Chapter 2

# Disease Emergence and Resurgence

"Ingenuity, knowledge, and organization alter but cannot cancel humanity's vulnerability to invasion by parasitic forms of life. Infectious disease which antedated the emergence of humankind will last as long as humanity itself, and will surely remain, as it has been hitherto, one of the fundamental parameters and determinants of human history." (McNeill)[1]

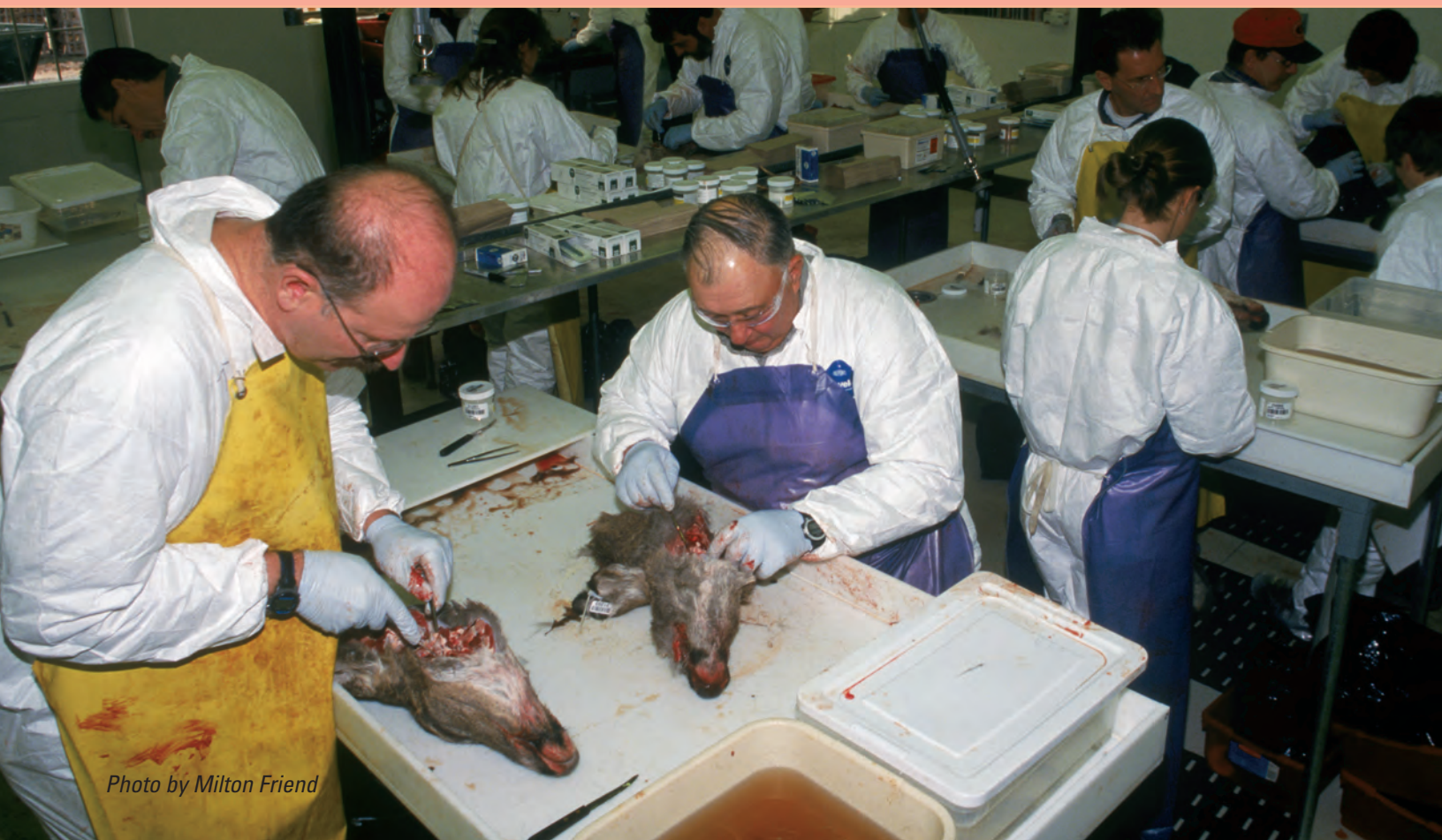


*Photo by Milton Friend*

# Contents



**Bolded words** within the text indicate terms that are defined in the Glossary.

Chapter 2

# Disease Emergence and Resurgence

A profusion of emerging diseases has affected humans since the early 1980s, and pathogens of animal origin or products of animal origin cause many of these.[2] Some of these diseases had not been established previously, such as AIDS, and others are a resurgence of diseases thought to have been controlled, such as tuberculosis in developed nations. This change in the status of diseases affecting humans has resulted in emerging infections becoming a focus for national and global attention (Box 2–1).

Emerging and reemerging diseases have generally been defined as infectious diseases of humans whose occurrence during the past two decades has substantially increased or threatens to increase in the near future relative to populations affected, geographic distribution, or magnitude of impacts.[3–5] This concept has been expanded to also include other species and noninfectious diseases.[6–8] Disease emergence and reemergence are affecting a wide variety of species on a global scale. An overview of the scope of this problem is provided to increase awareness of the role of wildlife in the ecology of emerging/reemerging diseases and to explore some of the primary factors involved.

## Concepts

### *What is Disease?*

For general purposes, disease is broadly defined as any departure from health[9] resulting in bodily dysfunctions. Impairments to health caused by conditions such as arthritis, major depression, reproductive sterility, dementia, and Parkinson's disease are common, in addition to clinical illness from infections, such as influenza and death due to cancers and cardiac failure. For wildlife, disease primarily impairs populations by reducing offspring (e.g., brucellosis) or by reducing the probability for survival of individuals (e.g., plague). If enough individuals are affected, the collective effects can reduce the sustainability of the population.[8] Recent appearances of chytridiomycosis in **amphibian** populations have raised great concern about the sustainability of affected populations, especially those already in threatened and endangered status.[10–12]

### *Disease Agents*

Disease can result from exposure to a variety of infectious agents and also can be an outcome from other factors (Table 2.1). Infectious disease has been the human health focus for disease emergence and reemergence and is the primary orientation here. That is, the focus is on organisms that invade live hosts in a manner that generally involves multiplication of the organism within the host as a prerequisite for an outcome of disease. Noninfectious diseases such as botulism, other diseases involving natural (i.e., algal, fungal, etc.) and synthetic toxins (i.e., pesticides) that may be acquired from the consumption of food items and allergic responses are, in general, not addressed here. However, diseases caused by biotoxins (natural toxins) are noted for circumstances of special concern.

For wildlife, noninfectious diseases of microbiological origin have been a prominent component of disease emergence and reemergence. For example, type C **avian** botulism, *Clostridium botulinum*, is a "food poisoning" of wild birds and occasionally some other species (humans are resistant to type C toxin but not to most other types of botulinum toxin). This disease has evolved from being a problem in Western North America to becoming the greatest known cause of disease affecting free-ranging **waterbirds** throughout the world. Avian botulism was essentially limited to areas west of the Mississippi River prior to 1940 (Fig. 2.1),[13] and prior to 1960, had very limited occurrence outside of North America (Fig. 2.2). In addition to greatly expanding its geographic distribution within the USA since the mid-1970s (Fig. 2.1), unique epizootics have appeared since the mid-1990s at the Salton Sea in southern California. The type C botulism outbreaks in white **pelicans** and brown pelicans at the Salton Sea are the largest die-offs of pelicans ever reported from any cause and the first to be associated with fish.[14]

The occurrence of epizootics of type E botulism during 2000 and 2002 in Lake Erie of the North American Great Lakes system also is of significance because humans are highly susceptible to type E toxin.[15] An estimated 8,000 birds died during the summer outbreak of 2000, and more than 25,000 during the 2002 epizootic. Ring-billed **gull**, red-breasted merganser, common loon, and long-tailed **duck** were the primary species affected.[16] These epizootics are the largest mortalities recorded for birds due to type E botulism. The first wild **bird** epizootic from this toxin was in 1963 and involved extensive mortality of gulls and common loons in Lake Michigan, another of the Great Lakes, where periodic outbreaks have persisted.[17,18]

Little is known about the ecology of type E botulism in nature. Fish are susceptible to type E toxin and they may be the source of the toxin killing some fish-eating birds. Human cases of type E botulism acquired from commercial smoked fish from the Great Lakes resulted in changes in regulations governing the commercial preparation of smoked fish.[19,20] However, recreational fishermen sometimes smoke the fish

# Box 2–1 Infectious Disease: A Continuum of New Challenges and Opportunities

***"Infectious disease is one of the few genuine adventures left in the world" (Zinsser).[291]***


Time has vividly etched how infectious disease has influenced human life as evidenced by cultural mores, religious beliefs, the demography of peoples, the outcomes of wars and colonization attempts, economic status, and life-expectancy.[1,289–291,337] Thus, there is a foundation of self-interest involving personal health and economic well being in the current resurgence by the developed nations of the world to increase efforts for addressing infectious disease after decades of neglect. In addition, bioterrorism has become an increasing concern due to world change initiated by the infamous events of September 11, 2001, and by the anthrax-contaminated letters sent in the months that followed. As a result, previous reductions in resources for infectious disease programs are being restored[28] and enhanced.

*Photo courtesy of the Centers for Disease Control*

**People grieve by the NAMES Project AIDS Memorial Quilt**

Our globally interactive society results in the need for a global perspective in combating infectious disease because:

> "It is not possible to adequately protect the health of our nation without addressing infectious disease problems that occur elsewhere in the world…. Left unchecked, today's emerging disease can become the endemic disease of tomorrow" (CDC).[255]

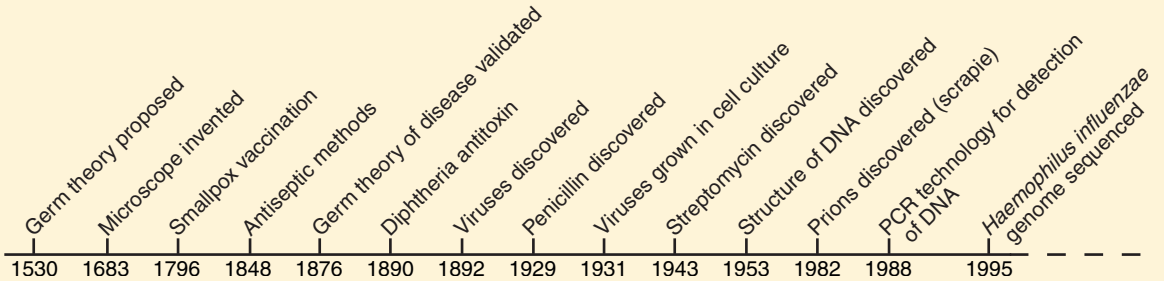
International collaboration, as in the 2003 onset of severe acute respiratory syndrome (SARS),[338] is essential for minimizing the potential impacts from emerging disease because pathogens are often hidden hitchhikers associated with commerce and human travel. Proactive rather than reactive efforts are needed to meet the challenges posed by:

> "…those ferocious little fellow creatures, which lurk in the dark corners and stalk us in the bodies of rats, **mice**, and all kinds of domestic animals [and wildlife]; which fly and crawl with the insects, and waylay us in our food and drink and even in our love" (Zinsser).[291]

These challenges are eternal because of the great adaptive capabilities of "those ferocious little fellow creatures"[22] and new opportunities continually provided to them by the periodic folly of human behavior and actions.

A recent editorial spoof, "New World Pathogen Strategy Disclosed,"[339] exploits pathogen adaptability and human frailties by taking the reader into a mythical convention of pathogens in which they are discussing the topic "Our Infective Future: The New Agenda." The keynote speaker, a contemporary prion, in addition to giving plaudits to HIV, the tuberculosis bacillus, and to the viruses Ebola, Hanta, Lassa, and Marburg for gains they have made, notes that:

> "*Homo [sapien]* is remarkably hospitable to us… And they are recklessly changing the climate, releasing many of us from our historical geographic costraints… Although they themselves deny that there is such a thing as a free lunch, we know better. There is a free lunch, and it is them."




**Disease and Discovery**

(adapted from Lederberg)[22]

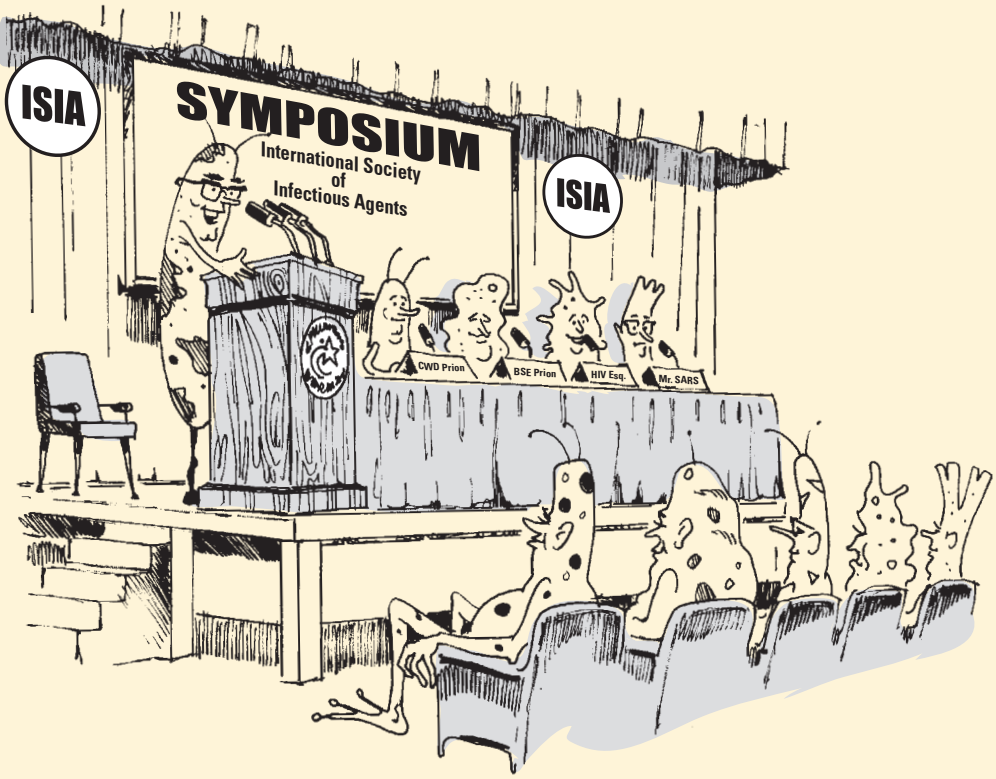



*Illustration by John M. Evans*

Human actions have always created new opportunities for pathogens, while disease emergence continues to provide new opportunities for humans to gain a better understanding of the ecological, behavioral, and social conditions that result in disease. The need to aggressively apply this knowledge is facilitated by a more informed public and collaboration among agencies, governments, and scientists that spans disciplines and political boundaries.

