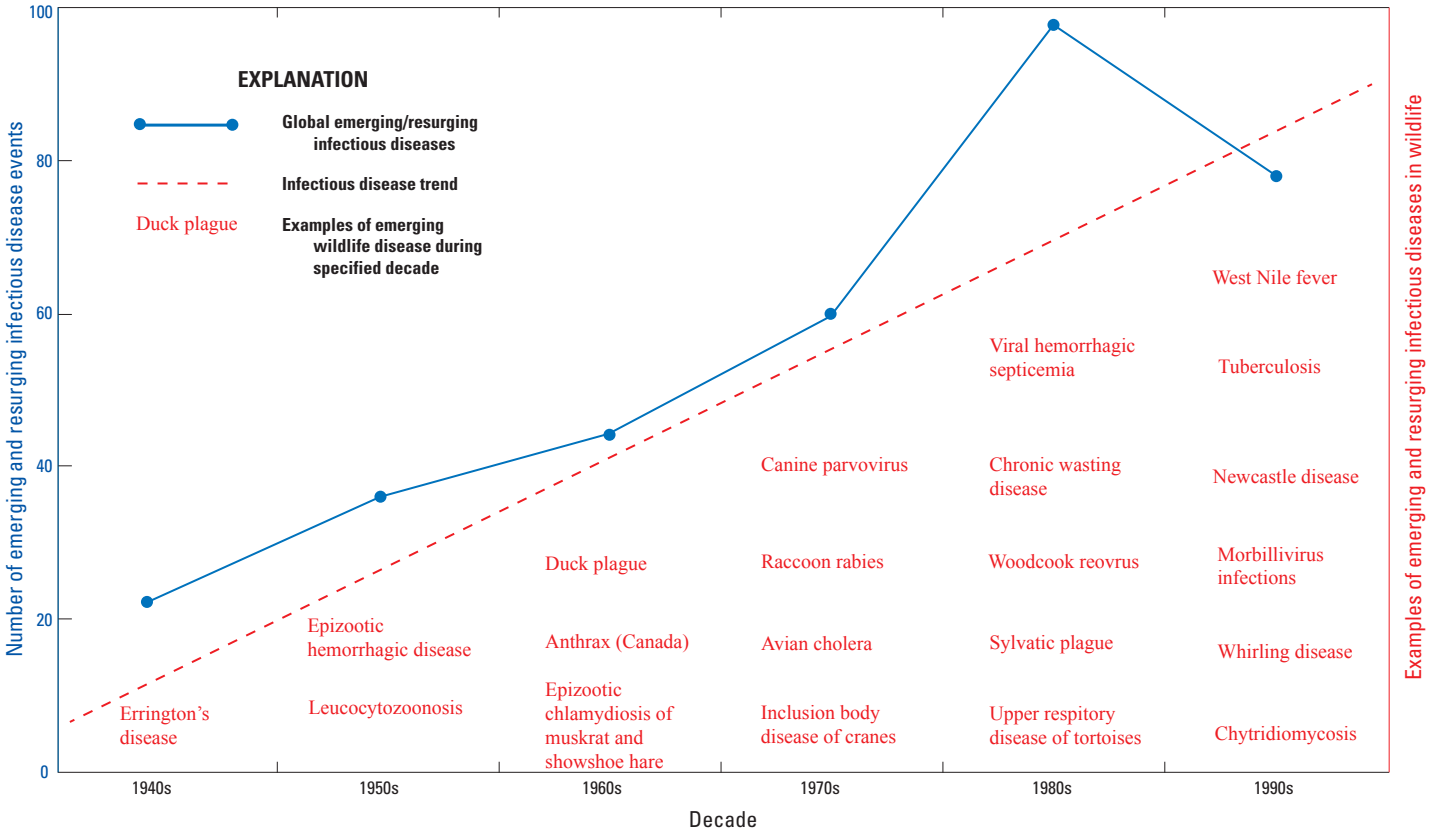


Figure 21. The number of emerging and resurging infectious disease events reported per decade during the 20th century (modified from Jones and others, 2008) and examples of emerging and resurging infectious diseases involving North American wildlife during those decades (compiled from Davis and others, 1970; 1971; Friend, 2006a). Although the number of disease events per decade peaked during the 1980s, the 1990s had the greatest total number of new and resurging wildlife diseases.

The current iteration of our New World highlights the need for and an increasing resolve to address infectious disease from a One Health perspective. Wildlife have center stage, because the majority of emerging zoonoses have wildlife roots and because an even greater number of infectious animal diseases are shared between wildlife and domestic species. In addition, infected wildlife are potential vehicles for nefarious terrorist attacks against society. Thus, our New World requires an expanded consideration of wildlife disease that includes noninfectious as well as infectious pathogens and the role of wildlife as sources for diseases impacting other species, including humans. Greater attention needs to be afforded to vector-borne diseases and disease transmission via contaminated environments, especially in urban areas. Clearly, wildlife disease considerations are now far more complex than they were as recently as the 1950s. Fortunately, our capability to address these considerations also is considerably more advanced.

The new dawn for much of humanity highlights the leading edge of two important transitions that have major ramifications for infectious disease emergence. The first of these transitions is the shifting demographics of human society; the second is the increasing colonization of human-built environments by wildlife. The intersection of these two competing transitions provides conditions for the perfect storm relative to infectious disease emergence and dynamic disease spread. In essence, metropolitan and suburban environments are the new wilderness where infectious agents apply their adaptive and evolutionary survival skills. As for earlier human population shifts to the city, current urban environments also have unseen risks for infectious disease emergence that warrant increased disease surveillance and monitoring of wildlife and other animals within those environments. Such actions are needed to provide "early warning" for the appearance of hazardous pathogens.

Wildlife disease has only recently become an important "New Frontier" for wildlife conservation. The challenges of coping with wildlife disease are likely to intensify within urban environments, where wildlife have more contact with other animals and humans. The diseases commented on and species impacts noted in this publication are but the tip of the iceberg. Since the 1970s, infectious pathogens have become an increasing cause for disease in all major ecosystems and classes of biological resources. From coral reefs to desert environments, invertebrate and vertebrate species are being severely challenged by disease. Coldblooded and **warmblooded** species alike are being confronted by pathogens not commonly encountered during their evolution. Clearly, urban environments present contemporary challenges for dealing with EIDs, human-wildlife relations, and wildlife conservation. These challenges go far beyond considerations associated with emerging zoonoses to the more difficult task of preventing these environments from becoming foci for disease eruption and transmission among free-ranging wildlife populations. Consider, for example, the potential consequences of pathogen exchanges between urban and transient wildlife during wildlife movements throughout the year, especially for birds during spring and fall migrations.

Although there are compelling wildlife conservation reasons for preemptive, proactive, and science-based development and implementation of coordinated efforts for addressing wildlife disease in urban environments, these are largely uncharted pursuits relative to benefitting wildlife living within those areas. How well they are or are not done will likely have substantial ramifications for the role of these environments in sustaining global biodiversity. Failure is not an option, for the outcome is likely to dictate the perspectives society develops and holds towards free-ranging wildlife for the foreseeable future.

Acknowledgments

I gratefully acknowledge the outstanding support and assistance provided by numerous U.S. Geological Survey (USGS) National Wildlife Health Center (NWHC) staff and contractors in the development of this presentation. NWHC librarian Mrs. Katherine Wesenberg and her staff of student service contractors—specifically Mrs. Lindsay Anderson, Ms. Lindsay Farrel, Ms. Kelly Haberstroh, Ms. Hannah Lindquist, and Mr. Stephen Nonte—were truly awesome in their responsiveness and technical abilities to locate and acquire much of the broad spectrum of literature and photograph images presented. Substantial amounts of this material are of a historic nature and (or) published in somewhat obscure journals and other media. Their timely and helpful supplemental efforts greatly reduced the time that would otherwise have been required to complete this project and also enhanced its content. The administrative support provided by Ms. Jessica Kurtz in typing the various drafts of the manuscript and in maintaining its associated electronic files during manuscript development was also invaluable, as was the high quality, timely support provided by NWHC information technology specialist Ms. Karen Cunningham. Also, I sincerely appreciate the quality input received from Ms. Elizabeth Enright of the USGS Science Publishing Network during conceptual development of this presentation, editorial feedback, and process-related guidance. Ms. Rosemary Stenback provided invaluable assistance in the development of some of the illustrations in addition to developing the publications layout. The contributions noted are those that quickly come to mind. I apologize for any oversights that may have occurred. The extended period of time in completing this publication and the frailties of a memory that accompanies advancing age makes it likely other contributions should have been mentioned but are not. Clearly, I could not have completed this project without the support provided.