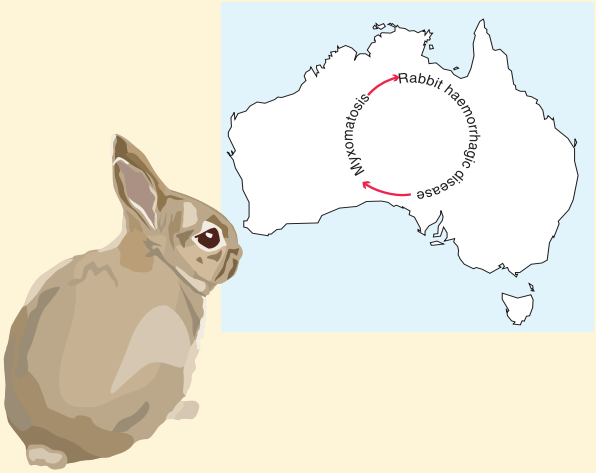
Numerous scientific conferences, workshops, other regional, national, and international meetings, and other actions are focusing on emerging infectious disease. Notable actions include:

- 1995—The Centers for Disease Control and Prevention (CDC) of the United States Department of Health and Human Services, Public Health Service initiates publication of "Emerging Infectious Diseases," a scientific journal for tracking and analyzing disease trends.
- 1996—The White House issues a "Presidential Decision Directive on Emerging Infectious Diseases," which establishes a national policy and implementation actions to address the threat of emerging infectious disease by improving domestic and international surveillance, prevention, and response measures.[340]
- 1998—CDC issues an emerging disease strategy document, "Preventing Emerging Infectious Diseases: A Strategy for the 21st Century."[341]
- 2002—CDC issues a mission-oriented document addressing approaches for the improvement of global capacity for disease surveillance and outbreak response, "Protecting the Nation's Health in an Era of Globalization: CDC's Global Infectious Disease Strategy."[342]
- Numerous global health Web sites focusing on emerging diseases are developed as well as numerous regional and disease-specific surveillance networks (see CDC Appendices A and E).[342]

The actions noted are but the tip of the proverbial iceberg of current response to emerging infectious disease. Major investments are also being made in the development of high level biosecurity facilities where studies can be carried out on the most hazardous pathogens. Advances being made have been greatly assisted by the reporting from all aspects of the news media including newspapers and weekly news magazines to television news shows and documentaries, and by the Hollywood spotlight that has made millions of people aware of emerging infections [e.g., "The Philadelphia Story" (AIDS) and "Outbreak" (Ebola)]. Therefore, it is fitting that the author of the book, "The Coming Plague: Newly Emerging Diseases in a World Out of Balance"[343] earned a Pulitzer Prize for journalism.

### Australia, Myxoma Virus, and the European Rabbit

Myxomatosis in Australia is a classic example of host-pathogen adaptation for the benefit of both parties.[26] Highly virulent strains of myxoma virus were introduced into European rabbit populations in an attempt to rid Australia of this introduced species. The biological control program depended upon mosquitoes to vector the disease and spread it among the rabbit population. After several failed introduction attempts, the virus established an epizootic foothold, causing a case-fatality rate of over 99 percent during the summer of 1952.[27] However, instead of dying out during the winter as expected, some virus survived and established enzootic foci for the disease. Those foci produced mutant strains of virus with reduced virulence, as evidenced by longer survival times for infected rabbits (weeks rather than days). Because of the longer survival times, the probability for mosquitoes to acquire and transmit the attenuated virus strains was far greater than that for the highly virulent strains. Within 3 years, this selective process resulted in the attenuated mutant viruses (70-percent to 90-percent case-fatality rate) becoming the predominant strains of myxoma virus in Australia.[27,28]




Drawing by Erinn Dornaus

In addition to virus mutations, genetic selection for survival was also occurring within the rabbit population. Within 7 years, the susceptibility of the rabbits to the original virulent virus when tested in the laboratory had fallen from 90 percent to 25 percent. Thus, attenuation of the myxoma virus and genetic selection for resistance to myxoma virus by the rabbit population has resulted in host-pathogen adaptation that prevents the virus from killing all of the hosts that sustain its presence. While some rabbits still die in Australia from myxomatosis, many survive infection to produce young. The European rabbit remains a pest species in Australia, but populations are at levels considerably reduced from those that existed prior to the establishment of myxomatosis.[26–29]

A biological postscript is currently being written to the myxomatosis story. Rabbit hemorrhagic disease virus escaped from experimental studies on an offshore island, reached mainland Australia in October 1995, and rapidly spread. Rabbit hemorrhagic disease (RHD) has decreased long-term average numbers of rabbits by 85 percent in some arid areas. In the coastal areas, the numbers of rabbits were reduced by 73 percent in the first year, but gradually recovered to only 12 percent below pre-RHD numbers in the third year.[30] As for myxomatosis, biological adjustments are occurring, although, now as a three-party interaction of RHD, myxomatosis, and rabbits.

The appearance of RHD has changed seasonal patterns of rabbit recruitment, rabbit abundance, and myxomatosis activity. RHD generally has a severe impact on rabbit populations through the breeding season, but compensatory recruitment after RHD activity declines allows rabbit numbers to recover somewhat. Because of the loss of susceptible rabbit hosts, the seasonal peak in myxomatosis activity is slightly delayed. RHD is outcompeting myxomatosis because it kills most rabbits (2 days for viremia) before they become infective for myxomatosis (8–10 days for viremia).[30] The final outcome from this competition remains to be learned.

they catch. Public education has been helpful in informing the public of proper temperature and time required at that temperature to destroy toxins that may be present.

It is likely that the recent bird mortalities on Lake Erie are an indicator of environmental changes that are resulting in increased levels of type E botulinum toxin within the food chain of this lake, creating potentially severe ramifications for human and wildlife health. Similar to type C botulism in pelicans at the Salton Sea, exotic species also appear to be a major factor in toxin production within the food chain of Lake Erie. Tilapia, an introduced fish species, is a primary source for toxin production at the Salton Sea. Other introduced species, such as the round goby fish, zebra mussel, and quagga mussel, are believed to be involved in toxin production or transport within Lake Erie.[21]

#### *Evolutionary Considerations*

In considering disease emergence and reemergence, one must recognize that disease is an outcome, not a cause; an outcome that can be viewed as a state of instability among coinhabitants of Earth due primarily to two associated instabilities, one of which is ecological and the other evolutionary.[22] The dynamic nature of these factors challenge the common belief that, "Given enough time, a state of peaceful coexistence eventually becomes established between any host and parasite."[23] Some notable evolutionary biologists chal-

lenge the concept of benign coexistence between parasites (including microbes) and their hosts as being at odds with the fundamental principles of evolution on which they are based.[24] Nevertheless, the long-term trend towards coadaptation between hosts and pathogens is to the advantage of both because very severe impacts may result in the elimination of both species.[25]

Coadaptation does not necessarily equate to benign coexistence. Mortality of the host may be replaced by disease that has less severe outcomes as pathogens mitigate their **virulence** in ways that do not compromise their continued existence, but may still negatively impact their hosts.

Infectious pathogens have great capability to make adjustments that provide them with suitable hosts (e.g., cross species barriers) and to sustain their invasiveness and spread (e.g., antibiotic resistance). Their superiority in numbers, species, and capability for genetic change allow pathogens to adapt to changing environmental and host conditions at a pace greater than humans can counteract in the short term.[25,31] The continual need to develop vaccines against the latest strain of influenza virus and the growing problem of resistance to antibiotics long used to successfully combat serious human illness are familiar examples of the ability of microbes to make adaptive changes that sustain their infectivity for humans despite our technological capabilities.

**Table 2.1.** Some of the many sources of human disease.

| **Infectious agents** | **Zoonotic examples** |
|---|---|
| Viruses | Rabies, West Nile fever |
| Bacteria | Tuberculosis, Lyme disease |
| Rickettsia | Rocky Mountain spotted fever, Q fever |
| Fungi | Coccidioidomycosis (valley fever), histoplasmosis |
| Metazoan parasites | Echinococcosis (hydatid disease), trichinosis |
| Protozoan parasites | Toxoplasmosis, giardiasis |
| Prions | Bovine spongiform encephalopathy (BSE) |
| **Noninfectious agents** | **Disease examples** |
| Microbial toxins | Botulism, enterotoxemia |
| Algal toxins | Domoic acid poisoning, saxitoxin (contaminated shellfish) |
| Plant toxins | Aflatoxicosis (contaminated peanuts), mushroom poisoning (*Amanita* spp.) |
| Synthetic chemicals | Pesticide poisoning, drugs |
| Heavy metals | Lead poisoning, mercury poisoning |
| Oil spills | Skin irritation from contact; liver disease from inhalation |
| **Other causes** | **Examples** |
| Neoplasia | Cancers |
| Genetic disorders | Down's syndrome, hemophilia |
| Diseases of immunity | Autoimmune disease, Chédiak-Higashi syndrome |
| Systemic diseases | Diabetes, gout |
| Deficiency diseases | Malnutrition, vitamin deficiencies |
| Psychoses | Depression, post-traumatic stress syndrome |
| Physiological disorders | Endocrine disruption, hypothermia |
| Trauma | Blunt impacts, gunshot |

### *The Human Influence*

The instability of ecological conditions encountered by pathogens is primarily a result of human actions that alter the physical and biological environment, the microbial and animal tenants (humans included) of these environments, and human interactions (including hygienic and therapeutic interventions) with pathogens.[22] The magnitude of human impacts contributing to this ecological instability is such, "…that humans may be the world's dominant evolutionary force."[32] Landscape disruption alone grossly reflects the magnitude of environmental change. About 40 to 50 percent of land on the Earth has been irreversibly transformed or degraded by human actions. An additional one-third of global land cover will be transformed over the next century.[33] Changes in biotic diversity and alterations in the structure and function of ecosystems are the two most dramatic ecological trends of the past century.[34] Disease emergence and reemergence should be expected as continuing outcomes from this accelerated magnitude of ecological instability and associated changes in species abundance, presence, and interactions.

## Perspectives

**"Most of the infectious diseases…have now yielded up their secrets…. Many illnesses…had been completely exterminated; others had [been brought] largely under control…." (Sigerist, 1931, cited by Cohen)[35]**

### *The Mirage of Health*

Human experiences with infectious disease have stimulated pursuit of a world free from the debilitation, suffering, and death that disease causes. Economic and other costs of disease stimulate this utopian vision, in addition to impacts on the personal health of individuals, families, and populations. Notable accomplishments in this quest during the 20th century include the global **eradication** of smallpox and major advances in the elimination of polio in much of the world.[36] Infectious disease mortality within the USA declined from 797 deaths per 100,000 individuals in 1900 to 36 deaths per 100,000 in 1980.[37] These and other accomplishments have resulted in overoptimistic perspectives regarding human dominance over infectious pathogens (Box 2–2).

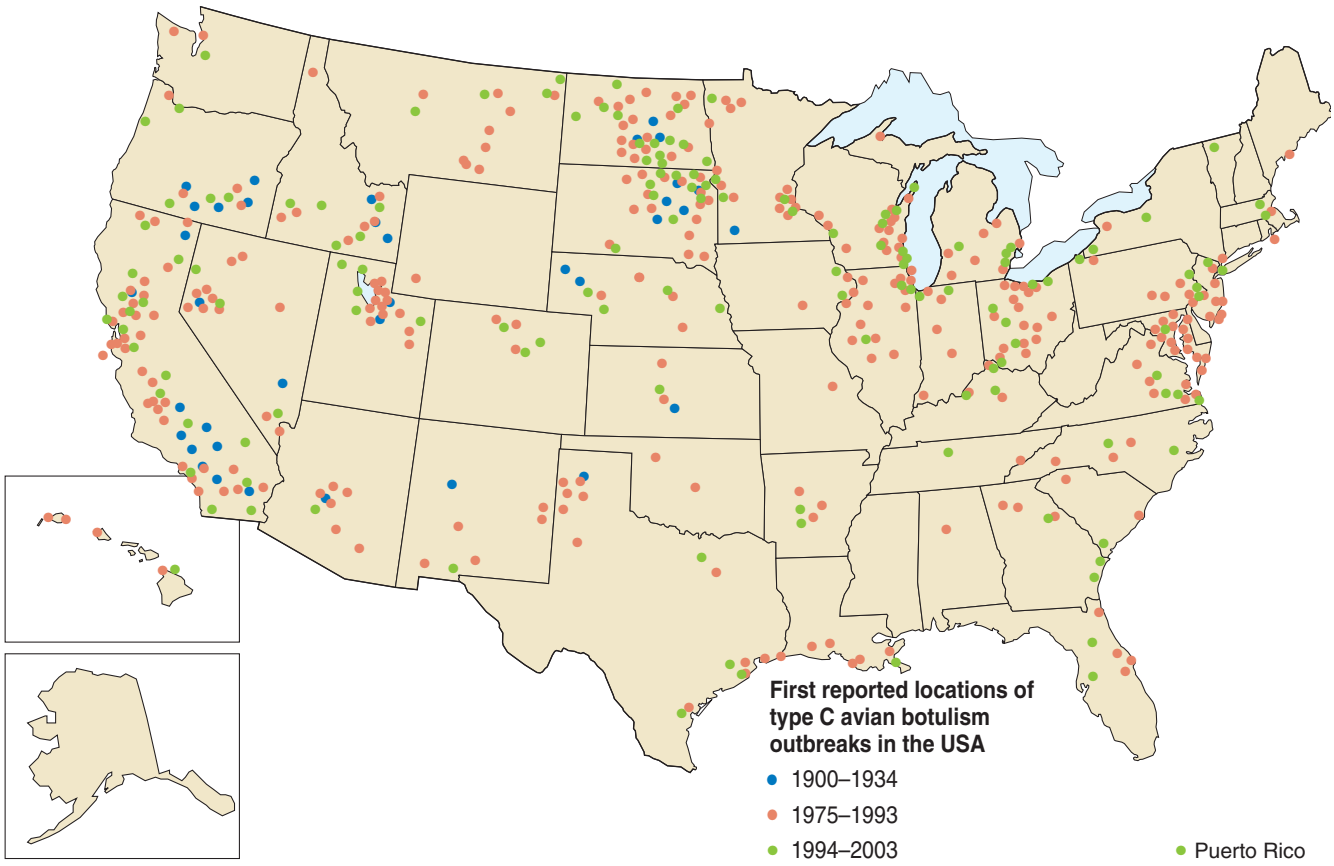



**Figure 2.1** Locations of type C avian botulism outbreaks in the United States.

The euphoria associated with accomplishments in the conquest of infectious disease is based on reflections from a mirage rather than images of lasting substance. Those perspectives and the prediction that the history of human infection will progress steadily toward virtual elimination of infectious disease were replaced during the 1980s by a resurgence of human infections occurring on a global scale.[3,38–40] Instead of infectious disease being conquered by the end of the 20th century, the last two decades of that century initiated the start of an era of emerging and reemerging diseases of humans that, once again, reflects humanity's vulnerability to invasion by parasitic forms of life.[1] As we enter the third millennium, microbial diseases remain as the most frequent cause of human mortality worldwide.[41]

### *The Process of Living*

**"Complete freedom from disease and from struggle is almost incompatible with the process of living" (Dubos).[42]**

Human impacts result in a continuum of environmental changes, ecological disturbances, and adjustments by microbes and parasites to survive and flourish as part of these changing conditions. The current lesson being relearned from history, as evidenced by the more than 30 diseases of humans that have emerged during the past quarter century, is that environmental change leads to the continual emergence of infectious disease.[7,35,43,44] Society is not only subject to diseases of antiquity, such as rabies and tuberculosis, but we have also facilitated the establishment of a host of new diseases, including some such as Legionnaires' disease and toxic shock syndrome that are products of technological advances.

### *The Devil's Cauldron*

It is increasingly evident that the human pursuit of the "good life" is tainting the elixir of life with a potpourri of ingredients that enhance disease emergence. In some respects it seems as if that elixir is being brewed in the "devil's cauldron" and that its consumption is a major factor leading to human disease. The number of infectious agents causing disease in humans is increasing and substantial, but difficult to quantify because of differences in the way disease agents are categorized and enumerated. For example, bacteria of

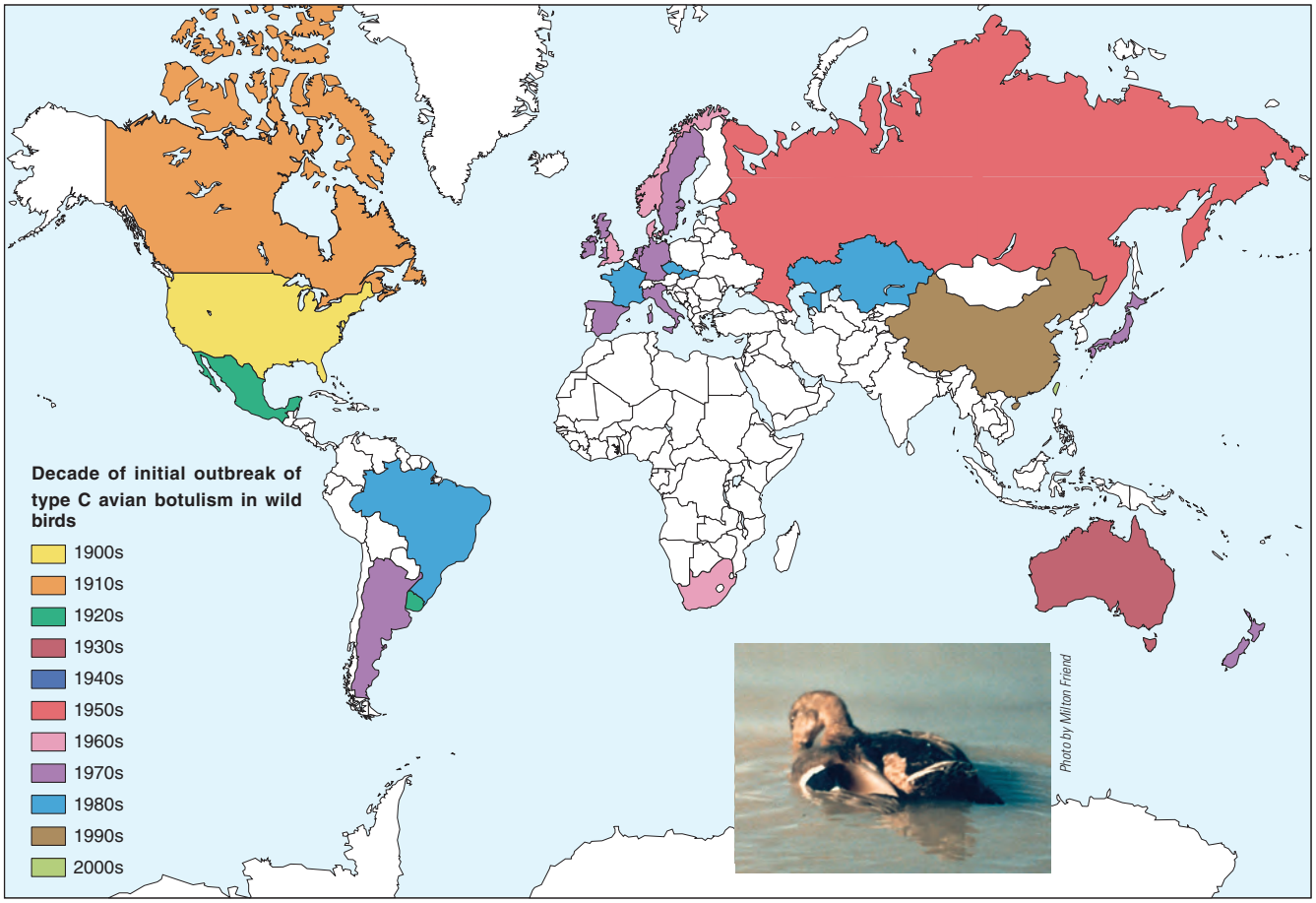



**Figure 2.2** Countries where type C avian botulism has occurred in wild birds (through 2003).

# Box 2–2

## Humans and Disease: From Despair to Optimism and a Return to Reality


Illustration by John M. Evans

The historic impacts of infectious disease on human society are incomprehensible for most individuals living in the developed nations of the world. Epidemics of early times reflect a world that illustrates the German term Durchsevchung, which means thorough saturation of a population with infection.[291] About 25 percent of the entire population of Europe was destroyed by the first waves of plague (*Yersinia pestis*) that swept through that continent. It is estimated that nearly 70 percent of the population was affected by the epidemic that began in 1348, with most infected individuals dying. Very few of those infected by another epidemic in 1361 survived, but only about 50 percent of the population was affected. In the Americas, one infected individual who came ashore from the ship of an expedition introduced smallpox into native populations, resulting in a death toll of over 3 million. Epidemics from diseases such as plague, smallpox, measles, typhoid fever, and typhus were common events associated with global developments of earlier times and took a high toll on human life throughout the world.[289]

Modern medicine and its associated technology, along with greater understanding of the ecology of infectious disease, has helped to combat many of the diseases that have had the greatest impacts on human health. Impressive accomplishments in reducing human cases of deadly and debilitating infectious diseases created hope and optimism that became translated into optimistic public statements by notable individuals. Especially noteworthy is the statement by medical historian Henry Sigerist[35] (p. 26) and those below by Nobel laureate Dr. Frank MacFarlane Burnet, and Dr. William Stewart, former U.S. Surgeon General of the USA.

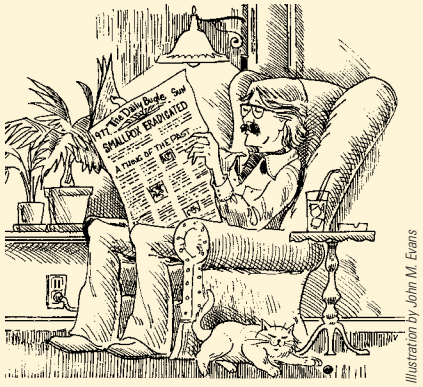



Illustration by John M. Evans

*Time period*

***1 AD***
- Plague
- Smallpox

***1000***
- Typhus

***1500***
- Yellow fever
- Measles

***1700***
- Yellow fever
- Flu

***1800***
- Cholera
- Smallpox

***1900***
- Plague
- Flu

***1925***
- Typhus

***1950***
- Dengue fever

***1975 to today***
- Lyme disease
- Legionnaire's disease
- HIV
- Ebola
- "Mad Cow"
- West Nile virus
- SARS

> "One can think of the middle of the 20th century as the end of one of the most important social revolutions in history, the virtual elimination of the infectious disease as a significant factor in social life."(Burnet)[344]
>
> "…it is time to 'close the book on infectious diseases.'" (Stewart)[345]
>
> "During the last 150 years the Western world has virtually eliminated death due to infectious disease."(Stewart, 1975, cited by Cairns)[346]

Similar statements were made by numerous other learned individuals of those times. These statements reflect general beliefs at the time they were made and a growing need to address a variety of disease conditions that, for the most part, do not involve infectious pathogens (e.g., heart disease and most cancers).

The resulting redirection from infectious disease to other human health issues has caused us to relearn two important lessons of history noted by Zinsser:[291]

- "Infectious disease is one of the greatest 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."

Consider for example that World Health Organization figures for 1990 indicate human annual deaths from acute respiratory infections of 4.3 million, diarrheal diseases due to bacterial or viral infections of 3.2 million, 3 million deaths from tuberculosis, and millions of additional deaths from a variety of other infectious diseases.

- "Swords and lances, arrows, machine guns, and even high explosives have had far less power over the fates of the nations than the typhus louse, the plague flea, and the yellow-fever mosquito."

During the Spanish-American War, typhoid fever was a major factor contributing to death from infection causing seven times the number of fatalities as battle wounds.[347] During the Civil War of the USA, infectious diseases, such as typhoid fever, malaria, smallpox, and diseases of dysentery and diarrhea killed three times as many soldiers as died from battle wounds.[289] Infectious disease has remained a formidable enemy in times of war and peace.

If these thoughts are viewed as simply reflections of the past, one should consider the reality of AIDS on the African continent; the 50–100 million annual cases of dengue fever, many of which occur in the Americas; preparation being undertaken to protect humans against the potential reappearance of smallpox; and the 2003 pandemic of severe acute respiratory syndrome (SARS)[338] that exemplifies:

***"Mother Nature is by far the worst bioterrorist out there." (Marjorie Pollack)[338]***

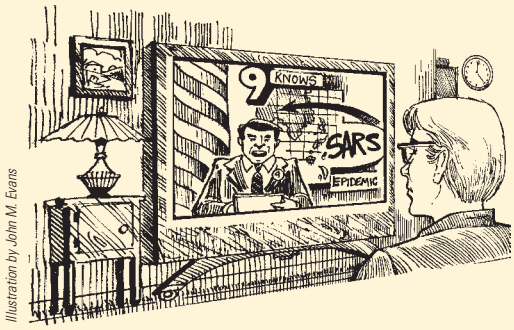



Illustration by John M. Evans


**Mortalities from the 10 most infectious global diseases (Johns Hopkins University).**

the genus *Salmonella* cause the disease salmonellosis. That genus contains more than 2,300 variants (serotypes) of *Salmonella* spp. and each **serotype** causing disease could be individually enumerated. Alternatively, salmonellosis might be considered a single disease, or different forms of the disease might each be enumerated separately. Therefore, one evaluation places the number of zoonoses among infectious diseases to be between 100 to 3,000 depending on the methods for enumeration. The lower figure is about 59 percent of the diseases listed in a particular book on communicable diseases in humans.[45] Another evaluation identifies 1,415 species of infectious agents as having been reported as causes of disease in humans. Of those, 61 percent are known to be zoonotic.[46]

On a percentage basis, diseases of bacterial or **rickettsial** origin are the predominant types of infectious diseases. Zoonoses are most commonly caused by helminthes (parasitic worms) and bacterial or rickettsial disease agents. However, helminthes are by far the group of pathogens most associated with zoonoses. About 95 percent of helminth species pathogenic to humans are known to be zoonotic compared with 50 percent of bacteria and rickettsia (Fig. 2.3).

### *Zoonoses and Disease Emergence*

Zoonoses are a prominent aspect of disease emergence. Over the past decade more than two-thirds of emerging diseases have animal origins,[47] an outcome that results in emerging zoonotic diseases being among the most important public health threats today.[48] Human exposure to zoonoses is not restricted to direct interface between humans and nature (Table 2.2). The emergence of numerous foodborne diseases is particularly noteworthy. Increased globalization of food supplies and "novel dining experiences" associated with human travel are presenting new opportunities for pathogens to encounter naive hosts. The associated costs to society from emerging diseases go beyond illness and death by altering our way of life and causing major economic burdens (Box 2–3).

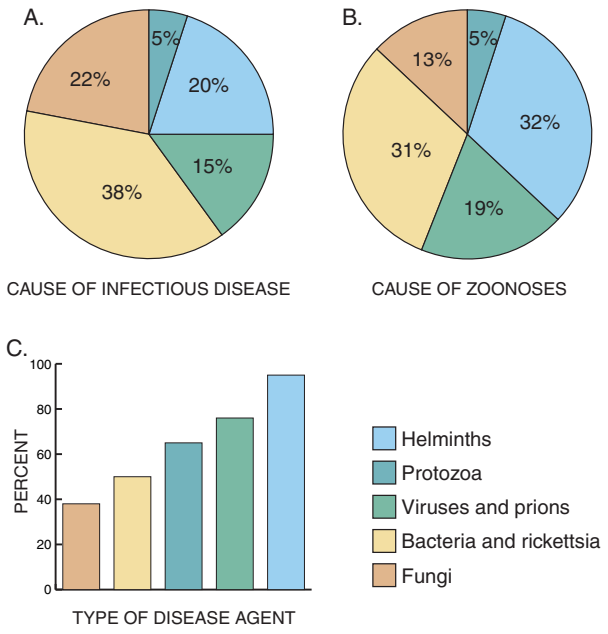



**Figure 2.3** (A) The percentage of infectious diseases caused by different classes of disease agents, (B) the percentage of agents within those classes causing zoonoses, and (C) the percentage of infectious agents known to be pathogenic for humans that are also zoonoses. (Adapted from data from Taylor et al.[46])

Many of the zoonoses affecting humans are of wildlife origin or wildlife have a role in their maintenance, transmission, and/or geographic spread. A recent analysis of the probability of known infectious agents becoming emerging diseases of humans disclosed that those agents infecting wildlife were twice as likely to become emerging diseases as those without wildlife hosts,[49] which suggests that wildlife are an important aspect of the resurgence of infectious disease in humans. Therefore, it is noteworthy that disease emergence in humans has been accompanied by disease emergence and geographic spread in free-ranging wildlife populations. **Companion animals**, primarily cats and dogs that come into contact with infected wildlife, can provide a "bridge" for transporting zoonoses of wildlife into households, veterinary clinics, boarding kennels, and animal shelters. Disease transmission to humans from companion animals often involves mechanical processes (i.e., contaminated mouth parts) and transfer of infected arthropod vectors (i.e., ticks) rather than infections acquired from a clinically ill dog or **cat**.