References Cited

Abbott, R.C., and Rocke, T.E., 2012, Plague: U.S. Geological Survey Circular 1372, 79 p., plus appendix, available at *http://pubs.usgs.gov/circ/1372*.

Abrantes, J., Van der Loo, W., Le Pendu, J., and Esteves, P.J., 2012, Rabbit haemorrhagic disease (RHD) and rabbit haemorrhagic disease virus (RHDV): A review: Veterinary research, v. 43, no. 12, accessed December 17, 2013, at *http://www.veterinaryresearch.org/content/43/1/12*.

Adams, L.W., 1994, Urban wildlife habitats: A landscape perspective: University of Minnesota Press, 160 p.

Alexander, K.A., Pleydell, E., Williams, M.C., Lane, E.P., Nyange, J.F.C., and Michel, A.L., 2002, *Mycobacterium tuberculosis*: An emerging disease of free-ranging wildlife: Emerging Infectious Diseases, v. 8, p. 597–601.

Anderson, R.C., and May, R.M., 1982, Population dynamics of human helminth infections: Control by chemotherapy: Nature, v. 297, p. 557–563.

Anderson, W.L., Havera, S.P., and Zercher, B.W., 2000, Ingestion of lead and nontoxic shotgun pellets by ducks in the Mississippi Flyway: Journal of Wildlife Management, v. 64, p. 848–857.

Ashford, D.A., Kaiser, R.M., Bales, M.E., Shutt, K., Patrawalla, A., McShan, A., Tappero, J.W., Perkins, B.A., and Dannenberg, A.L., 2003, Planning against biological terrorism: Lessons from outbreak investigations: Emerging Infectious Diseases, v. 9, no. 5, p. 515–519.

Ball, J., 2012, What Ebola virus means for primate populations: British Broadcasting Corporation, accessed December 17, 2013, at *http://www.bbc.co.uk/nature/20593634*.

Beer, J.V., 1988, Diseases of gamebirds and wildfowl: Fordingbridge, Hampshire, United Kingdom, The Game Conservancy, 112 p.

Bierer, B.W., 1974, History of animal plagues of North America: With an occasional reference to other diseases and diseased conditions (reprint of the 1939 ed.): U.S. Department of Agriculture, 97 p.

Bolzoni, L., and De Leo, G.A., 2013, Unexpected consequences of culling on the eradication of wildlife disease: The role of virulence evolution: The American Naturalist, v. 181, no. 3, p. 301–313.

Brisson, D., Brinkley, C., Humphrey, P.T., Kemps, B.D., and Ostfeld, R.S., 2011, It takes a community to raise the prevalence of a zoonotic pathogen: Interdisciplinary Perspectives on Infectious Diseases, v. 2011, Article ID 741406, p. 6, *doi:dx.doi.org/10.1155/2011/741406*.

Brothwell, D., and Sanderson, A.T. 1967, Diseases in antiquity: Springfield, Illinois, Charles C. Thomas, 766 p.

Burnet, F.C., 1940, Natural history of infectious disease (1st ed.): New York, The MacMillan Company, 310 p.

Burnet, F.C., 1953, Natural history of infectious disease (2nd ed.): The University of California, University Press, 356 p.

Burnet, F.C., 1962, Natural history of infectious disease (3rd ed.): New York, The MacMillan Company, 377 p.

Burnet, F.C., and White, D.O., 1972, Natural history of infectious disease (4th ed.): Cambridge, Cambridge University Press, 278 p.

Cameron, J., 1929, The Bureau of Biological Survey: Its history, activities, and organization: New York, Johns Hopkins Press, 339 p.

Carter, S.P., Delahay, R.J., Smith, G.C., Macdonald, D.W., Riordan, P., Etherington, T.R., Pimley, E.R., Walker, N.J., and Cheeseman, C.L., 2007, Culling-induced social perturbation in Eurasian badgers *Meles meles* and the management of TB in cattle: An analysis of a critical problem in applied ecology: Proceedings of the Royal Society of London, Series B, v. 274, p. 2769–2777.

Carson, R., 1962, Silent spring: Boston, Houghton Mifflin Company, p. 386.

Centers for Disease Control and Prevention, 1994, Addressing emerging infectious diseases, a prevention strategy for the United States: Atlanta, Georgia, U.S. Department of Health and Human Services, Public Health Service, p. 31

Centers for Disease Control and Prevention, 2005, Compendium of measure to prevent disease associated with animals in public settings: National Association of State Public Health Veterinarians, Inc. (NASPHV), CDC Recommendations and Reports, no. 54(RR04), 28 p., accessed December 7, 2012, at *http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6004a1.htm*.

Cleaveland, S., Laurenson, M.K., and Taylor, L.H., 2001, Diseases of humans and their domestic mammals: Pathogen characteristics, host range and the risk of emergence: Philosophical Transactions of the Royal Society of London, Series B, Biological Sciences, v. 356, p. 991–999.

Cohen, J.E., 2003, Human population: The next half century: Science, v. 302, p. 1172–1175, accessed at *http://www.sciencemag.org*.

Constantine, D.G., 2009, Bat rabies and other lyssavirus infections: U.S. Geological Survey Circular 1329, 68 p., available online at *http://pubs.usgs.gov/circ/circ1329/*.

Crawford, D.H., 2007, Deadly companions: How microbes shaped our history: New York, Oxford University Press, 250 p.

Daszack, P., Cunningham, A.A., and Hyatt, A.D., 2000, Emerging infectious diseases of wildlife—Threats to biodiversity and human health: Science, v. 287, p. 443–449.

Davies, J.C., III, 1970, The politics of pollution: New York, Pegasus, 231 p.

Davis, J.W., Anderson, R.C., Karstad, L., and Trainer, D.O., (eds.), 1971, Infectious and parasitic diseases of wild birds: Ames, Iowa State University Press, 344 p.

Davis, J.W., Karstad, L.H., and Trainer, D.O., (eds.), 1970, Infectious diseases of wild mammals: Ames, Iowa State University Press, 421 p.

Dean, D.J., 1961, Streptothricosis: A new deer disease transmissible to man: The Conservationist (New York State Conservation Department Information Leaflet, p. B–39–B–40.

Dean, D.J., Gordon, M.A., Severinghaus, C.W., Kroll, E.T., and Reilly, J.R., 1961, Streptothricosis: A new zoonotic disease: New York State Journal of Medicine, v. 61, p. 1283–1287.

de Castro, F., and Bolker, B., 2005, Mechanisms of disease-induced extinction: Ecology Letters, v. 8, no. 1, p. 117–126.

Deplazes, P., Hegglin, D., Gloor, S., and Romig, T., 2004, Wilderness in the city: The urbanization of *Echinococcus multilocularis*: Trends in Parasitology, v. 20, p. 7784.

Drewin, R.C., and Bizean, E.G., 1978, Cross-fostering whopping cranes to sandhill crane foster parents, *in* Temple, S.A. ed., Endangered birds: Management techniques for preserving threatened species: Madison, University of Wisconsin Press, p. 201–222.

Ellis, D.H., Lewis, J.D., Gee, G.F., and Smith, D.G., 1992, Population recovery of the whooping crane with emphasis on reintroduction efforts: Past and future: Proceedings of Sixth North American Crane Workshop, p. 142–150.

Elton, C., 1931, The study of epidemic disease among wild animals: Journal of Hygiene, v. 31, p. 435–456.

Ewald, P.W., 1994, Evolution of infectious disease: New York, Oxford University Press, 298 p.

Ferber, D., 2000 Human diseases threaten great apes: Science, v. 289, p. 1277–1278.

Fenner, F., and Ratcliffe, F.N., 1965, Myxomatosis: Cambridge, Cambridge University Press, 379 p.

Fine, P.E.M., Aron, J.L., Berger, J., Bradley, D.J., Berger, H.J., Knox, E.G., Seeliger, H.P.R., Smith, C.E.G., Ulm, K.W., and Yekutiel, P., 1982, The control of infectious disease group report, *in* Anderson, R.M., and May, R.M., eds., Population biology of infectious diseases: Berlin, Springer-Verlag, p. 121–147

Fishbein, D.B., and Robinson, L.E., 1993, Rabies: New England Journal of Medicine, v. 329, p. 1632–1638.

Fleming, G., 1871, Animal plagues: Their history, nature, and prevention: London, Chapman and Hall, 548 p.

Food and Agriculture Organization of the United Nations, 2009, First detection of Ebola-Reston virus in pigs: 5 p., accessed January 14, 2009, at *http://www.porkworld.com.br/index.php?documento=5418*.

Franson, J.C., Friend, M., Gibbs, S.E.J., and Wild, M.A., eds., 2014, Field guide to wildlife diseases: U.S. Geological Survey Techniques and Methods, book 15 [various sections and chapters], available online at *http://dx.doi.org/10.3133/tm/15/*.