The current magnitude of disease emergence in wildlife populations is unprecedented and appears to have begun about a decade earlier than that for humans. In general, wildlife have a greater intimacy with the environment than humans and that intimacy may provide enhanced sensitivity to environmental changes that are important indices for disease emergence. Therefore, disease surveillance in free-ranging wildlife populations may provide an early warning system.[50] This concept has been selectively applied for monitoring arboviruses, influenza, and some other diseases. For example, virus activity in birds has been the most sensitive index for the presence of West Nile virus. A greater focus on monitoring wildlife diseases may be especially valuable for protecting human health in natural areas with expanded human presence and for protecting economic interests associated with the domestic animal industry.

### *Wildlife and Zoonoses*

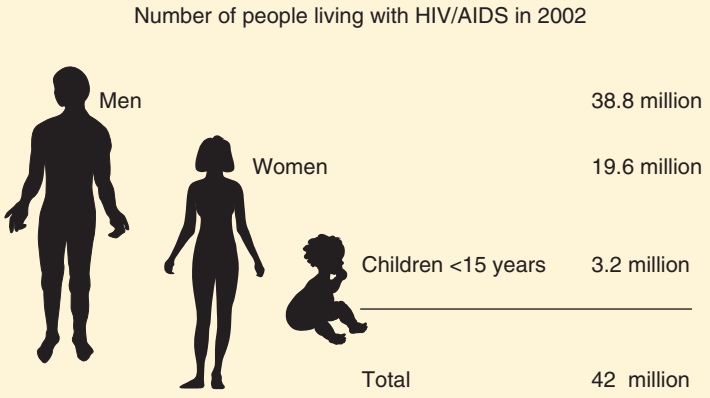
Contact with wildlife, including animals being handled, animal bites, and the consumption of animals, all provide opportunities for the direct transmission of zoonoses. However, wildlife often have other roles in the ecology of diseases affecting humans (Box 2–4). Especially noteworthy are the ability of infectious disease agents to cross species

**Table 2.2.** Common routes for human exposure to zoonoses.

| Type of exposure | Activity examples | Wildlife and zoonoses examples |
|---|---|---|
| Direct contact with infected animals | • Handling infected organs and tissues when processing carcasses for consumption | Rabbits, muskrats, and tularemia |
| | • Handling infected live animals for biological, clinical, and other purposes | Migratory birds and chlamydiosis |
| | • Processing carcasses for scientific study | Staphylococcal and *Erysipelothrix* infections from deer and migratory birds |
| | • Bite by infected animal | Carnivores, bats, and rabies |
| Consumption of infected animal meat and other products | • Preparation of smoked fish at temperatures too low to destroy potential pathogens | Salmonids, whitefish, and type E botulism |
| | • Preparation of game sausage contaminated with parasites | Cougar jerky and trichinosis |
| | • Inadequate cooking of infected meat | Deer and *Escherichia coli* |
| | • Raw consumption of parasite-laden foods | Cod and cod worm |
| Bites by infected or contaminated vectors | • Outdoor activities that provide exposure to ticks, mosquitoes, and other arthropods | Birds and West Nile fever |
| Contact with contaminated environments | • Skin contact in infested environments | Waterfowl, rodents, snails, and swimmers itch |
| | • Aerosol exposure caused by disturbing soils and other substrates heavily laden with infectious agents | Insectivorous bats, blackbirds, and, histoplasmosis |
| Ingestion of contaminated water | • Drinking untreated water from naturally flowing surface waters and lakes | Aquatic rodents and giardiasis |
| Companion animal bridge | • Contact with pets that have consumed diseased wildlife | Cats, prairie dogs, and plague |
| | • Bites from infected ticks that transfer from wildlife to pets and their humans | Cats, rodents, and tularemia |

# Box 2–3 Social Impacts of Emerging Infectious Disease

The definition of disease in medical dictionaries spans many pages to provide succinct generalizations of disease conditions from A (i.e., Acosta's disease or acute mountain sickness) to Z (i.e., zymotic disease or a disease due to the action of an enzyme…).[349] In contrast, the definition of disease in a standard dictionary, while still focusing on impacts on organism form and function, is brief and includes an added dimension, "…a harmful development (as in a social institution)."[350] Disease affects our economy, behavior, and governmental regulations. Thus, emerging infectious diseases often have impacts that extend far beyond the clinical manifestations of specific diseases on individuals, the economic costs for diagnosis and treatment, and those collective costs on individuals, families, and populations. AIDS is but one of many examples.




The emergence of AIDS has been accompanied by social stigma for individuals testing positive for HIV, regardless of whether or not they have clinical disease. Various forms of discrimination have appeared in the work place and in other components of society as a response to beliefs, perspectives, and fear of AIDS. A variety of regulatory and procedural changes have been implemented that impact health-care providers, blood banks, and education processes. Other adjustments in human behavior, our activities, and our way of life have also resulted from the emergence of this disease. Clearly, the burden of AIDS extends far beyond the pathogenesis of the causative virus. Similar broad-based responses are often associated with wildlife species that harbor diseases of concern.

## Chronic Wasting Disease

Deer hunting is a traditional activity for millions of Americans and in many rural areas it remains an important social activity with significant economic ramifications for communities. This activity also has significance for wildlife management agencies. For example:

- In 1996, hunters spent $897 million within Wisconsin in pursuit of their hunting activities. Those expenditures support a great deal of employment and provide a foundation for wildlife programs such as land acquisition and management, wildlife education, and research.[351]
- During recent years, more than 600,000 Wisconsin deer hunters have been spending nearly $500 million annually in pursuit of their sport.[352]
- Deer hunting licenses in Wisconsin contribute $21 million, or about one-third of the Wisconsin wildlife management budget.[353]

As with AIDS, public perceptions and fear about chronic wasting disease (CWD) are causing major adjustments in human behaviors, regulatory processes, agency and scientific priorities, and resource allocations. The general basis for human concern about CWD lies in the causative agent being a prion, the same type of agent responsible for bovine spongiform encephalopathy (BSE) or "mad cow disease." Transformation of that agent has resulted in a

Photo by Christina Sigurdson

variant Creutzfeldt-Jakob agent that has caused approximately 100 human fatalities.[354] Public concern is that a similar variant may evolve from prions associated with CWD. Agriculture agencies also are concerned because several captive elk and deer herds associated with game ranching and commerce have been infected by CWD. Another concern is that a high prevalence of CWD in wild cervids may enhance the potential for a variant to evolve and infect livestock.

Because of CWD's negative impacts on deer and elk health and survival, and the social and economic importance of these species, wildlife conservation interests also are involved. The focus for wildlife agencies is eradication of CWD where possible and preventing its spread to other states where this disease does not already exist. The result of these concerns is an unprecedented effort focused on combating a disease affecting free-ranging wildlife.

- A multiagency plan involving the collaboration of 9 federal agencies, 14 state agencies, 4 universities, the International Association of Fish and Wildlife Agencies, and others was developed to guide a coordinated effort to combat CWD.[348]
- CWD has been present in Colorado for several decades and recently the Colorado Division of Wildlife completed a 5-year Strategic Plan that establishes disease management and elimination as one of its highest priorities.[355]

Fiscal support for many state wildlife agencies is highly dependent upon license sales. Concern about consuming deer meat reduced Wisconsin deer license sales, which negatively impacts fiscal resources for carrying out deer and other wildlife conservation responsibilities. During 2001, the Wisconsin Department of Natural Resources sold over 688,000 licenses to hunt deer. Survey results indicated a 10 to 20 percent reduction during 2002.[352,353] This relatively small percentage reduction results in a substantial loss of revenue and is compounded by the costs to combat CWD.

The 2002 appearance of CWD in white-tailed deer in Wisconsin has been costly. The resources required to combat CWD, even with supplemental funding, burdens agency capabilities by redirecting funds and agency staff, thereby, compromising the ability to address other needs.

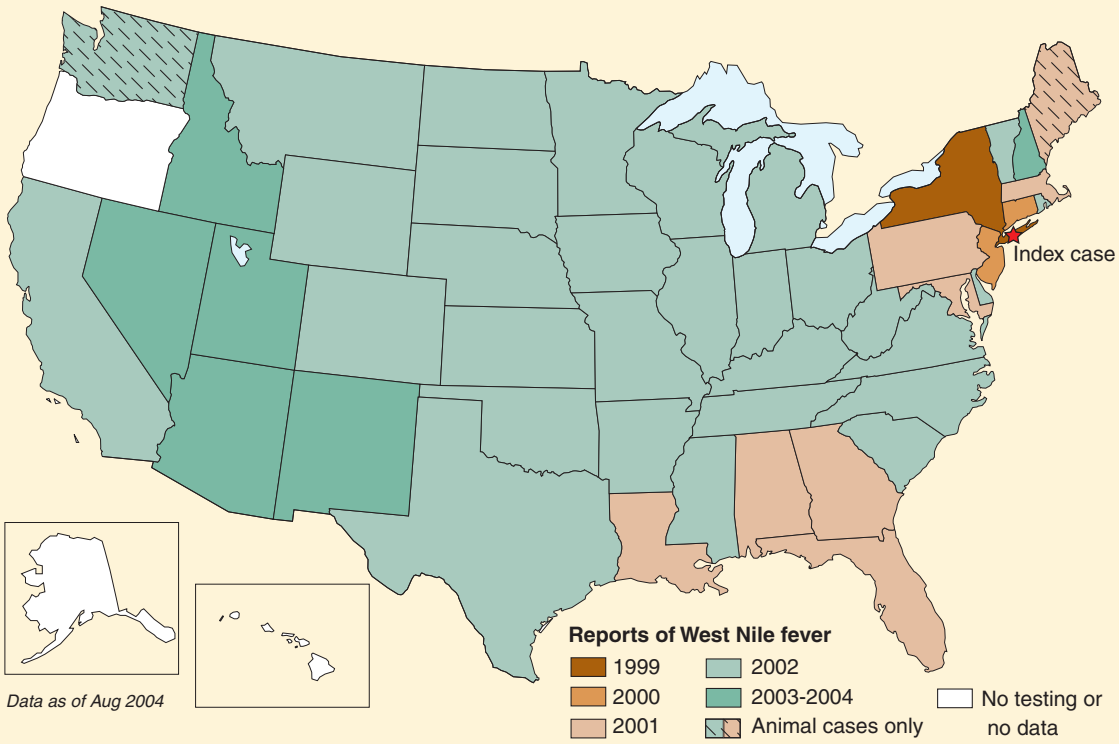
- An intensive surveillance and testing program was implemented to determine the geographic distribution of CWD in Wisconsin. Hunter participation is a major component of these types of programs and the testing provides hunters with evaluations of the deer they harvest.
- Construction of a state facility was required to process the estimated 40,000 Wisconsin deer heads for sample extraction during 2002. Also, many people were needed to collect the deer heads in the field, to process them for sample extraction, and to do laboratory evaluations.

There also are general community costs associated with CWD. For example:

- The projected net loss to Wisconsin's economy as a whole from reduced spending by nonresident deer hunters alone was estimated to be approximately $5 million to $10 million for 2002.[352]

Photo by Milton Friend

- A variety of regulations promulgated in response to CWD and the processes for their enforcement impose still other costs, including adjustments in human activities. Those regulations establish inter- and intrastate conditions for the movement of live elk and deer, their carcasses, and components of those animals that have been harvested by hunters and commercial activities.[351,354–357]
- CWD in North America has resulted in the suspension by South Korea and Japan of the importation of deer, elk, and their products from the United States and Canada.[356]
- Indemnity payments are provided by the United States Department of Agriculture (USDA) for the voluntary depopulation of captive **cervid** herds within the United States that are infected with CWD.[356]
- Small business operations such as taxidermists, processors of elk and deer, and a variety of other services provided to deer hunters also are negatively impacted, as are deer and elk farms found to be positive for CWD.




Impacts from CWD are most notable in rural areas where deer hunting is a popular activity and are felt by those communities in many ways.

- Motels, restaurants, gas stations, and a number of other local businesses in rural areas are quite dependent upon deer hunting to bring business to their community during deer season.
- The revenue for a small, rural Wisconsin feed store in an area removed from the CWD focal area fell by tens of thousands of dollars due to the 2002 statewide ban on deer feeding, one of the disease-control actions initiated. A large business operation projected a reduction of $300,000 in revenue due to that ban.[353]
- A small business that sells archery equipment suffered a reduction of more than 50 percent of normal sales because of reduced deer-hunting activity.[353]

Clearly, the economic impacts in Wisconsin associated with CWD have substantial ramifications. Impacts of this disease outbreak on agencies and local communities are striking, especially considering the absence of a single documented human or livestock case of disease attributed to CWD during the more than two decades that this disease has been present in limited areas of the Western United States.

## West Nile Fever

The 1999 appearance in North America of West Nile fever (WNF) is another vivid example of human impacts associated with disease emergence in wildlife. Unlike CWD, WNF is clearly a zoonosis. Its appearance was first detected because of a cluster of human cases, including several deaths, in the New York City area. The human cases occurred along with a cluster of bird deaths, primarily **crows**. Since 1999, this disease has spread across the USA and into Canada. The host range for WNF includes horses as well as other domestic animals, a broad array of wildlife species (primarily birds), and humans. Thus, like CWD, attempts to combat WNF have an interagency orientation and are multifaceted.

- Shortly after the diagnosis of WNF in New York City, the Centers for Disease Control and Prevention (CDC) and the USDA cosponsored a workshop and developed guidelines for disease

*Photo from USGS files*

Photo by Milton Friend

surveillance, prevention, and control. Experts from federal, state, and city agencies joined members of the academic community and the private sector in that undertaking.[358]

- National guidelines developed for the control of West Nile virus (WNV) place a high priority on monitoring for the virus and providing guidance for the timing of that activity based on geographic regions in the USA.[358]

- Training workshops, protocols for diagnostic and surveillance activities, and data management are some of the integrated efforts established to combat WNV.

Many agencies are incurring substantial costs for the surveillance and testing programs needed for guiding actions to protect human health. In addition, because of the risks to human health, mosquito abatement activities have increased, as well as the level of protective measures required for processing wildlife in disease diagnostic laboratories.

- In early December 2000, the CDC provided 16 States and local health departments along the East Coast of the USA with $2.5 million to enhance their surveillance for WNV and to develop local measures to prevent outbreaks. Pennsylvania anticipated it would spend $9.8 million in addition to CDC funds to develop internal mosquito-control and surveillance plans.[360]

- During the spring of 2001, New York received a $3.9 million grant from CDC to combat WNV, in addition to the $21.9 million for local virus control activities proposed by the Governor in the State budget to cover 2000–2001 costs.[359]

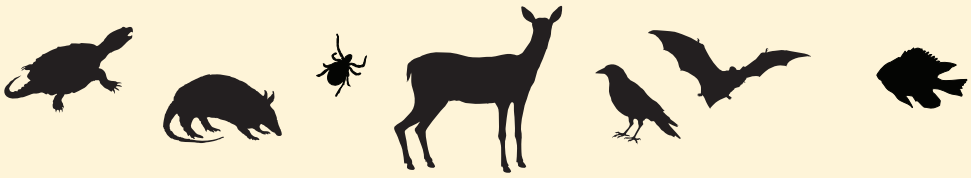
- During 2000 and 2001, the CDC provided more than $58 million to State or local health departments to develop or enhance epidemiologic and laboratory capacity for WNV and other mosquito-borne diseases. In fiscal year, 2002, approximately $35 million in federal funds were awarded by the CDC to these agencies to address the continued spread of the virus.[361]

- Other societal costs include major investments in research on disease ecology and evaluation of vaccination as a means for combating WNF.