Friend, M., 1995, Conservation landmarks: Bureau of Biological Survey and National Biological Service, *in* LaRoe, E.T., Farris, G.S., Puckett, C.E., Doran, R.D., and Mac, M.J., eds., Our living resources: A report to the nation on the distribution, abundance and health of U.S. plants, animals, and ecosystems: U.S. Department of the Interior, National Biological Service, p. 7–9.

Friend, M., 1999a, Avian cholera, *in* Friend, M., and Franson, J.C., eds., 1999, Field manual of wildlife diseases, general field procedures and diseases of birds: U.S. Geological Survey, Biological Resources Division, Information and Technology Report 1999–01, p. 75–92, available online at *http://www.nwhc.usgs.gov/publications/field_manual/index.jsp*.

Friend, M., 1999b, Lead, in Friend, M., and Franson, J.C., eds., 1999, Field manual of wildlife diseases, general field procedures and diseases of birds: U.S. Geological Survey, Biological Resources Division, Information and Technology Report 1999–01, p. 317-334, available online at *http://www.nwhc.usgs.gov/publications/field_manual/index.jsp*.

Friend, M., 2006a, Disease emergence and resurgence: The wildlife-human connection: U.S. Geological Survey Circular 1285, 388 p., available online at *http://www.nwhc.usgs.gov/publications/disease_emergence/index.jsp*.

Friend, M., 2006b, Tularemia: U.S. Geological Circular 1297, 78 p., available online at *http://www.nwhc.usgs.gov/publications/tularemia/*.

Friend, M., and Franson, J.C., eds., 1999, Field manual of wildlife diseases, general field procedures and diseases of birds: U.S. Geological Survey, Biological Resources Division, Information and Technology Report 1999–01, 367 p., 8 appendixes, available online at *http://www.nwhc.usgs.gov/publications/field_manual/index.jsp*.

Geissler, E., and van Courtland Moon, J.E., eds., 1999, Biological and toxin weapons: Research, development and use from the Middle Ages to 1945: Oxford, Stockholm International Peace Research Institute and Oxford University Press, 279 p.

Goldberg, T.L., Gillespie, T.R., Rwego, I.B., Wheeler, E.R., Estoff, E.E., and Chapman, C.A., 2007, Patterns of gastrointestinal bacterial exchange between chimpanzees and humans involved in research and tourism in western Uganda: Biological Conservation, v. 135, p. 511–517.

Goldberg, T.L., Paige, S.B., and Chapman, C.A., 2012, The Kibale ecoHealth project: Exploring connections among human health, animal health, and landscape dynamics in western Uganda, *in* Aguirre, A.A., Ostfled, R.S., and Daszak, P., 2012, New directions in conservation medicine: Applied cases of ecological health: New York, Oxford University Press, p. 452–465.

Gortázar, C., Ferroglio, E., Höfle, U., Frölich, K., and Vicente, J., 2007, Diseases shared between wildlife and livestock: A European perspective: European Journal of Wildlife Research, v. 531, p. 241–256.

Grange, W.B., 1949, The way to game abundance: With an explanation of game cycles: New York, Charles Scribner's Sons, 365 p.

Green, R.E., Hunt, W.G., Parish C.N., and Newton, I., 2009, Effectiveness of action to reduce exposure of free-ranging California condors in Arizona and Utah to lead from spent ammunition, *in* Watson, R.T., and others, eds., Investigation of lead from spent ammunition: Implications for wildlife and humans: Boise, Idaho, Boise State University, p. 240–253.

Guerra de Macedo, Carlyle, 1987, Prologue *in* Acha, P.N., and Szyfres, Boris, Zoonoses and communicable diseases common to man and animals (2nd ed.): Washington, D.C., Pan American Health Organization, Scientific Publication no. 503, p. xiii.

Hamilton, M., ed., 1970, This little planet: New York, Charles Scribner's Sons, 241 p.

Hill, D.E., and Dubey, J.P., 2014, Toxoplasmosis: U.S. Geological Survey Circular 1389, 84 p., available online at *http://pubs.usgs.gov/circ/1389/*.

Hubálek, Z., 2003, Emerging human infectious diseases: Anthroponoses, zoonoses and sapronoses: Emerging Infectious Disease, v. 9, no. 3, p. 403–404.

Huntington, E., 1931, The Matamek conference on biological cycles, 1931: Science, New Series, v. 74, no. 1914, p. 229–235.

Jones, K.E., Patel, N.G., Levy, M.A., Storeygard, A., Balk, D., Gittleman, J.L., and Daszak, P., 2008, Global trends in emerging infectious diseases: Nature, v. 45, p. 990–994.

Kahn, R.E., Morozov, I., Feldmann, H., and Richt, J.A., 2012, 6th international conference on emerging zoonoses: Zoonoses and Public Health, v. 59 (suppl. 2), p. 2–31.

Kirkwood, J.K., 2008, Salmonellosis in songbirds (order Passeriformes), *in* Fowler, M.E., and R.E. Miller, eds., Zoo and wild animal medicine (6th ed.): Saint Louis, Missouri, W.B. Saunders, p. 116–169.

Kirkwood, J.K., and MacGregor, S.K., 1998, Salmonellosis in provisioned free-living greenfinches (*Carduelis chloris*) and other garden birds, *in* Proceedings of the European Association of Zoo and Wildlife Veterinarians and the British Veterinary Zoological Society 2nd Scientific Meeting: Chester, United Kingdom, p. 229–233.

Kohn, G.C., 1995, Encyclopedia of plague and pestilence: New York, Facts on File, Inc., 408 p.

König, A., 2008, Fears, attitudes and opinions of suburban residents with regards to their urban foxes: European Journal of Wildlife Research, v. 54, p. 101–109.

Lack, D., 1954, The natural regulation of animal numbers: Oxford, The Claredon Press, 343 p.

Lederberg, J., Address to Conference of the Committee on Disarmament, August 3, 1970: Congressional Record, September 11, 1970, p. E–8123–8124.

Leopold, A., 1933, Game management: New York, Charles Scribner's Sons, 481 p.

Leopold, A., 1949, A Sand County almanac: New York, Oxford University Press, 226 p.

Liautard, A., and Ellis, R.W., eds., 1908, American Veterinary Review: New York, American Veterinary Review, v. 33, p. 334.

Louv, R., 2005, Last child in the woods: Chapel Hill, North Carolina, Algonquin Books, 390 p.

MacMillian, S., 2012, New ILRI study maps hotspots of human-animal infectious diseases and emerging disease outbreaks: 36 p, accessed July 20, 2012, at *http://www.ilri.org/ilrinews/index.php/archives/categroy/story-type/report.*

MacPhee, R.D.E., and Greenwood, A.D., 2013, Infectious disease, endangerment, and extinction: International Journal of Evolutionary Biology, v. 2013, 9 p.

MacPhee, R.D.E., and Marx, P.A., 1997, The 40,000 year plague: Humans, hyperdisease, and first-contact extinctions, *in* Goodman, S.H., and Patterson, B.D., eds., Natural change and human impact in Madagascar: Washington, D.C., Smithsonian Institution Press, p. 169–217.

Mahy, B.W.J., and Brown, C.C., 2000, Emerging zoonoses: Crossing the species barrier: Revue Scientifique et Technique, Office International des Epizooties, v. 19, p. 33–40

Mateo, R., Vallverdu-Coll, N., Lopez-Antia, A., Taggart, M.A., Martinez-Haro, M., Guitart, R., and Ortiz-Santaliestra, M.E., 2014, Reducing Pb poisoning in birds and Pb exposure in game meat consumers: The dual benefit of effective Pb shot regulation: Environment International, v. 63, p. 163–168.

McCabe, R.A., 1987, Aldo Leopold, the professor: Madison, Wisconsin, Rusty Rock Press, Palmer Publications, 172 p.

McCallum, H., Tompkins, D.M., Jones, M., Lachish, S., Marvanek, S., Lazenby, B., Hockig, G., Wiersma, J., and Hawkins, C.E., 2007, Distribution and impacts of Tasmanian devil facial tumor disease: EcoHealth, v. 4, p. 318–325. (Also available at *http://dx.doi.org/10.1007/s10393–007–0118–0*.)

McDiarmid, A., 1962, Diseases of free-living wild animals: Rome, Food and Agricultural Organization of the United Nations, p. 119.

McLean, R.G., 2002, West Nile virus: A threat to North American avian species: Transactions of the 67th North American Wildlife Natural Resource Conference, p. 62–74.

McLean, R.G., and Ubico, S.R., 2007, Arboviruses in birds, *in* Thomas, N.J., Hunter, D.B., and Atkinson, C.T., eds., Infectious diseases of wild birds: Ames, Iowa, Blackwell Publishing, p. 20–28.