WNF also has ramifications for wildlife conservation and education programs. Many thousands of birds have died from this disease. Also, the specter of WNF looms as an ominous shadow over wildlife rehabilitation. The rehabilitation of sick and injured wildlife is a popular activity and one that is primarily carried out by the private sector rather than by government agencies. Thousands of individuals participate, the majority as volunteers that have very limited training and knowledge of animal diseases. In general, the facilities where these activities are conducted are inadequate for the containment of WNF in the event of an outbreak. Also, protective measures for people are seldom adequate to prevent disease exposure in the event infectious disease is brought into the facility. The emergence of WNF calls for additional knowledge of disease risks within wildlife rehabilitation programs and adjustments in how rehabilitation programs are conducted. WNF has also struck zoos, causing many bird deaths, and threatens captive breeding programs that enhance the populations of endangered avian species.

CWD and WNF are but two of the multitude of emerging and reemerging infectious diseases confronting society. Human activities and behavior are major factors contributing to disease emergence. Hopefully, greater appreciation of the effects of these diseases on our way of life and things that we value will result in behavior that reduces the spread of pathogenic microorganisms.

# Box 2–4 Wildlife and Zoonoses: Different Roles for Different Diseases



Wildlife may contribute to zoonoses in ways other than direct transmission between wildlife and humans. For influenza, the greatest wildlife contribution is the transfer of genetic material between influenza viruses that leads to disease emergence in humans, not direct contact between humans and wildlife. For some diseases, such as Lyme disease and ehrlichiosis, the major role for wildlife is disease maintenance in nature; for other diseases such as giardiasis, the primary role is environmental contamination by wildlife (e.g., shedding infectious agents into surface waters) leading to human infections. Birds infected with West Nile virus serve as a source for infection of mosquitoes that then infect humans, and the disease spreads through the movements of infected birds. The following examples highlight some of the major roles wildlife have in the ecology of zoonoses, besides direct contact transmission of the disease.

### Tick Production

Lyme disease is typically contracted from the bite of infected ticks and not from contact with wildlife that may harbor the causative spirochete bacterium. Tick populations are dependent upon having adequate numbers of hosts to feed on as their growth and reproduction requires blood meals to provide the necessary nourishment. Typically, when larvae emerge from the egg, they feed on small rodents, such as mice; nymphs and adults feed on larger mammals. Thus, mice and white-tailed deer are the species that contribute to the maintenance of tick populations, and through that contribution, to the transmission of Lyme disease.

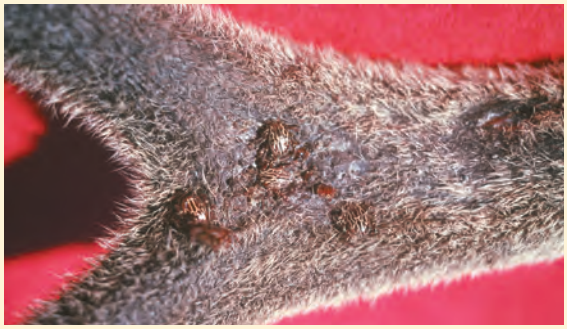


Photo by Milton Friend

### Gene Pool Contributions

Migratory birds, especially shorebirds, are an important source of influenza viruses but rarely suffer clinical illness or mortality from those viruses. However, recombination is a characteristic of influenza viruses, and involves the transfer of genetic material between different influenza viruses to produce new virus strains. These exchanges involve mammals, especially swine, as well as birds and

Photo by Milton Friend

are the source of virus variants that are lethal for poultry and other variants that cause disease in humans.

### Developmental Hosts

Many metazoan parasites require one or more wildlife hosts for the parasite to become pathogenic for humans. For example, wildlife species such as red foxes and coyotes are definitive hosts for the tapeworm *Echinococcus multilocularis*, the cause of hydatid disease; they are essential components of the disease cycle. Infected wild carnivores imported into areas where this parasite is not yet established pose a significant threat to human health by introducing the parasite into the wildlife populations of the new area.

Photo by Milton Friend

Photo by Elizabeth Ciganovich

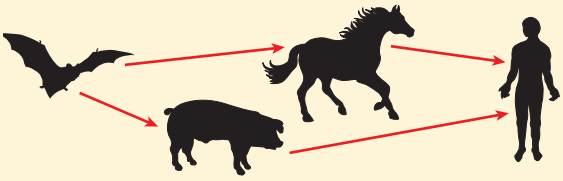
### Environmental Contamination

Giardiasis is a common waterborne disease of humans. Cysts of the protozoan parasite that cause this disease are shed in the feces of infected animals, such as beaver, and are immediately infective. Surface waters become contaminated in this manner and unless adequately treated, become a source for human infections.

### Amplification Hosts

The ability of arthropods to become infected (biological transmission) or for their mouth parts and excretions to be contaminated at levels sufficient for mechanical disease transmission is a function of the number of organisms present in the blood meal taken by the arthropod. The rapid spread of West Nile fever in North America has been facilitated by the high level of viremia in infected crows and some other bird species. Mosquitoes feeding on these birds become infected and continue the transmission cycle when they take their next blood meal from another susceptible host.

USGS file photo



### Interspecies Transfers

Wildlife often harbor microbes and parasites that are not pathogens for them, but become disease agents for other species that interface with those wildlife or environments contaminated by them. Human infections occur as a result of contact with other species, not with the wildlife host. Among numerous examples are the recent emergence of Nipah and Hendra virus infections. Both involve domestic animals as the source of human infections and fruit bats as the wildlife reservoir hosts.[48]

Photo by Milton Friend

### Spread of Infection

The movement patterns of wild birds have long been associated with the spread of infectious disease, including zoonoses.[362–367] Arthropod vectors often are “hitchhikers” that transfer to new environments and geographic areas during bird and other wildlife movements. These arthropods may provide means for transmission of indigenous pathogens or they may be infected with diseases new for the environments they enter. Infected wildlife also may serve as a source for infection of local arthropod populations as occurs for mosquitoes and West Nile fever. Earlier studies have suggested that infected migrating birds are the source for repeated West Nile virus (WNV) introductions in the central highlands of South Africa. Also, experimental studies and isolations from nature indicate that WNV can adapt to ticks and may be transferred by tick bite.[363]

barriers. Recent examples include HIV-1 and HIV-2, Hendra and Nipah viruses, *Streptococcus iniae*,[51] and other disease agents, suggesting that this ability may be more important than was recognized previously.[48] Also, the natural movements of wild birds can contribute to zoonoses by introducing arthropod vectors, by transporting disease agents, and by other means.[52–55] This multiplicity of roles is interactive with environmental conditions. Therefore, the dynamics of environmental disruptions and change can greatly influence the role of wildlife in the ecology of zoonoses.

## Disease Emergence in Wildlife

> **"Pathogens that infect wildlife are twice as likely to become emerging diseases of humans as pathogens without wildlife hosts" (Cleaveland et al.).[49]**

More noteworthy disease events have affected free-ranging wildlife during the 20th century than have been collectively reported previously. Currently, infectious disease has become established as a prominent cause of mortality for wild birds, some **enzootic** diseases have increased in frequency of occurrence and geographic distribution, and rare or previously unreported diseases have taken a large toll on wildlife. The large number of avian mass mortality events in the USA and Canada stands as testimony to the toll of wildlife affected by disease (Fig. 2.4). Large numbers of other types of wildlife from amphibians to fishes to **mammals** are also victims of disease. Not all of these diseases are zoonoses, but in many instances there is no clear distinction between zoonoses and diseases that are not,[56] because host susceptibility is mediated by a number of factors.[52] Impairment of the immune system, such as from HIV infections, poor nutrition, and other means, can result in disease from organisms generally of low virulence for humans.[57–59] Tuberculosis due to human infection with avian strains of *Mycobacteria* in AIDS cases is an example.[60]

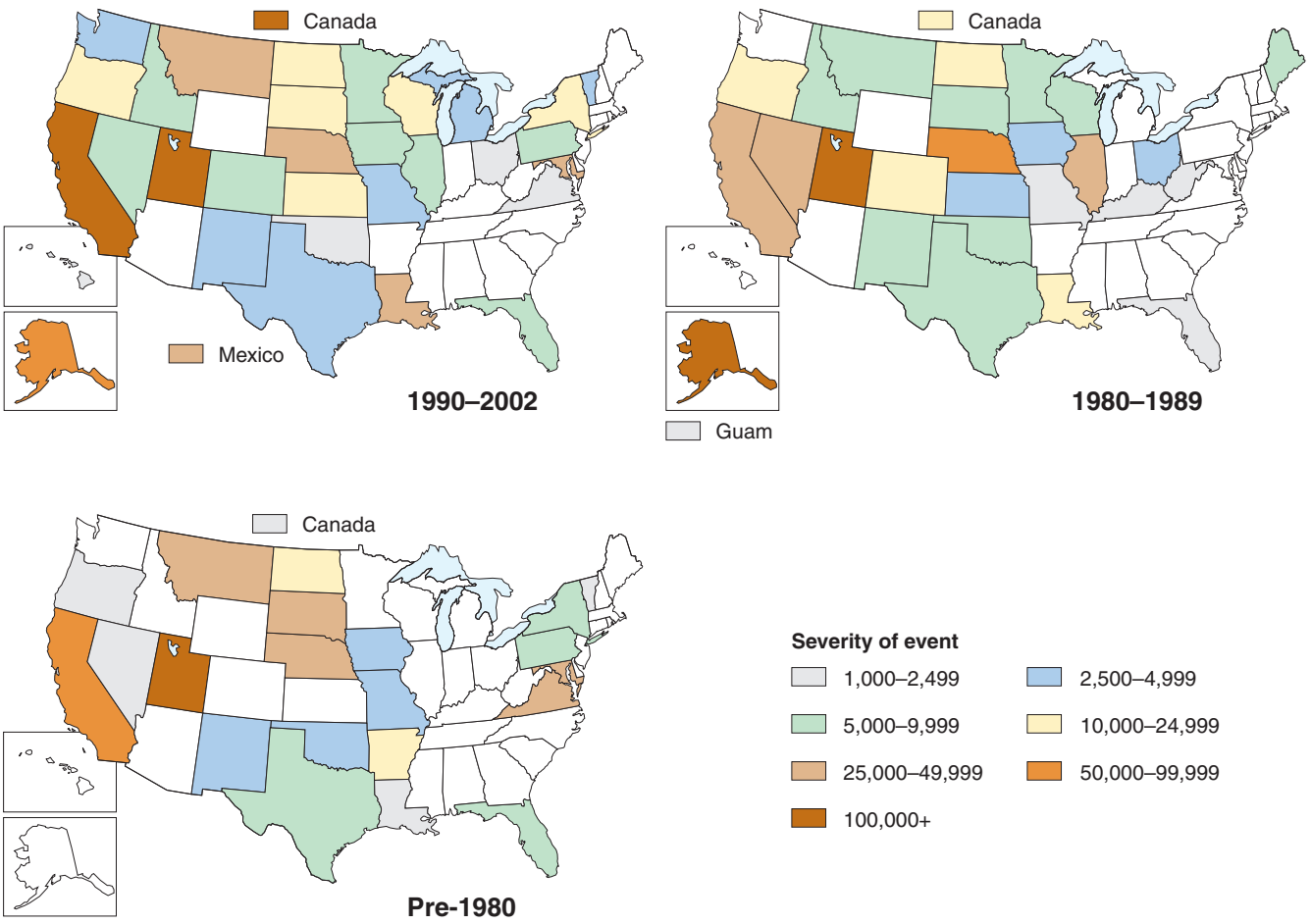



**Figure 2.4** Avian mass-mortality events within different time periods in the United States.

### *Marine Environment*

**"Of the natural factors that influence abundance of marine organisms, few are more spectacular or less understood than disease" (Sinderman).[61]**

Human actions are increasingly challenging the oceans' capabilities to sustain the abundance and diversity of life. Introductions of nonindigenous pathogens and other aquatic organisms from discharges from land, ballast water, and other means are altering ocean ecosystems, degrading the quality of the marine environment, and contributing to disease emergence in a wide variety of nearshore and offshore marine species (Fig. 2.5). Human health and well-being also are jeopardized by disease emergence and reemergence in those species as a result of:

- Consumption of **finfish** and **shellfish** contaminated by biological toxins (e.g., "red tides"), toxic chemicals, and microorganisms;
- Reductions of fish stocks by disease, placing further stress on already overharvested fish populations that are important as a source of food for many people;
- Increased risk for exposure to pathogens when swimming in contaminated waters;
- Direct exposure to "red tides" causing serious illness; and
- Economic impacts associated with contamination of beaches, shellfish beds, and finfish.[62]

The frequency of infectious disease events in marine ecosystems and the broad spectrum of marine species affected are unprecedented and have far-reaching implications for the integrity of those ecological systems and the biological services they provide. Therefore, it is not surprising that disease emergence in the marine environment was the focus for two international meetings in 1999 in which direct linkage between human disease and the marine environment were explored.[63] The contributions of the marine environment to the maintenance and spread of cholera was one of the topics considered (Box 2–5).[335]

#### Plant Communities

**Seagrass** beds, such as eelgrass and turtlegrass, that serve as important habitat for a variety of **waterfowl**, **shrimp**,