MacNeill, A.C., and T. Barnard, 1978, Necropsy results in free-flying and captive Anatidae in British Columbia: Canadian Veterinary Journal, v. 19, p. 17–21.

Meine, C., 1988, Aldo Leopold: His life and work: Madison, The University of Wisconsin Press, 638 p.

Monath, T.P., Kahn, L.H., and Kaplan, B., 2010, Introduction: One health perspective: ILAR Journal, v. 51, p. 193–198.

Mouritsen, K.N., and Poulin, R., 2002, Parasitism, climate oscillations and the structure of natural communities: Oikos, v. 97, no. 3, p. 462–468. (Also available at *http://dx.doi.org/10.1034/j/1600-0706.2002.970318.x*.)

Morse, S.S., 1995, Factors in the emergence of infectious diseases: Emerging Infectious Disease, v. 1, p. 7–15.

National Science Foundation, 2010, Domestic cats, and wild bobcats and pumas, living in same area have same disease: Press Release 12–020, accessed March 27, 2012, at *http://www.nsf.gov/news/news_summ.jsp?cntn_id=123011*.

Nol, P., and Friend, M., 2006, Is this safe to eat?, *in* Friend, M., ed., 2006, Disease emergence and resurgence: The wildlife-human connection: U.S. Geological Survey Circular 1285, p. 207–230.

Nol, P., Rocke, T.E., Gross, K., and Yuill, T.M., 2004, Prevalence of neurotoxic *Clostridium botulinum* type c in the gastrointestinal tracts of tilapia (*Oreochromis mossambicus*) in the Salton Sea: Journal of Wildlife Diseases, v. 40, 3, p. 414–419.

Novick, S., and Cottrell, D., eds., 1967, Our world in peril; An environment review: Greenwich, Connecticut, Fawcett Publication, Inc., 498 p.

O'Brian, B., 2012, Conservation in the city: U.S. Fish and Wildlife Service, National Wildlife Refuge System, Refuge Update, v. 9, no. 6, p. 9.

Osborn, F., 1968, Our plundered planet: New York, Pyramid Books, 176 p.

Organ, J.F., 2012, The evolution of a professional society: The Wildlife Professional, v. 6, no. 3, p. 24–31.

Palmer, T.S., 1912, Chronology and index of American game protection, 1776–1911: U.S. Department of Agriculture Biological Survey Bulletin no. 41.

Pappaioanou, M., 2010, Achieving effective inter-sectoral collaboration to prevent, detect and control the emergence and spread of zoonotic diseases: London, Chatham House Working Paper EERG/CGHS: 01/10, accessed December 17, 2013, at *http://www.chathamhouse.org/sites/default/files/public/research/eEnergy/environmenanddevelopment/0410zoonoticdiseases_wp.pdf*.

Parish, C.N., Heinrich, W.R., and Hunt, W.G., 2007, Lead exposure, diagnosis, and treatment in California condors released in Arizona, *in* Mee, A. and Hall, L.S., eds., California condors in the 21st century: Cambridge, Massachusetts, Nuttall Ornithological Club and American Ornithologists Union, Series in Ornithology, no. 2, p. 97–108.

Pedersen, A.S., and Davies, T.J., 2009, Cross-species pathogen transmission and disease emergence in primates: EcoHealth, no. 6, no. 4, p. 496–508. (Also available at *http://dx.doi.org/10.1007/s10393-010-0284-3*.)

Poinar, G., Jr. and Poinar, R., 2008, What bugged the dinosaurs?: Insects, disease, death in the cretaceous: Princeton, New Jersey, Princeton University Press, 264 p.

Quammen, D., 2012, Spillover: Animal infections and the next human pandemic: New York, W.W. Norton & Company, 592 p.

Quortrup, E.R., 1945, A report on brucellosis investigations, Yellowstone National Park, December 1–20, 1944: U.S. Fish and Wildlife Service internal report, submitted to Dr. Coburn, Director Dever Wildlife Research Center by Dr. Quortrup, Bear River Wildlife Disease Research Station, Utah, 15 p. plus table.

Quortrup, E.R., and Shillinger, J.E., 1941, 3,000 wild bird autopsies on western lake areas: Journal of the American Veterinary Medical Association, v. 99, no. 776, p. 382–387.

Randerson, J., 2002, Watch out for the tourists: New Scientist, v. 174, no. 2346, p. 10.

Rapport, D., Constanza, R., Epstein, P.R., Gaudet, C., and Levins, R., eds., 1998, Ecosystem health: Malden, Massachusetts, Blackwell Science, Inc., 372 p.

Reed, N., 2012, City critters: Wildlife in the urban jungle: Victoria, British Columbia, Orca Book Publishers, 134 p.

Rocke, T.E., and Friend, M, 1999, Avian Botulism, *in* Friend, M., and Franson, J.C., eds., 1999, Field manual of wildlife diseases, general field procedures and diseases of birds: U.S. Geological Survey, Biological Resources Division, Information and Technology Report 1999–01, p. 271–281, available online at *http://www.nwhc.usgs.gov/publications/field_manual/index.jsp.*

Rocke, T.E., Nol, P., Pelizza, C., and Sturm, K.K., 2004, Type C botulism in pelicans and other fish-eating birds at the Salton Sea: Studies in Avian Biology, no. 27, p. 136–140.

Roess, A.A., Galan, A., Kitces, E., Li, Y., Zhao, H., Paddock, C.D., Adem, P., Goldsmith, C.S., Miller, D., Reynolds, M.G., Zaki, S.R., and Damon, I.K., 2010, Novel deer-associated parapoxvirus infection in deer hunters: New England Journal of Medicine, v. 363, no. 27, p. 2621–2627.

Roizman, B., and Hughes, J.H., 1995, Preface, *in* Roizman, B., ed., Infectious diseases in an age of change: The impact of human ecology and behavior on disease transmission: Washington, D.C., National Academy of Sciences, National Academies Press, p. iii.

Rudd, R.L., 1964, Pesticides and the living landscape: Madison, The University of Wisconsin Press, 320 p.

Scanlon, B., 2008, Evergreen *E. coli* outbreak traced to elk droppings: Rocky Mountain News, November 11, 2008, accessed November 13, 2008, at *http://www.rockymountainnews.com/news/2008/nov/11/evergreen-e-coli-outbreak-traced-elk-droppings/*.

Sheffield, J., 2010, Theodore Roosevelt, "Conservation as a national duty (13 May 1908)": Voices of Democracy, 5, p. 89–108.

Smith, K.F., Sax, D.F., and Lafferty, K.D., 2006, Evidence for the role of infectious disease in species extinction: Conservation Biology, v. 20, p. 1349–1357.

Smithcors, J.F., 1958, The history of some current problems in animal disease VII, rabies: Veterinary Medicine, v. 53, p. 149–154.

Stedman, T.L., 2006, Steadman's medical dictionary, 28th ed.: Philadelphia, Lippincott Williams & Wilkins, 2100 p.

Steele, J.H., 1975, History of rabies, *in* Baer, G.M., ed., 1975, The natural history of rabies: Volume 1: New York, Academic Press, p. 1–29.

Taylor, L.H., Latham, S.M., and Woolhouse, M.E.J., 2001, Risk factors for human disease emergence: Philosophical Transactions of the Royal Society of London, Series B, v. 356, p. 983–989.

Trainer, D.O., and Karstad, L.H., 1970, Epizootic hemorrhagic disease, *in* Davis, J.W., Karstad, L.H., and Trainer, D.O. eds., Infectious diseases of wild mammals: Ames, Iowa State University Press, p. 50–59.

U.S. Fish and Wildlife Service, 2011, Conserving the future: Wildlife refuges and the next generation: U.S. Fish and Wildlife Service, the National Wildlife Refuge System, 93 p., accessed December 17, 2013, at *http://americaswildlife.org/wp-content/uploads/2012/01/Final-Document-Conserving-the-Future.pdf*.

U.S. Fish and Wildlife Service, 2012, North American bat death toll exceeds 5.5 million from white-nose syndrome: news release on January 17, 2012, accessed December 17, 2013, at *http://www.fws.gov/northeast/feature_archive/Feature.cfm?id=794592078*.

Van Dyke, F., 2008, Conservation biology: New York, McGraw Hill, 478 p.

Waltner-Toews, D., 2004, Ecosystem sustainability and health: A practical approach: New York, Cambridge University Press, 150 p.

Weber, N.L., Carter, S.P., Dall, S.R.X., Delahay, R.J., McDonald, J.L., Bearhop, S., and McDonald, R.A., 2013, Badger social networks correlate with tuberculosis infection: Current Biology, v. 23, no. 20, p. R915–R916.