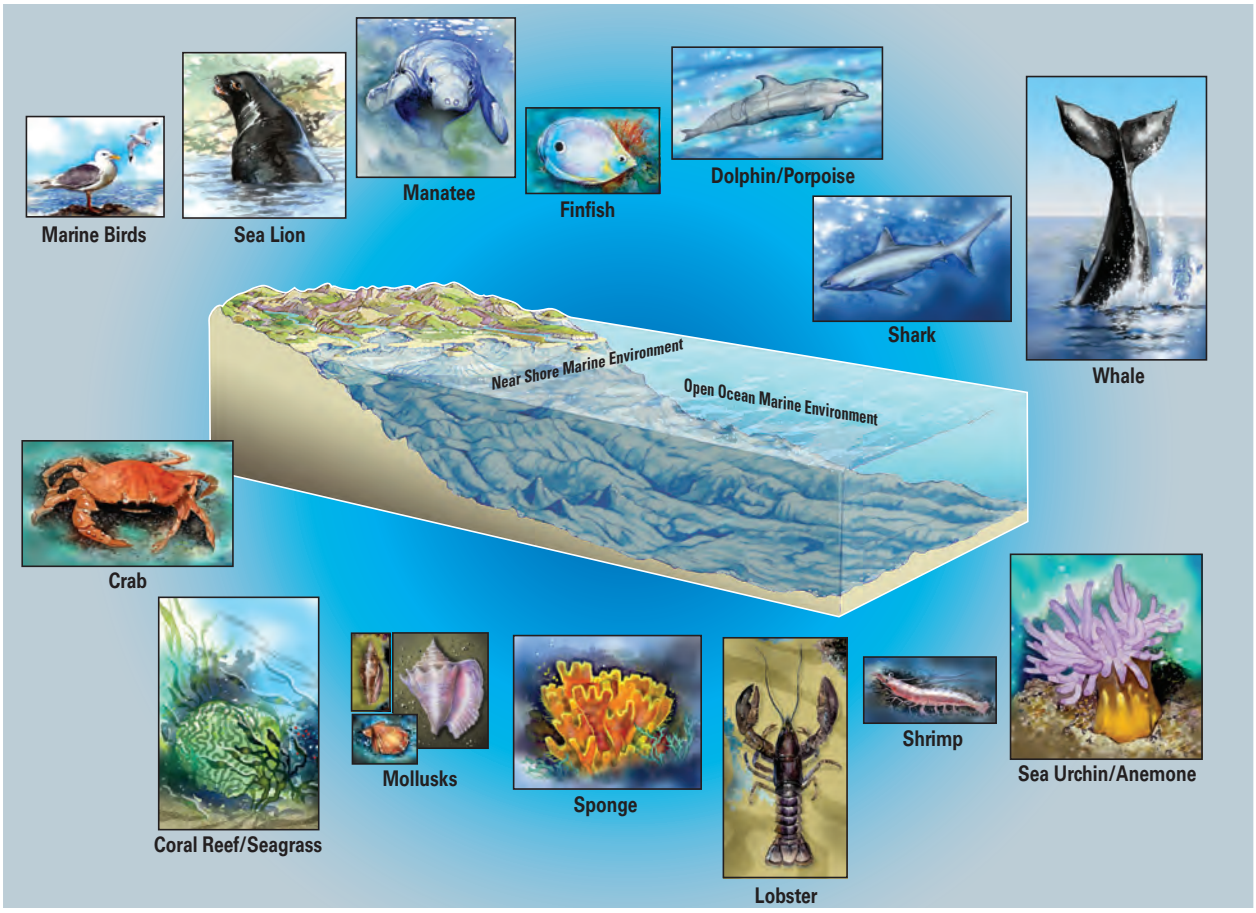



**Figure 2.5** Examples of species affected by disease emergence in the marine environment.

# Box 2–5 Cholera and the Marine Environment

Illustration by John M. Evans

Cholera (*Vibrio cholerae*) is an ancient "voyager" whose capacity to result in pandemic spread has left many footnotes to the story of civilization.[289,291] Despite great advances in the control of many infectious diseases, cholera remains as an epidemic disease claiming hundreds of thousands of lives each year. The seventh pandemic is ongoing and includes noteworthy epidemics that began in 1991 in India, Bangladesh, and the Americas.[368]

The continuing challenges posed by cholera, one of the most feared infectious diseases of humans, are integrally linked to marine environments.[369]

- "Historically, most of the major epidemics or outbreaks of cholera around the world have originated in coastal regions…There is compelling evidence that *V. cholerae* always is present in the aquatic environment and proliferates under non-epidemic conditions while still attached to, or associated with eucaryotic organisms…**Zooplankton** play a significant role as a reservoir of *V. cholerae* in the environment."[369]
- Cholera eruptions appear to be stimulated by global changes taking place, including ecosystem alterations resulting from human actions.[369]
- Cholera was reintroduced into South and Central America during 1991–92 following over more than 100 years of absence.[370] By the end of August 1992 more than 600,000 cases and 5,000 deaths were reported from 20 countries.[371] From January 1, 1991 to September 1, 1994, more than 1 million cases, including 158 cases in the USA, and nearly 10,000 deaths occurred in 21 countries in the Western Hemisphere.[372]
- Since 1991, approximately 120 countries worldwide have reported indigenous cases of cholera and in nearly half of them cholera has been a recurring problem.[369]

The marine environment is the natural habitat of *V. cholerae*; crustaceans and copepods are natural hosts for sustaining this organism. Linear correlation exists between the growth of *V. cholerae* and increased sea-surface temperature. Plankton blooms are dependent on warm ocean temperature. Cases of cholera are correlated with the response of phytoplankton to increased temperature and the subsequent appearance of the zooplankton blooms that harbor the cholera organisms. Other factors also are involved but the relations just noted illustrate the importance of the marine environment for sustaining *V. cholerae*.[368]

The occurrence of the cholera bacterium in coastal waters of the USA has been well documented. However, despite the environmental presence of *V. cholerae* there is a paucity of cholera cases obtained from these waters because of the advanced sanitation practices and facilities that prevent the secondary spread of *V. cholerae* through drinking-water contamination.[373] While these safeguards have served the people of the USA well, it is sobering to recognize that *V. cholerae* is present in our coastal waters, patiently waiting for an opportunity to mount a successful invasion. It is also sobering to recognize that modern technology does not provide an invincible shield against waterborne diseases such as cholera. The 1993 invasion of cryptosporidiosis that resulted in 403,000 infections via the drinking water for Milwaukee, Wisconsin[336] should be considered a "wake-up call."


Adapted from the World Health Organization

**scallops**, fish, and other aquatic species have been severely degraded in many areas and essentially eliminated in some. Many factors are involved, including the fungal pathogen, *Labyrinthula zosterae*. This marine slime mold is responsible for "seagrass wasting disease" along the Atlantic coast of the USA. Outbreaks of this disease off the coast of New England have most recently occurred during the 1980s and again in 1997. Mass mortality of turtlegrass in Florida Bay is also associated with *Labyrinthula*.[62]

Disease in **seagrass communities** is noted to illustrate the pervasive nature of infectious disease occurring within natural biological systems. Because **plant communities** are a fundamental building block for biological communities, disease impacts on plant species can have far-reaching ecological impacts. For example, the seagrass community of Florida Bay supports over 100 species of finfish and over 30 crustacean species, including both permanent residents and species that temporarily occupy this habitat as a major nursery.[64] Seagrasses not only provide habitat for many species but they also are an important part of the food web for some species. Nutritional degradation of food webs can negatively impact immunocompetency in animals just as poor nutrition affects immunocompetency in humans.

Seagrass wasting disease is not a new disease. In the 1930s, a similar disease of unknown etiology almost eliminated eelgrass in the North Atlantic. That disease decimated eelgrass beds along the Atlantic coast from North Carolina (USA) to Nova Scotia (Canada). However, healthy eelgrass populations were reestablished by the 1960s over most of the affected area. Reappearance of the same, or a similar disease, occurred in 1987[65] in eelgrass beds on the border of New Hampshire and Maine (USA) and that same year in turtlegrass beds of Florida Bay.[64] Seagrass epizootics that began in the 1930s and again in 1987 were not limited to the eastern seaboard of North America. Seagrass mortality during both time periods also occurred in Europe and along the Pacific coast of the USA.[65]

### Coral Reef Communities

**Coral reefs** also sustain higher forms of life. Not only are coral reefs one of the world's most spectacular ecosystems, they also are a critical resource for millions of people and are inhabited by between one-half million and 2 million species, if not more.[66] Coral reefs are home to about 25 percent of all marine species[67] and recently have become a focus for investigations because of the emergence of diseases and other factors impacting reef viability. Disease has caused a dramatic loss of coral reef species and degradation of coral reefs in many areas of the world (Fig. 2.6). The magnitude of loss that has occurred is unprecedented in recent geologic history.[68]

During the late 1980s, white-band disease almost eliminated the dominant coral-space occupier in lagoonal reefs in Belize.[69] On a regional scale, white-band disease has probably been the most significant factor in reducing populations of elkhorn and staghorn corals. Elkhorn coral, previously one of the most important and most common species of coral in the Caribbean, is now rare. The abundance of corals in Jamaica declined from a mean of 52 percent coral reef habitat along the coastline from 1977 to 1980 to 3 percent from 1990 to 1993.[70,71]

The continuum of new diseases and reef species being affected (Box 2–6) suggests that the coral reef systems are badly stressed and that additional diseases will continue to emerge. For example, in 1997, "rapid-wasting disease" appeared as a new pathology affecting the massive *Montastraea* and *Colpophyllia* corals of Caribbean reefs. Coral reefs of Florida vividly illustrate that disease impacts are increasing relative to the number of species being affected and geographic distribution of diseased coral. A 1999 evaluation found 82 percent of all reef study locations were affected, which is a 404-percent increase over 1996 and that 85 percent of all reef corals were affected, a 218-percent increase over 1996.[63]

In addition to disease affecting hard corals, **soft corals** such as sea fans, along with **sponges** and **sea urchins** also have been affected by emerging diseases. The rapid spread since the 1980s by the variety of novel pathologies of reef organisms suggests that disease agents are entering naive populations that have little ability to reject their invasion.[72] The effects from these diseases threaten the viability of many reef systems. For example, a bright orange bacterial pathogen


**Figure 2.6** Locations of of coral disease. (Compiled from Spalding and Green[66] and the World Conservation Coral Disease Monitoring Center—NOAA coral disease database.)

# Box 2–6 Emerging Disease and Coral Reefs

Coral reefs throughout the world have been severely degraded during recent decades. Emerging diseases are a major factor in this degradation, primarily through the destruction of **scleractinian** stone-like corals that provide the basic framework for reefs. Initial reports of disease affecting reef-building corals appeared during the early 1970s and were viewed at that time as unique situations. Today, disease has been observed in more than 100 coral species (primarily **hard corals** but also some soft corals) on reefs in more than 50 countries.[66] The areas involved include popular diving locations such as the Caribbean islands, Fiji, the Red Sea, and the Great Barrier Reef of Australia. However, the prevalence and diversity of coral disease appears to be greatest in the tropical western Atlantic,[374] primarily within the Caribbean.[66] The number of distinct diseases being observed within this area, as well as globally, has increased substantially since the 1970s.

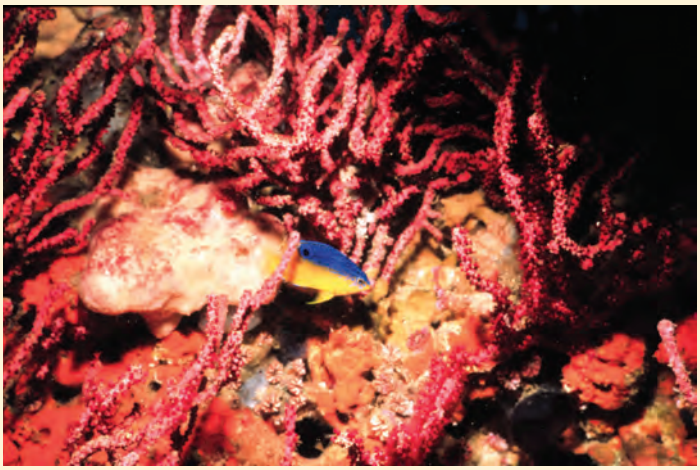


*Photo courtesy of the National Oceanic and Atmospheric Administration*

Coral reef and tropical fish off of the coast of North Carolina.

## Disease of Scleractinian Corals

### Black-Band Disease

Black-band was the first disease reported to affect scleractinian corals and was first described in 1973 from Belize. Subsequently, reports followed during the 1970s from reefs off Bermuda and the Florida Keys.[374] Black-band disease is now known to exist throughout the Caribbean, in reefs of the Indo-Pacific (Philippines, Fiji), the Red Sea, and the Great Barrier Reef.[374–376] Hard corals such as star coral, fire corals, and soft corals such as **gorgonians** (sea fans) are affected. **Acroporids** (branching corals) have been found infected on the Great Barrier Reef.[377] Significant mortality from black-band disease has occurred in at least 13 species of coral[378] and it is a major factor in the recent decline (1990s) of hard corals on reefs off Jamaica.[375]

Black-band disease is caused by a microbial mat consisting of a complex of organisms. The most dominant species are the cyanobacterium *Phormidium corallyticum* and bacterium of the genus *Beggiatoa*. Other species in the mat complex include numerous heterotrophic bacteria (organisms that derive energy from consumption or absorption of other organisms), marine fungi, and bacteria of the genus *Desulfovibrio*.[379,380]

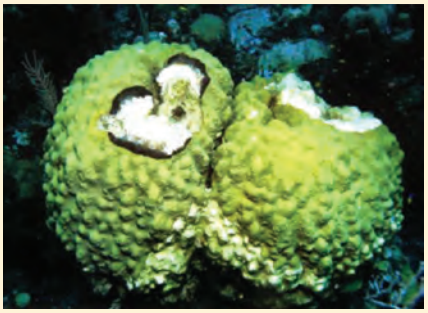


*Photo courtesy of the National Oceanic and Atmospheric Administration*

Black-band disease.

### Red-Band Disease

Red-band infections of corals were first noted during the early 1980s and thought to be a variant of black-band disease infecting sea fans off Belize,[374] but was described as a separate disease based on observations made during 1991 at a site southwest of Bimini in the Bahamas.[381,382] This disease is also known to be present on the west coast of Puerto Rico and in the Florida Keys[383] and may be present as brown-band disease on the Great Barrier Reef. Infections have occurred in 20 coral species in five scleractinian families.[374]

A microbial mat similar to that for black-band disease is involved but differs in species composition, migration across the coral, and daily activity.[381] Red-band disease is associated with a cyanobacterium of the group *Oscillatoria* spp. but the primary cyanobacteria present in the red-band may differ between geographic locations. Other organisms known to be part of the mat complex are other cyanobacteria, the bacterium *Beggiatoa*, heterotrophic bacteria, and the nematode *Araeolaimus*.[374,383]

### White-Band Disease

**Acroporid** corals from St. Croix, U.S. Virgin Islands, were first reported infected with white-band disease in the 1970s. Massive mortality of elkhorn corals occurred in 1977 on the reefs of Buck Island and Tague Bay and was part of a progressive destruction of the majority of Caribbean *Acropora* during the late 1970s and early 1980s.[374,383] This disease is widespread, occurring in reefs throughout the Caribbean from the Florida Keys to Panama and Nicaragua. It is also present in reefs of the Philippines, the Red Sea, the Gulf of Oman (Arabian Sea), and the Great Barrier Reef.[374] White-band disease attacks multiple species of scleractinian corals but has been most destructive of branching corals.

The original form of white-band disease that emerged in the 1970s is referred to as Type I. Type II, a more aggressive form relative to the speed of disease progression in infected coral, emerged during the early 1990s,[377,383] and has only been found in the Bahamas. Both diseases appear to be due to bacterial infections. Bacterial aggregates have been identified in some, but not all cases of Type I disease. Specific species of bacteria have not been identified as the cause for this disease. Bacteria similar to *Vibrio carchariae* have been identified as a probable agent for Type II disease.[384]

### Yellow-Band Disease (Yellow-Blotch Disease)

Some authors refer to this disease as yellow-blotch disease in the Caribbean and yellow-band disease in the Arabian Gulf. Yellow-band disease was first reported as ring bleaching in the 1970s.[385] In 1990 it was first associated with bleached corals in the Cayman Islands,[386] and in 1994 it was first noted as an independent disease in the lower Florida Keys.[374] Yellow-band disease is now known to occur in many Caribbean reefs.[383] Recent transect studies (1997–1998) revealed that this disease affects as much as 90 percent of star coral.[385] It is the latest coral disease in Colombian waters (observed in April 1998) and the cause of a major epizootic affecting several coral species.[387] Yellow-band disease has also been observed in pristine reefs in San Salvador waters[377] and in the Arabian Gulf at Jebel Ali in Dubai, United Arab Emirates.[388]

Yellow-band disease affects star coral in the Florida Keys and in the Netherlands Antilles, but different species

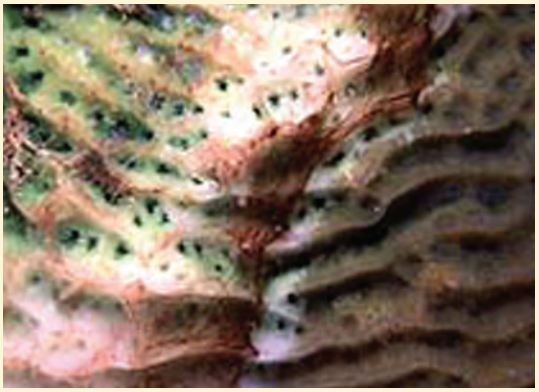


*Photo courtesy of the National Oceanic and Atmospheric Administration*

Red-band disease.

*Photo courtesy of the National Oceanic and Atmospheric Administration*

White-band disease.

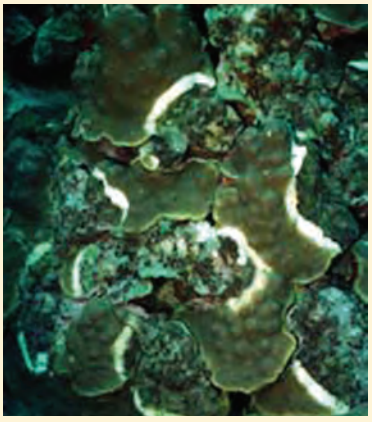


*Photo courtesy of the National Oceanic and Atmospheric Administration*

Yellow-band disease.

including branching corals are affected in the Arabian Gulf. Prior to its appearance in Colombian waters, this disease had only been known to affect two species of corals (star coral and mountainous star coral). An additional seven coral species were found affected in Colombia.[387] The cause of this disease is unknown, but may be of bacterial origin.[374]

### Rapid Wasting Disease

This disease syndrome was first noted in Bonaire, Netherlands Antilles during late 1996. It is a rapidly spreading new condition that exists throughout the Caribbean affecting star coral and brain coral, two of the major reef builders of this region.[377,383] A filamentous fungus and a ciliate (protozoan) parasite associated with the fungus were originally thought to be responsible for rapid wasting disease.[72,383,389] However, recent observations indicate that **parrotfish** feeding on the coral may be the primary cause of this syndrome.[388]

### Dark-Spot Disease

First observed in 1990, this disease affects massive starlet coral and some other star corals throughout the Caribbean. Transects during 1997–1998 disclosed up to 56 percent of those species of corals to be affected.[383,385] Dark-spot disease was the first record of a coral disease in Colombia (1990 at the Rosario Islands) and has affected 10 coral species in reefs of that country.[387] The pathogen involved is unknown.

### White Pox Disease

Elkhorn coral was found affected by white pox disease around 1995 in the Florida Keys. Rapid geographic expansion has followed and this disease now occurs throughout most of the Caribbean. An unknown infectious agent is believed to be the cause for this disease.[72,383]

### Coral (white) Plague

There are two distinct forms of white plague. Type I is a slowly progressing infectious disease and was first reported in 1977 on Alligator Reef in the Florida Keys. It has been documented for several species of nonbranching corals such as brain coral and fleshy coral. Type II white plague was also first observed in Alligator Reef (1995), but in contrast to Type I, is a rapidly spreading disease.[377,383] The 17 scleractinian coral species infected is the greatest number of these corals ever reported for any disease in the Caribbean region. Only nonbranching corals are affected. Type II white plague is the first known disease of elliptical star coral, the primary species affected during epizootics.[390]

Three major epizootics of Type II white plague have occurred in different reef areas of south Florida: the middle Keys in 1995; the southern Keys and Dry Tortugos during 1996; and reefs north of Miami during 1997. White plague (Type I and Type II combined) was first reported in Colombian reefs in 1994 affecting only one species (*Montastraea cavernosa*). It is now widespread and has affected 21 hard coral species.[387] A single dominant bacterium associated with the disease line has been isolated and shown to be contagious under experimental conditions. This organism is most closely related to *Sphingomonas*.[390]

## Disease of Other Reef Organisms

### Coralline Algal Disease [Coralline Lethal Orange Disease (CLOD)]

The orange-yellow growth of an unidentified bacterium that attacks coralline algae (*Porolithon* spp.) gives this disease its name.[383] Initially observed in June 1993, coralline algal disease has spread over 10,000 km, affecting

*Photo courtesy of the National Oceanic and Atmospheric Administration*

Dark-spot disease.

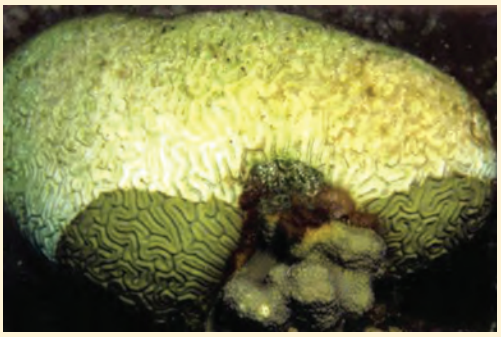


*Photo courtesy of the National Oceanic and Atmospheric Administration*

Coral (white) plague.

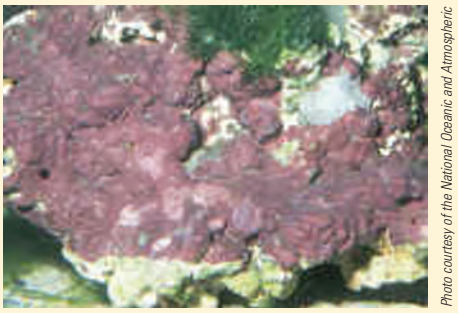


*Photo courtesy of the National Oceanic and Atmospheric Administration*

Coralline algae disease.

South Pacific reefs from the Cook Islands to the Mariana Islands.[391] In 1996, a new condition that attacks these algae but has a different appearance appeared in the Caribbean. Between 25 to 75 percent of the coralline algae has been killed at some Caribbean sites.[383]

### Sea Fan Disease

Sea fans are soft coral life forms. Mass mortality events involving these species were first reported in the Caribbean during the 1980s: Trinidad (1981/82); Costa Rica (1982/83); Panama (1982/83); Colombia and San Andrea Island (1986/88). The causative agent, although unknown, was highly virulent, resulting in almost total mortality. Disease appeared to be restricted to the Caribbean continental coasts. A second, less virulent, epizootic wave, extending at least 2,500 km, began in January 1995. This event reached at least from Trinidad westwards to the Panama/Colombia border in the southern Caribbean, and northwestwards to the Bahamas and the Florida Keys in the northern Caribbean.[392] This latest event has been shown to be caused by a fungus (*Apergillus sydowii*).[393-395]

### Sponge Disease

Die-offs of barrel sponges have been reported from the Florida Keys since the 1980s. In 1996, mass mortality (40–50 percent) affected the barrel sponge population in reefs along Palm Beach, Florida. The previous year mortality occurred off Key Largo in the Florida Keys.[396] Mortality is caused by a rotting disease that leaves holes in the sponge frame.

A rapidly spreading disease of large barrel sponges *(Xestospongia muta)* appeared in the Belize Barrier Reef Tract during 1996 and spread to Curacao, Tobago, and Panama. Several different species of sponges were affected in Panama and a different species of barrel sponge in Tobago.[383] The pathogen involved has not been identified for any of the sponge disease events.

*Photo courtesy of the National Oceanic and Atmospheric Administration*

Sea fan disease.

### Sea Urchin Disease

During 1983 and 1984 the black long-spined sea urchin suffered mass mortality from disease throughout its entire geographic range. That initial epizootic is thought to be the most widespread epizootic ever recorded for a marine **invertebrate**.[397,398] Approximately 3.5 million square km (not counting Bermuda) were impacted by this event.[398] In 1983, Jamaican reefs alone lost about 100 million sea urchins during an 8-week period.[71] A second epizootic followed in 1984, further stressing any survivors from the previous event.[397] Densities of this species in Jamaica were reduced by 99 percent from pre-die-off estimates and have remained suppressed.[71] A similar die-off struck the Florida Keys during May 1991.[569] Additional mass mortalities from 1995–1997 affected sea urchins in Puerto Rico, Antigua, Aruba, Jamaica, and Curacao.[383] Mass mortalities from 1980 to 1982 reduced green sea urchin populations in Nova Scotia by about 90 percent.[62] The pattern of mortality associated with sea urchin die-offs is consistent with infectious disease, but the causative agent(s) have not been determined. An amoeboid protist, *Labyrinthula* spp., is thought be the cause of the Nova Scotia die-off.

Numerous other maladies have also appeared as diseases of reef organisms during recent years. For example, in 1996 an unnamed new disease appeared in Brazil in a colonial benthic (bottom dwelling) organism, commonly found on shallow reefs in the western Atlantic. Bacteria are thought to be the primary pathogens, and fungi and other organisms are most likely secondary invaders; it is widespread along the Brazilian coast but not seen elsewhere.[399]

Noted reef biologists are obviously quite concerned about the magnitude of disease:

"The spread of coral reef diseases has become so commonplace, and with such intensity, that they have become the major cause of accelerating coral mortality in many locations and are likely to become far more prevalent in coming years" (Goreau et al.).[383]

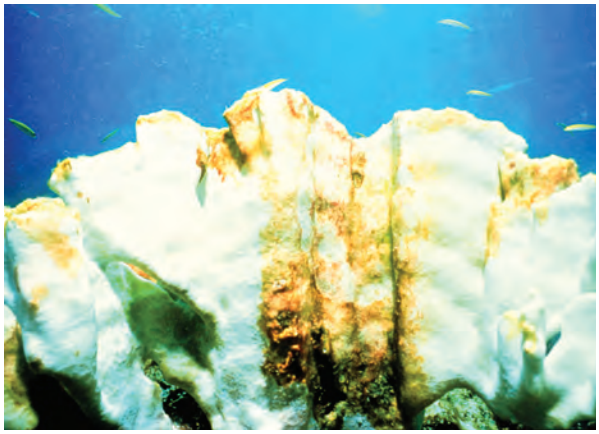


Photo courtesy of S. Miller, OAR/National Undersea Research Program (NURP), University of North Carolina at Wilmington

**Figure 2.7** Bleached coral.

that is lethal to coralline algae, living organisms that cement dead corals together to make reefs, was first recorded in the Cook Islands of the Pacific Ocean in June 1993. Within a year, this disease had spread over a distance of at least 6,000 kilometers. In 1992, coralline lethal orange disease (CLOD) was nonexistent at Great Astrolabe Reef sites in Fiji, but by 1993, it was present in 100 percent of the reefs. Because coralline algae play critical roles in forming reef rims throughout the Indo-Pacific region, CLOD may significantly affect reef ecology and reef building processes.[73]

Coral bleaching is an additional pathology of reef systems that is occurring over broad geographic areas. This malady (Fig. 2.7) is seen as a whitening of corals due to loss of symbiotic algae and/or their pigments.[74] The first description of coral bleaching was in 1984, but scientists in French Polynesia made the first observations 11 years earlier. Coral bleaching occurs regularly in the Indian and Pacific Oceans and the Caribbean Sea, and is now common at many sites.[74] A major coral bleaching event occurred throughout the Caribbean in late 1995 (Table 2.3). For some places, such as Mexico, Cuba, Honduras, and Belize, this was the first occurrence. Bleaching was most evident in the western, central, and southern Caribbean.[75]

The most geographically extensive and severe mass bleaching event occurred during 1998[66] (Fig. 2.8). High sea surface temperatures associated with El Niño were among the factors responsible for coral bleaching.[69,76] This pathology has long-term impacts because of the magnitude of mortal-

**Table 2.3.** Relative severity of coral bleaching[a] within different areas of the Caribbean during a 1995 bleaching event.[7,75]

| Unremarkable | Slight | Highly evident | Severe |
|---|---|---|---|
| Tobago | Barbados | Bahamas (San Salvador) | Bonaire |
| | Bermuda | Belize[b] | Cayman |
| | Costa Rica | Colombia | Curacao |
| | Saba | Cuba[c] | Jamaica |
| | St. John | Dominican Republic | Venezuela |
| | | Honduras[b] | Mexico[b] |
| | | Puerto Rico | |

[a]Unremarkable=percentage of coral affected too little to be noticeable; slight=bleaching of some coral evident but only a low percentage of coral affected; highly evident=bleaching readily visible because of the moderate to high amount of coral affected; severe=bleaching widely occuring and affecting most of the area.

[b]Bleaching was a minor occurrence previous to this event.

[c]First bleaching event

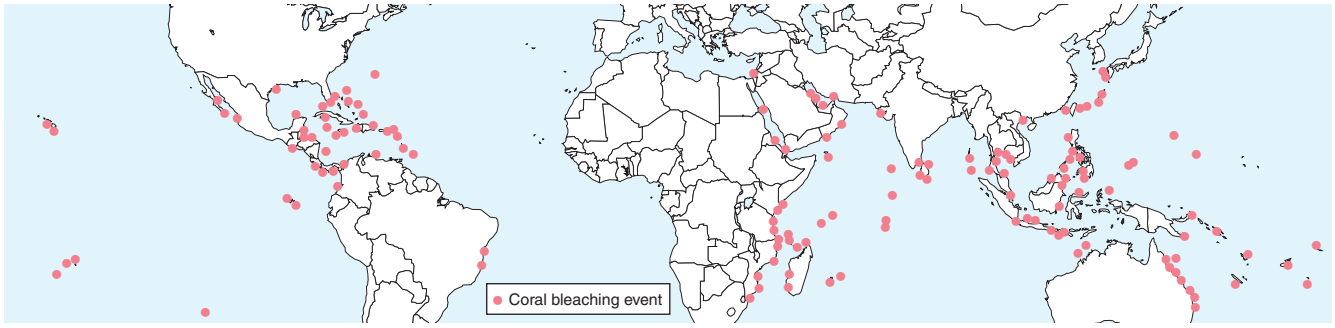



**Figure 2.8** Locations of coral bleaching, 1998. (Data modified from Spalding and Green[66] and the World Conservation Coral Disease Monitoring Center—NOAA coral disease database.)

ity[76] and the damage to the coral's reproductive capacity that persists beyond the period of stress from elevated water temperatures.[77] Also, coral bleaching is often followed by the appearance of infectious disease. It is not clear whether the damage caused by bleaching results in invasion by infectious disease or whether the physical appearance of bleached corals masks the observability of lesions from some infectious diseases that already may be present.