Webster, N., 1799, A brief history of epidemic and pestilential diseases: With the principal phenomena of the physical world, which precede and accompany them, and observations deduced from the facts sheet: in two volumes: v. I.: Hartford, Connecticut, Hudson and Goodwin, 352 p.

Weese, J.S, McCarthy, L., Mossop, M., Martin, H., and Lefebvre, S., 2007, Observation of practices at petting zoos and the potential impact on zoonotic disease transmission: Clinical Infectious Diseases, v. 45, p. 10–15.

Wetmore, A., 1919, Lead poisoning in waterfowl: U.S. Department of Agriculture Bulletin 793, 12 p.

Wheelis, M., 1999, Biological warfare before 1914, *in* Geissler, E., and van Courtland Moon, J.E., eds., 1999, Biological and toxin weapons: Research, development and use from the Middle Ages to 1945: Oxford, Stockholm International Peace Research Institute and Oxford University Press, p. 8–34.

Wheelis, M., 2002, Biological warfare at the 1346 siege of Caffa: Emerging Infectious Diseases, v. 8, p. 971–975.

Williams, E.S., Kirkwood, J.K., and Miller, M., 2001, Transmissible spongiform encephalopathies, *in* Williams, E.S., and Baker, I.K. eds., Infectious diseases of wild mammals: Ames, Iowa State Press, p. 292–301.

Wobeser, G.A., 1994, Investigation and management of disease in wild animals: New York, Plenum Press, 265 p.

Wolfe, N.D., Dunavan, C.P., and Diamond, J., 2007, Origins of major human infectious diseases: Nature, v. 447, p. 279–283.

Wong, D., Wild, M.A., Walburger, M.A., Higgins, C.L., Callahan, M., Czarnecki, L.A., Lawaczeck, E.W., Craig, E., Levy, J., Patterson, G., Sunenshine, R., Adem, P., Paddock, C.D., Zaki, S.R., Petersen, J.M., Schriefer, M.E., Eisen, R.J., Gage, K.L., Griffith, K.S., Weber, I.B., Spraker, T.R., and Mead, P.S., 2009, Primary pneumonic plague contracted from a mountain lion carcass: Clinical Infectious Disease, v. 49, p. e.33–38.

Yuill, T., 1970, Myxomatosis and fibromatosis, *in* Davis, J.W., Karstad, L.H., and Trainer, D.O., eds., Infectious diseases of wild mammals: Ames, Iowa State University Press, p. 104–130.

Zinsser, H., 1935, Rats, lice, and history: Boston, Little, Brown and Company.

Zinsstag, J., Schelling, E., Waltner-Toews, D., and Tanner, M., 2011, From “one medicine” to “one health” and systemic approaches to health and well-being: Preventive Veterinary Medicine, v. 101, p. 148–156. (Also available at *http://dx.doi.org/10.1016/j.prevetmet.2010.07.003*.

Additional Reading

USGS National Wildlife Health Center publications about wildlife disease are available online at *http://www.nwhc.usgs.gov/publications/*. General sources of information about wildlife disease not cited in the text are listed below.

Alibek, K, and Handelman, S., 1999, Biohazard: The chilling true story of the largest covert biological weapons program in the world—Told from inside by the man who ran it: New York, Random House, 336 p.

Atkinson, C.T., Thomas, N.J., and Hunter, D.B., (eds.) 2008, Parasitic diseases of wild birds: Ames, Iowa, Wiley-Blackwell, 595 p.

Cohen, M.L., 2000, Changing patterns of infectious disease: Nature, v. 406, p. 762–767, available online at *http://dx.doi.org/10.1038/35021206*.

Davidson, W.R., 2006, Field manual of wildlife diseases in the southeastern United States (3rd ed.): Athens, University of Georgia, College of Veterinary Medicine, 428 p. plus glossary, index, and one appendix.

Fairbrother, A., Locke, L.N., and Hoff, G.L. (eds.), 1996, Noninfectious diseases of wildlife: Ames, Iowa State University Press, 219 p.

Thomas, N.J., Hunter, D.B., and Atkinson, C.T., (eds.) 2007, Infectious diseases of wild birds: Blackwell Publishing Professional, 484 p.

Williams, E.S., and Barker, I.K., (eds.), 2001, Infectious diseases of wild mammals: Ames, Iowa State University Press, 558 p.

Wobeser, G.A., 1997, Disease of wild waterfowl (2nd ed.): New York, Plenum Press, 324 p.

Wobeser, G.A., 2005, Essentials of disease in wild animals: Blackwell Publishing Professional, 256 p.

Wobeser, G.A., 2007, Disease in wild animals: Investigation and management: New York, Springer, 393 p.

Glossary

Developed in part from Morris, C., (ed.), 1992, Academic press dictionary of science and technology: San Diego, Harcourt Brace Jovanovich, 2432 p.

A

alveolar hydatid disease Infection of humans with larval forms (hydatid cysts) of the tapeworm *Echinococcus multilocularis*.

amphibians Coldblooded animals characterized by moist, smooth skin that live both on land and in water at various life stages and that have gills at some stage of development; that is, frogs, toads, salamanders.

antelope Deerlike mammal with true horns rather than antlers; in North America the pronghorn antelope; in the Old World numerous species; for example, eland, blackbuck, and impala.

aquatic furbearers Wildlife furbearers whose sustaining habitat is primarily aquatic; for example, muskrat and beaver.

arthropod A member of the phylum Arthropoda, invertebrate animals that have exoskeletons, segmented bodies, and jointed legs, including insects, crabs, spiders, etc.

arvicoline rodents Water voles (*Arvicola terrestris)* the largest miroline rodents of the world; heavily trapped for its fur and a major victim and carrier of tularemia.

B

bacteria (bacterium, singular) Microscopic, unicellular organisms that have distinct cell membranes and lack a distinct nucleus surrounded by a nuclear membrane.

bats Mammals in which the forelimbs have developed as wings, making them the only mammals in the world naturally capable of flight. There are estimated to be about 1,100 species of bats worldwide, accounting for about 20 percent of all mammal species.

benthic The bottom area of deep water bodies such as the ocean.

Black Death The common name given to the bacterial disease plague (especially bubonic plague) during medieval times.

bubonic plague A severe bacterial disease of humans due to infection by *Yersinia pestis*; acute regional enlargement and inflammation of lymph nodes (buboes) is typical of this most common form of plague in humans. The "Black Death" of earlier times.

C

calicivirus A family of RNA viruses (Caliciviridae) that cause disease in various species of wildlife, domestic animals, and humans (infants).

canid A mammal within the family Canidae; for example, wolves, coyotes, jackals, foxes, and other doglike animals.

canine rabies Typically a term used to denote rabies infection involving dogs, but the term is often extended to rabies in doglike species such as the wolf.

carnivore Mammals with teeth and other body adaptations for feeding on flesh; primarily species belonging to the order Carnivora such as wolves, bears, raccoons, weasels, civets, hyenas, and tigers.

civet cats Any one of the multiple genera of medium-sized carnivores within the family Viverridae. Utilized as food in some parts of Asia and thought to be the source of the virus causing severe acute respiratory syndrome (SARS) in humans.

coldblooded Species such as fishes and reptiles, which have blood that varies in temperature to approximately that of the surrounding environment.

commensal rodents Small rodent species (for example, mice, rats) that live with or near people and depend on humans, at least partially, for food and shelter.

cranes Long-legged wading birds; within North America, the sandhill and whooping cranes.

crow family Along with jays and magpies, the crow family constitutes the Corvidae within the order Passeriformes; crows themselves are cosmopolitan, medium-sized, chunky birds with all black (ebony hued) coloration.

crows Primarily the American and fish crows in North America.

D

deer Pertaining to the white-tailed deer (including subspecies) and mule deer (black-tailed deer) as native species in North America. The European red deer, axis deer (chital) from Asia, and fallow deer (Europe and Asia minor) are exotic species maintained in captive herds.

definitive host An organism in which sexually mature stages of a parasite occur.

doves Pigeonlike birds belonging to the family Columbidae. Domestic pigeons are often referred to as "rock doves." Within North America, mourning doves and white-winged doves are the most abundant wild species.

dreissenid mussels Invertebrate bivalves of the family Dreissenidae (type genus *Dreissena*); the zebra mussel (*Dreissena polymorpha*) is an exotic species within the Great Lakes Basin and is believed to be a factor in the ecology of avian botulism type E within that basin.

E

emerging infectious diseases (EIDs) Infectious diseases that have newly appeared and (or) increased in frequency of occurrence within the past three decades, or threaten to increase in the near future relative to populations affected, geographic distribution, or magnitude of effects.

enzootic An animal disease that commonly is present within a population or geographical area.

epizootic A disease affecting a greater number of animals than normal; typically, epizootics involving many animals in the same region at the same time.

extant To stand out or above; currently existing.

F

fibroma A noncancerous tumor primarily consisting of fibrous or fully developed connective tissue.

flying fox Large, fruit-eating bats with a foxlike appearance of the head. These bats are also commonly referred to as "fruit bats."

fomite(s) Inanimate objects, not inherently harmful, that may harbor pathogenic microorganisms or toxins and thus serve as an agent of pathogen transmission; for example, a blanket contaminated with smallpox during earlier times; a letter contaminated with ricin during current time.

furbearers Here, wildlife species raised in captivity or pursued in nature because of the economic value and utility of their pelts by humans. Examples include muskrat, beaver, fox, and mink.

free-ranging wildlife Wildlife living unconfined in nature.

G

game (species) As used here, "game species" refers to species that humans pursue during hunting and sport fishing activities for subsistence, recreation, or for both purposes.

game disease Diseases associated with game species.

game farm A facility devoted to the production and rearing of game species.

garden birds Typically songbirds and other small landbirds that frequent birdfeeders in urban and suburban areas.

gnotobiotic Pertains to germ- (pathogen) free environments in which host rearing and maintenance occurs in the presence of microflora and microfauna specifically and entirely known in their entirety.

great apes Apes of the family Pongidae, such as the gorilla, chimpanzee, and orangutan.

grouse Ground-dwelling, chickenlike birds of the family Tetronidae with short-to-medium-length tails (in contrast to pheasants); referring here to the ruffed grouse (*Bonasa umbellus*), unless specified otherwise.

H

hawks Birds of prey in the family Accipitridae; characterized by a strong hooked bill and powerful toes with hooked nails.

hummingbirds Brightly colored, small, New World birds within the family Trochilidae with long, slender bills and rapid wing movements that allow them to hover.

I

infectious disease A disease caused by the invasion of a host by pathogenic microorganisms. The pathogen may be a bacterium, virus, fungus, parasite, or a prion (infectious protein).

L

laissez faire Without regulation or control.

land birds Bird species that utilize terrestrial environments as primary habitat; for example, doves, songbirds, and many others.

M

marsupials Mammalian species having an external abdominal pouch (marsupium) for carrying their young until their development is complete; young of these species are born in a very underdeveloped state and must be carried and nourished for a prolonged period of time; for example, opossums, kangaroos, koala, and wombats.

megafauna In this publication, "megafauna" refers to the large animals of prehistoric time such as the dinosaurs, mammoths, and others.

metazoan The multicelled animals of the animal kingdom, in contrast to parasites, which are single-celled species, such as protozoans.

mice Any of numerous small rodents of the family Muridae having pointed snouts; long, nearly hairless tails; and small rounded ears.

N

natural resource agencies Those agencies that have jurisdictional authority mandates to provide for the stewardship of living biological resources.

O

offal The viscera, refuse, or waste (for example, the entrails) discarded from harvested animals.

One Health See chap. A1 of the "Field Manual of Wildlife Diseases" (Franson and others, 2014) for an expanded explanation; a renewed effort to stimulate greater interdisciplinary approaches and collaboration between human, domestic animal, and wildlife agency programs to combat infectious disease emergence and spread, for the good of all.

owls Nocturnal, predatory birds of the order Strigiformes, distinguished by a large head, an apparent absence of a neck, forward-positioned eyes, and having strong talons.

P

pandemic An epidemic of infectious disease that is spreading through human populations across a large region, such as a continent or worldwide.

pathogen Typically, a microorganism capable of inducing disease, but broadly including all disease-inducing agents.

pathogenic The ability to cause disease.

pelicans In North America, the American white and the brown pelican.

postmortem Examination and dissection of animal carcasses performed after the death of the animal. Also, changes that occur in tissues after death.

protozoa (protozoan) One-celled animals with recognizable nucleus, cytoplasm, and cytoplasmic structures, such as amoebas, ciliates, flagellates, and sporozoans.

R

raptors Synonymous with birds of prey. Birds, including hawks, owls, falcons, and eagles, that feed on flesh.

remote-vaccination programs The delivery of vaccines to animals in nature without human presence; for example, oral delivery of rabies vaccine to foxes via vaccine laden baits dropped by aircraft and ingested by foxes.

reservoir The host population that maintains the disease agent in nature and provides a source of infection to susceptible hosts.

rickettsia A genus of bacteria typically found in the guts of lice, fleas, ticks, and mites that transmit these pathogens by their bite when they feed on humans and other animals. Rocky Mountain spotted fever, typhus, and rickettsial pox are examples of the diseases associated with these organisms.

S

signs Observable evidence of disease in animals (similar to symptoms in humans).

songbirds Small perching and singing birds, typically of the order Passeriformes, including sparrows, finches, and cardinals.

spongiform encephalopathy Pathological lesions involving degenerative disease of the brain consisting of extensive vacuolization of the cerebral cortex (outer portion of the main portion of the brain).

T

tapeworms Segmented parasitic flatworms; also referred to as cestodes.

U

upland game birds Birds of the order Galliformes such as grouse, partridge, pheasant, quail and wild turkey that are mainly grain-eating, heavy-bodied, ground-nesting birds that do not migrate and are capable of only relatively short, rapid flight.

V

vector An insect or other living organism that carries and transmits a disease agent from one animal to another.

voles Small rodents of the genus *Microtus* (and related genera) that typically have small ears, a stout body, and a rather blunt nose (in contrast to mice).

W

warmblooded Species, such as birds and mammals, that have a constant body temperature, independent of the surrounding environment.

waterbirds Bird species that utilize water environments as primary habitat; for example, waterfowl, wading birds, gulls and terns, cormorants, and many others.

waterfowl Birds within the family Anatidae, collectively; all species of ducks, geese, and swans.

Z

zoonotic potential Pathogens with the capability of transmitting infectious disease between animals and humans and vice versa.

Appendixes

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Acquired immunodeficiency syndrome (AIDS)	Human immunodeficiency virus (HIV)	Virus	AIDS is a zoonotic infection that initially transferred from nonhuman primates to humans and is now a disease maintained within human populations by human-to-human transmission.	
Alveolar hydatid disease	*Echinococcus multilocularis*	Tapeworm	Alveolar hydatid disease is an important resurging zoonotic disease of humans; the red fox is an important reservoir host.	
Anthrax	*Bacillus anthracis*	Bacterium	Anthrax is an Old World disease of antiquity that remains a significant cause of wildlife mortality in Africa. American bison in northern Canada have been known to be infected since 1960s, and occasional wildlife epizootics afflict deer in the United States.	
Aspergillosis	*Aspergillus fumigatus*	Fungus	Aspergillosis epizootics occasionally afflict wild waterfowl that feed on moldy grain.	
Avian botulism type C ("western duck disease," "western duck sickness")	*Clostridium botulinum* type C	Bacterial toxin	Avian botulism type C is the greatest killer of waterbirds, primarily waterfowl and shorebirds. Early 20th century epizootics of "Western duck disease" and "western duck sickness" in western North America killed millions of waterbirds and initiated investigations leading to the eventual identification of the specific toxin.	
Avian botulism type E	*Clostridium botulinum*	Bacterial toxin	Mortality from avian botulism type E in fish-eating birds has increased greatly in the Great Lakes region since 2000.	
Avian cholera	*Pasteurella multocida*	Bacterium	The importance of avian cholera as a disease of wild waterfowl has increased greatly since the early 1970s.	
Avian influenza (*see also* influenza)	Orthomyxovirus	Virus	Influenza viruses have varying combinations of hemagglutinin (H) and neuraminidase (N) antigens that are related to their nomenclature. During the 21st century, strains highly capable of causing disease have evolved, for example, H5N1, that cause mortality in birds and humans (*see also* table 5).	
Avian pox	Avipoxvirus	Virus	Avian pox is a major cause of mortality in some native Hawaiian forest birds.	
Avian tuberculosis	*Mycobacterium avium*	Bacterium	Avian tuberculosis is a zoonotic disease that causes major problems in facilities for propagating of captive birds and displaying waterfowl.	

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Baylisacariasis	*Baylisascaris procyonis*	Nematode (roundworm)	Baylisascaris is a zoonotic parasite of the raccoon that poses increasing risk for human infection, especially in urban and suburban areas.	
Bluetongue	Bluetongue viruses	Virus	Along with epizootic hemorrhagic disease (EHD), bluetongue is the cause of hemorrhagic disease of white-tailed deer and other wild ruminants in the United States and Canada.	
Bovine tuberculosis	*Mycobacterium bovis*	Bacterium	Bovine tuberculosis is a resurging zoonotic disease recently established in game farms that raise members of the deer family, from which it has spread to free-ranging wildlife (*see also* table 1).	
Brucellosis	*Brucella abortus*	Bacterium	Brucellosis is an important zoonotic disease that causes abortion in cattle (Bang's disease). The transfer of brucellosis to American bison has resulted in long-standing conflict in the Greater Yellowstone Basin involving the threat wildlife pose for reinfection of area cattle.	
Bubonic plague (*see also* sylvatic plague)	*Yersinia pestis*	Bacterium	Bubonic plague is a severe clinical form of plague in humans characterized by regional enlargement and inflammation of lymph nodes (buboes). Bubonic plague was knows as the "Black Death" during the medieval period.	
Canine distemper	Canine distemper virus (CDV)	Virus	Canine distemper is an important disease of wild and domestic **canids**, and it afflicts wildlife such as raccoons, skunks, and ferrets (*see also* table 1).	
Chlamydiosis (psittacosis, ornithosis, avian chlamydiosis)	*Chlamydophila psittaci* (formerly *Chlamydia psittaci*)	Bacterium	Chlamydiosis is a worldwide zoonotic disease of domestic and wild birds. Multiple strains (serovars) account for disease in different avian species as well as muskrat, snowshoe hare, and cattle.	
Chlamydiosis of koalas	*Chlamydophila* sp.	Bacteria	Widespread chlamydiosis infection in Australian koala populations is a barrier to koala population recovery.	
Chronic wasting disease (CWD)	Abnormal protein	Prion	CWD spread across the United States and Canada during the past three decades.	
Chytridiomycosis (Chytrid fungus)	*Batrachochytrium dendrobatidis*	Fungus	Chytridiomycosis is the cause of multiple local amphibian population declines and species extinctions since the later part 20th century.	
Cryptosporidiosis	*Cryptosporidium parvum*	Protozoan	Cryptosporidiosis is a globally important, often water borne, diarrheal disease of humans and animals that is spread by fecal contamination.	

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Devil facial tumor disease (DFTD)	Transmissible tumor	Virus	The impending extinction of the Tasmanian devil from the recent appearance of this cancer is expected.	
Duck plague (duck virus enteritis)	Duck plague virus	Virus	Duck plague entered the United States in 1967; a major epizootic in 1973 struck the Lake Andes National Wildlife Refuge in South Dakota.	
E. coli (Colibacillosis)	*Escherichia coli* O157:H7	Bacterium	A multistate *E. coli* epizootic involving people at the start of 1993 focused attention on this emerging foodborne zoonotic disease.	
Eastern equine encephalitis (EEE)	*Alphavirus*	Virus	EEE is a zoonotic viral disease transmitted by mosquitoes to birds and other animals, including humans, that has been present in North America since at least the 1930s. Horses are more commonly infected than humans; in the United States, captive-reared pheasants and whooping cranes have died from infection.	
Ebola (Ebola hemorrhagic fever; EBOV)	Filovirus (five species of EBOV have emerged)	Virus	Ebola is a highly fatal zoonotic disease; 40–90 percent of infected humans die. Fruit bats are the likely reservoir species. Ebola is epizootic in nonhuman primates, which undermines conservation efforts for those species.	
Echinococcosis	*Enchinococcus multilocularis*	Tapeworm	Aveolar echinococcosis can follow accidental ingestion of the eggs of the parasite shed into the environment via the feces of infected red foxes and some other carnivores. The disease is often fatal in humans. Urban colonization by red foxes has increased potential human exposure during recent years (*see also* table 1).	
Epidemic typhus (flea-borne typhus)	*Rickettsia typhi*	Bacterium (Rickettsia)	Domestic rats are the most important reservoir of epidemic typhus, and they support a basic infection cycle of rat-to-flea-to-rat, with accidental transmission of the rat flea to humans.	
Epizootic hemorrhagic disease (EHD)	EHD viruses	Virus	*See* bluetongue.	
Giardiasis	*Giardia* spp.	Protozoan	*Giardia duodenalis* is the common species in American beaver.	
Hantavirus pulmonary syndrome (HPS)	Sin Nombre virus (in Spanish, the "nameless virus") and other hantaviruses present in the United States	Virus	HPS was first reported in North America in 1993; the deer mouse is primary reservoir for Sin Nombre virus. (*See also* table 5.)	

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Heartworm (canine)	*Dirofilaria immitis*	Nematode	Heartworm is primarily transmitted to wild carnivores by mosquitoes that have fed on infected domestic dogs.	
Hendra (Hendra virus infection)	Genus *Henipavirus* within the family of Paramyxoviridae	Virus	Hendra is a zoonotic disease first reported in 1994 in Australia as the cause of death in a horse trainer and horses. Fruit bats appear to be the source for infection.	
House finch conjunctivitis (mycoplasmal conjunctivitis; mycoplasmosis)	*Mycoplasma gallisepticum*	Bacterium	Infected house finches were first reported at a Washington, D.C.-area birdfeeder in 1994. By 2002 infection had spread across the entire house finch range in the United States.	
Influenza (*see also* avian influenza)	Orthomyxovirus	Virus	Influenza is the primary cause of pandemics affecting humans. "Genetic drift" and "gene swapping" between influenza viruses maintained in nature and with low ability to cause disease can result in highly infectious strains such as the "Spanish flu" of the early 1900s.	
Koala retrovirus	Lymphoid neoplasia (tumor)	Virus	Koala retrovirus is the most common form of tumor in koalas, and it affects both free-ranging and captive koalas. The disease has been found with leukemia in koalas.	
Lead poisoning	Particulate lead	Heavy metal	Spent lead ammunition, lost fishing line sinkers, and jig heads are the primary sources of wildlife exposure to toxic levels of lead.	
Leptospirosis	*Leptospira interrogans*	Bacterium	Skunks and raccoons are the common reservoir hosts of strains (serovars) of leptospirosis that infect humans.	
Lyme disease	*Borrelia burgdorferi*	Bacterium	Lyme disease is a tick-borne disease of humans and various animal species. The white-footed mouse is the primary animal host in the United States. White-tailed deer serve as an important producer of ticks involved in the disease transmission cycle.	
Measles	Measles virus	Virus	Human infection of mountain gorilla populations with measles has become another, recent challenge for the survival of the mountain gorilla.	
Monkeypox	Orthopoxvirus	Virus	Monkeypox first appeared in the United States during 2003 in association with the wildlife pet trade; humans, as well as wildlife, became infected.	
Mycoplasmal conjunctivitis	*Mycoplasma gallisepticum*	Bacterium	After the 1994 appearance of mycoplasmal conjunctivitis in house finches at an East coast birdfeeding station, the disease spread rapidly across the entire house finch range in the United States.	

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Myxomatosis	Myxoma virus	Virus	Myxomatosis is an insect-transmitted pox virus disease of wild and domesticated rabbits. The disease has been used for biological control of European rabbit populations.	
Newcastle disease (NDV)	Avian paramyxovirus type 1	Virus	A major epizootic of NDV, primarily affecting double-crested cormorants, began during 1990 in the Great Lakes region (*see also* table 1).	
Nipah	Paramyxovirus	Virus	Nipah is one of several recently emerging deadly zoonoses causing human mortality. Bats are its reservoir in nature (*see also* table 5).	
Orf (contagious ecthyma)	Virus of the genus *Parapoxvirus*	Virus	Orf is a common zoonotic disease that infects people who work with sheep, goats, and wild ruminants.	
Ornithosis (psittacosis)	*Chlamydophila psittaci*	Bacterium	Ornithosis is a zoonotic disease that has caused serious health problems in biologists exposed to waterfowl and other infected wild birds.	
Parapoxvirus infections	Viruses within the genus *Parapoxvirus*	Virus	Mammalian poxviruses within each genus are named for the species infected or the disease caused, such as contagious ecthyma virus, red squirrel pox virus, and squirrel fibroma virus.	
Parvovirus (CPV)	Canine parvovirus	Virus	CPV first appeared in dogs in Europe. CPV antibodies were detected in coyotes in United States during 1979 and in a Minnesota wolf population during 1975–77. CPV probably mutated from feline parvovirus or another closely related virus.	
Pesticide poisoning	DDT and its metabolites (primarily DDE)	Synthetic pesticide	The organic chemistry synthesis of DDT was a Nobel prize achievement. DDT saved countless human and animal lives from malaria and other arthropod-borne diseases, but unforeseen negative environmental impacts (primarily from DDE metabolite) resulted in a ban against the further use of DDT.	
Petroleum toxicosis	Crude oil	Environmental contaminant	Petroleum toxicosis is a major cause of periodic waterbird and marine mammal mortality.	
Phocine distemper	Morbillivirus	Virus	Epizootics of phocine distemper in Europe have killed thousands of harbor seals since the end of the 1980s.	
Plague	*Yersinia pestis*	Bacterium	See sylvatic plague.	

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Pneumonia complex	*Pasteurella* and *Mannheimia* sp.	Bacteria	Since the early 1980s, bacterial pneumonia complex has caused repeated heavy losses of bighorn sheep in Montana and other western states. The disease is a serious threat for conservation of bighorn sheep.	
Rabbit hemorrhagic disease (RHD)	**Calicivirus** of the genus Lagovirus	Virus	RHD was first described in China in 1984, and it spread rapidly to many other countries. The disease was purposely introduced into Australia and New Zealand as a biological control of populations of imported European rabbits.	
Rabies	Rhabdovirus	Virus	An epizootic of raccoon rabies in the mid-Atlantic states during the late 1970s spread throughout the Northeastern United States by the mid-1990s.	
Salmonellosis	*Salmonella typhimurium*	Bacterium	Salmonellosis is a common disease of songbirds that causes occasional large-scale epizootics at birdfeeding stations.	
Sarcoptic mange	*Sarcoptes scabiei*	Ectoparasite	Sarcoptic mange is a common disease of red fox (*see also* table 1).	
Sea otter encephalitis	*Toxoplasma gondii* and *Sarcocystis neurona*	Protozoan	Nearly 40 percent of the 323 southern sea otters necropsied at the National Wildlife Health Center between 1992–2002 died from infectious disease. Of those, approximately 20 percent died from infection by toxoplasma or sarcocystis, neither of which had previously been found to infect populations of sea otters.	
Severe acute respiratory syndrome (SARS)	Coronavirus (CoV)	Virus	SARS is one of several recent (2003) emerging deadly zoonotic diseases for humans. Its origin has been traced to bats (*see also* table 5).	
Streptothricosis	*Dermatophilus* spp.	Fungus	Streptothricosis is a rare disease in the United States. It was first reported in New York state in 1960 from a white-tailed deer bagged during the hunting season and subsequently in 4 of 10 people who handled that deer.	
Sylvatic plague (*http://pubs.usgs.gov/circ/1372/*)	*Yersinia pestis*	Bacterium	Sylvatic plague, a zoonotic disease, is a major challenge for the survival of the endangered black-footed ferret.	
Toxoplasmosis (*http://pubs.usgs.gov/circ/1389/*)	*Toxoplasma gondii*	Protozoan	Bobcats and other wild or domestic felines are the source of toxoplasmosis eggs (oocysts) that infect humans and other species (*see also* table 1).	
Trichomoniasis	*Trichomonas gallinae*	Protozoan	Pigeons and doves are frequently infected with the protozoan that causes trichomoniasis, often at birdfeeding stations.	

Appendix 1. Diseases Cited and Their Causative Pathogens

Developed from Friend (2006a, 2006b); Friend and Franson (1999); Davis and others (1970); Davis and others (1971); Abbott and Rocke (2012); Hill and Dubey (2014); Constantine (2009). The first use of a word defined in the glossary is shown in **bold** type (not including words in table headings).

Disease	Pathogen	Pathogen type	Comments	Text page citation
Tuberculosis (*see also M. bovis* and *M. avium*)	*Mycobacterium tuberculosis*	Bacterium	Mycobacterium tuberculosis is the main cause of tuberculosis in humans. During recent years, it is increasingly being detected in free-ranging wildlife.	
Tularemia (*http://www.nwhc.usgs.gov/publications/tularemia/*)	*Francisella tularensis*	Bacterium	Tularemia is a tick-borne zoonotic disease of rabbits and a water-borne disease of muskrat and American beaver. For humans, the water-borne disease is less virulent that the tick-borne disease.	
West Nile fever	West Nile virus (WNV)	Virus	West Nile fever is caused by a mosquito-transmitted virus that was first found in the United States in 1999. West Nile fever now extends across the nation and kills large number of birds annually (*see also* table 5).	
White-nose syndrome	*Geomyces destructans*	Fungus	White-nose syndrome was first described in 2007 in little brown bats within New York State. Since then, millions of bats have died as infection continues to spread widely within the United States and into Canada.	

Appendix 2. Common and Scientific Names for Species Cited

Common name	Scientific name
American alligator	*Alligator mississippiensis*
American beaver	*Castor canadensis*
American bison	*Bison bison*
American black duck	*Anas rubripes*
American robin	*Turdus migratorius*
American white pelican	*Pelecanus erythrorhynchos*
American woodcock	*Scolopax minor*
Bald eagle	*Haliaeetus leucocephalus*
Banded mongoose	*Mungos mungo*
Big brown bat	*Eptesicus fuscus*
Bighorn sheep	*Ovis canadensis*
Black-faced honeycreeper	*Melamprosops phaeosoma*
Black-footed ferret	*Mustela nigripes*
Bobcat	*Lynx rufus*
Brown pelican	*Pelecanus occidentalis*
Brushtail possum	*Trichosaurus vulpecula*
California brown pelican	*Pelecanus occidentalis*
California condor	*Gymnogyps californianus*
California ground squirrel	*Otospermophilus beecheyi*
California sea otter	*Enhydra lutris nereis*
Canada goose	*Branta canadensis*
Chamois	*Rupicapra rupicapra*
Chimpanzee	*Pan troglodytes*
Chukar partridge	*Alectoris chukar*
Civet cats	*Viverridae spp.*
Common loon	*Gavia immer*
Cooper's hawk	*Accipiter cooperii*
Cormorants	*Phalacrocorax* spp.
Coyote	*Canis latrans*
Crows	*Corvus* spp.
Deer	*Odocoileus* spp.
Deer mouse	*Peromyscys species,* including *P. maniculatus*
Desert tortoise	*Gopherus agassizii* (formerly *Scaptochelys agassizii)*
Eastern chipmunk	*Tamias striatus*
Elk	*Cervus elaphus*
Ethiopian wolf	*Canis simensis*
European badger	*Meles meles*
European greenfinch	*Carduelis chloris*
European rabbit	*Oryctolagus cuniculus*
Eurpoean red squirrel	*Sciurus vulgaris*

Appendix 2. Common and Scientific Names for Species Cited

Common name	Scientific name
European water vole	*Arvicola terrestris*
Flying fox	*Pteropus* spp.
Golden toad	*Bufo periglenes*
Gray squirrel	*Sciurus carolinensis*
Greater sage grouse	*Centrocercus urophasianus*
Green-winged teal	*Anas crecca*
Harbor seal	*Phoca vitulina*
Hawaiian crow	*Corvus hawaiiensis*
House finch	*Carpodacus mexicanus*
Iberian lynx	*Lynx pardinus*
Ibex	*Capra ibex*
Koala	*Phascolarctos cinereus*
Little brown bat	*Myotis lucifugus*
Mallard (mallard duck)	*Anas platyrhynchos*
Mountain gorilla (gorilla)	*Gorilla gorilla*
Mountain lion	*Puma concolor*
Mourning dove	*Zenaida macroura*
Mule deer	*Odocoileus hemionus*
Northern pike	*Esox lucius*
Pelican	*Pelecanus* spp.
Peregrine falcon	*Falco peregrinus*
Pronghorn (pronghorn antelope)	*Antilocapra americana*
Puma	*Puma concolor*
Quagga mussel	*Dreissena bugensis*
Raccoon	*Procyon lotor*
Red fox	*Vulpes vulpes*
Reindeer	*Rangifer tarandus*
Ring-necked pheasant	*Phasianus colchicus*
Round goby	*Neogobius melanostomus*
Salt marsh harvest mouse	*Reithrodontomys raviventris*
Sandhill crane	*Grus canadensis*
Sea otter	*Enhydra lutris*
Sharp-shinned hawk	*Accipiter striatus*
Snowshoe hare (varying hare)	*Lepus americanus*
Southern sea otter	*Enhydra lutris nereis*
Striped skunk	*Mephitis mephitis*
Suricates	*Suricata suricatta*
Tasmanian devil	*Sarcophilus harvisii*
Tilapia	*Oreochromis spp.* (formerly *Tilapia* spp.)
Tundra swan	*Cygnus columbianus*

Appendix 2. Common and Scientific Names for Species Cited

Common name	Scientific name
Vole	Family *Muridae*
White Pekin duck	*Anas platyrhynchos*
White-faced ibis	*Plegadis chihi*
White-tailed deer	*Odocoileus virginianus*
Whooping crane	*Grus americana*
Wild turkey	*Meleagris gallopavo*
Wolf	*Canis lupus*
Wyoming toad	*Bufo baxteri*
Yosemite toad	*Bufo canorus*
Zebra mussel	*Dreissena polymorpha*

Index

For more information concerning this report, contact:

Director
U.S. Geological Survey
National Wildlife Health Center
6006 Schroeder Road
Madison, WI 53711-6223
jsleeman@usgs.gov

or visit our Web site at:
http://www.nwhc.usgs.gov/

ISSN 2330–5703 (online)
http://dx.doi.org/10.3133/cir1401