### Finfish and Shellfish

Infectious disease emergence and reemergence in fish is a worldwide concern[78] because of impacts being experienced by wild stocks of shellfish and finfish and those affecting **mariculture**, shrimp farming, and other forms of **aquaculture**. Several diseases of marine species that are caused by biotoxins also are noted because of the implications for human and wildlife health (Box 2–7).

Marine **mollusks**, such as **oysters**, **clams**, and **abalone**, and **crustaceans**, such as shrimp, have experienced increasing numbers of mass mortality events during recent years. These events have been caused by a growing number of infectious agents and by other factors.[79–81] Many recognized infectious diseases are associated with the commercial farming of these species. Pathways for pathogens to move between wild and commercial stocks of marine shellfish exist because of the close associations between these populations. Wild populations are the broodstock for some of these species and aquaculture often occurs within estuarine areas. The greater surveillance of cultured stock and the interface that often exists with wild stock can obscure the origin (wild vs. cultured stock) of diseases. Also, these relations make it difficult to separate the natural geographic distribution of the causative agents from distribution caused by industry movement of broodstock and commercial shipment of products (Box 2–8).

Infectious disease also is occurring more frequently and in greater numbers of species of marine finfish than previously reported.[78,82] Like shellfish, these diseases most often are first detected among captive populations of finfish, especially those raised in aquaculture facilities. Like shellfish, **salmon** and some other finfish are reared in estuarine environments that can provide a water corridor for disease transfer between wild and cultured stocks of finfish (Fig. 2.9). Two situations regarding infectious disease in shellfish and finfish are likely: aquaculture may be the probable source for many of the emerging infectious diseases being encountered (Table 2.4) and/or aquaculture simply facilitates the detection of infectious agents present in wild populations.

Egtved disease, or viral hemorrhagic septicemia (VHS), is an example of how the interface between farmed and wild fish stocks can result in the emergence of highly virulent pathogens. This disease is caused by infection with viral hemorrhagic septicemia virus (VHSV). Different strains of VHSV exist in Europe and North America. The European

Photo courtesy of the National Oceanic and Atmospheric Administration

**Figure 2.9** An aquaculture net pen offshore of Catalina Island, California, 2000. The walkways provide access for commercial fish feeding and pen maintenance.

strain is highly virulent for **salmonids**, causing mortality in juvenile fish that has approached 100 percent and up to 25–75 percent in adults. In contrast, the North American strain is relatively avirulent for the salmonids evaluated, but causes occasional self-limiting epizootics in its Pacific herring **reservoir host**. The high virulence of the European strain of VHSV is thought to be the result of a mutant strain evolving from infection of rainbow **trout**.[83]

VHS is the most serious viral disease of farmed rainbow trout and occurs widely in mainland Europe.[84] Rainbow trout were imported from North America into Europe in the late 1800s. Later infection by VHSV may have resulted in contaminated water from cultured fish infecting ocean salmon.[83] Recent findings suggest that rainbow trout initially became infected from a marine source rather than vice versa and that Atlantic herring fed to farmed fish may have been the original source for infection.[85]

VHSV was first isolated in North American salmon in 1988 and recommendations have been made to eradicate VHSV-infected **hatchery** stocks to reduce the possibility of the North American strain evolving into a more virulent salmonid virus.[83] The 1994 appearance of VHS in Scotland was the first in the British Isles and occurred in tank-reared turbot. All of the fish on the infected farm were destroyed to combat this infection.[84]

Among the viral diseases infecting marine finfish, the nodaviruses and the iridoviruses are the most prominent emerging diseases because of the frequency of disease events and the number of different fish species affected. Nodaviruses cause behavioral abnormalities prior to death because of their predilection for nerve tissue. This group of viruses has infected over 20 species of marine fish that belong to 11 different families; infections have been found throughout much of the world, except for the Americas and Africa.[86] Atlantic salmon, sea bass, grouper, and Atlantic halibut are among popular food fish being infected.[78] Red sea bream iridoviral disease is a major representative of the iridoviruses and first appeared in cultured red sea bream in Japan in 1990. Since

# Box 2–7 Biotoxins and Disease Emergence

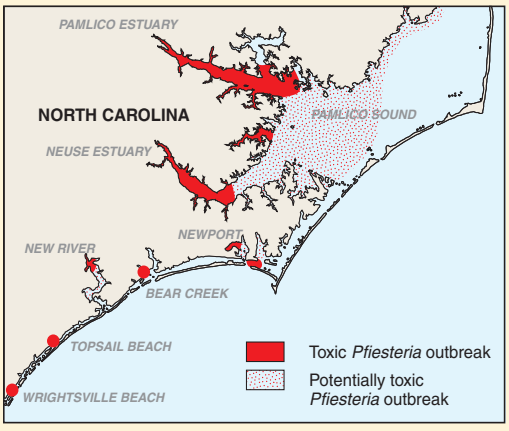
Photo by Milton Friend

Large-scale algal blooms in marine environments have become an increasing focus for concern and study since the 1970s because of the potential for toxic side effects. Referred to as harmful algal blooms (HABs), these events include such conditions as red tides, brown tides, and cyanobacterial blooms. Wildlife die-offs,[327,400] especially those involving fish,[401,402] contamination of shellfish beds, and human illness have all been associated with HABs.[62, 402] During the past decade, *Pfiesteria* has become a high-profile disease because of large-scale fish kills and a reported association with human illness.

*Pfiesteria piscicida* is the representative species for a novel group of **dinoflagellates** (single-celled, plantlike organisms) first discovered in the 1980s.[403] Association of these organisms with HABs was first described in 1992[401] and the taxonomy for dinoflagellates was resolved in 1996.[404] An estimated loss attributed to *Pfiesteria* of more than 1 billion fish in 1991 occurred in the Neuse and Pamlico Estuaries of the Albemarle Pamlico System of North Carolina (USA).[62] This System is the second largest estuary on the USA mainland and has been the site of numerous *Pfiesteria*-related fish kills between 1991 and 2000.[405] *Pfiesteria* was first linked to mortality in these fish by assays of water samples from a mass mortality site of Atlantic menhaden.[401] *Pfiesteria* has also been implicated as the cause of mortality in a variety of other **estuarine fish** along the Atlantic coast.[405,406] Blue crab also have been killed by *Pfiesteria* in some of those events. Laboratory studies have disclosed that a broad range of finfish (at least 33 species) and four species of estuarine invertebrates are susceptible to *P. piscicida* and *Pfiesteria*-like dinoflagellates.[403]

Initially, it was thought that the open sores in Atlantic menhaden were caused by *P. piscicida*. However, skin and muscle ulcers in fish can result from numerous causes, are commonly associated with fungi, and, in general, are referred to as ulcerative mycosis.[407,408] In one study, a highly pathogenic fungus, *Aphanomyces invadans*, not *Pfiesteria* toxins, was found to cause skin ulcers in menhaden.[408]

Whether or not *Pfiesteria* is a threat for human health and to what extent is controversial. The first association between human illness and exposure to *Pfiesteria* was reported among laboratory personnel working with the organism during the early 1990s.[409] In 1997, additional cases of human illness were associated with exposure to waterways where the dinoflagellate was present.[410] These and other reports suggest that chronic or recurrent high-level exposure to *Pfiesteria* toxin may result in a




Adapted from Glasgow et al., 2001[145]

distinctive clinical syndrome characterized by difficulties in learning and memory.[411] However, the general conclusion reached during the *National Conference on Pfiesteria: From Biology to Public Health*[412] is that, "The consequence of human exposure to *Pfiesteria* toxin and the magnitude of the human health problem remains obscure."[403] Because of environmental conditions present, the states of Delaware, Florida, Maryland, North Carolina, South Carolina, and Virginia are most likely to be affected by the presence of *P. piscicida* in their estuaries.[413] The high density of the human population along the eastern seaboard of the USA, the recreational use of estuarine areas of that region, and the commercial importance of those areas for finfish and shellfish ensures that *Pfiesteria* will remain a focus for intensive investigations until questions of human health risk are resolved.

In 1996, another type of dinoflagellate resulted in an unprecedented epizootic that killed approximately 150 West Indian manatees along the southwest coast of Florida. A red tide dinoflagellate bloom (primarily *Gymnodinium breve*) that produced brevetoxin was identified as the cause of that epizootic, the largest reported disease event affecting this species. The estimated population of West Indian manatees is only 3,000 animals;[414] it is one of the most endangered marine mammals in the coastal waters of the USA.

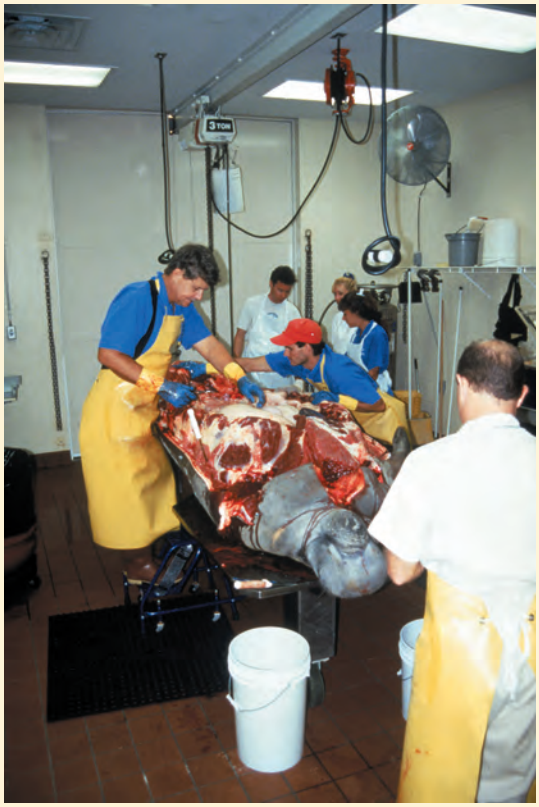


Photo courtesy of the Florida Marine Research Institute

Manatee postmortem exam.

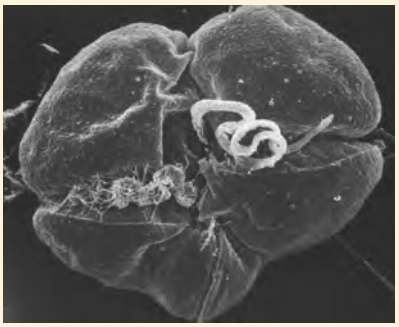


Photo courtesy of the Florida Marine Research Institute

*Gymnodinium breve.*

Red tide blooms are common on Florida's west coast, thereby providing the potential for manatees to periodically become exposed to brevetoxin. The infrequency of **manatee** mortality from this cause suggests that brevetoxicosis may be cumulative and require prolonged exposure and/or high dose exposure to this toxin.[414] Retrospective analysis of tissues from a smaller 1982 epizootic[415] support the involvement of brevetoxin as a component of that event.[414]

A bloom of *G. breve* is also believed to have been the cause of an epizootic involving lesser scaup in the Tampa Bay area on the west coast of Florida. Several thousand birds died during that event.[326,327] Another type of biotoxin, domoic acid (DA), caused the mortality of about 300 birds in Santa Cruz, California (USA) in 1991. Brown pelicans and Brandt's cormorants were the primary affected species. The toxin was associated with a bloom of the diatom *Pseudonitzschia australis*. That event was the first reported incident of DA poisoning in free-ranging wildlife, the first documentation of DA in finfish (northern anchovy), the first report of DA being produced by the diatom *P. australis*, and was the first report of DA outside of Canada's Atlantic Coast.[416] Previously, the only documented case of DA poisoning had occurred in 1987 on Prince Edward Island, Canada. More than 100 human cases, including three deaths resulted from the consumption of cultured blue mussels.[259] Shortly after the bird event in California, DA was found in clams and **crabs** harvested in Washington and Oregon, and human cases may have resulted from ingestion of clams.[320]

Poisoning of humans by DA is known as amnesic shellfish poisoning because of memory loss that sometimes occurs. DA has joined several other types of shellfish poisoning and ciguatera fish poisoning as examples of diseases caused by biotoxins that appear to be increasing.[252,259,320] These diseases are associated with coastal marine ecosystems. The general increase in their occurrence in humans is associated with degradation of the marine environment.[62,320] Human exposure to these toxins is not limited to consumption of contaminated foods. Aerosol exposure from contaminated environments has resulted in respiratory entry and disease. Therefore, HABs affect recreation (e.g., swimming) and food consumption along with the attendant economic consequences that often result.

**Table 2.4.** Examples of important emerging and reemerging diseases of marine finfish in North America (contribution of F. Panek, U.S. Geological Survey).[a]

| Disease | Type | Period of emergence | Geographic area | Comments |
|---|---|---|---|---|
| Damselfish neurofibromatosis (DNF) | Virus-like agent | 1980s | Florida and Caribbean coral reefs | • Transmissible cancer affecting bicolor damselfish.[462]<br>• Exhibits many traits in common with neurofibromatosis type-1 in humans, including multiple plexiform neurofibromas and areas of hyperpigmentation.[463] |
| Infectious salmon anemia virus | Orthomyxo-like virus | Late 1990s | Maine and New Brunswick, Canada | • Highly infectious disease of Atlantic salmon. First reported within Norwegian aquaculture facilities.[464]<br>• First case confirmed in Maine net pens mid-February, 2001. |
| *Streptococcus iniae* | Bacteria | 1970s | USA Atlantic and Gulf coast waters and coral reefs | • Worldwide distribution and usually associated with poor water quality or environmental conditions<br>• Well-known in fish culture since the 1950s; epizootics associated with wild fish since 1970s; most recently implicated as cause of mass mortalities of coral reef fishes.[465]<br>• Recent human cases associated with processing fish.[51] |
| Mycobacteriosis | Bacteria | Mid-1990s | Coastal waters | • A subacute to chronic wasting disease known to affect 167 species of freshwater and saltwater fishes.<br>• Occurs in all coastal waters of the USA.<br>• *Mycobacterium marinum* is primary agent although seven *Mycobacterium* species may be involved.[466]<br>• Causes “fish-handler’s” disease in humans |
| Epizootic ulcerative syndrome | Fungus (oomycete) | 1984 | Coastal waters | • Widespread disease in estuarine fish along the USA Atlantic coast; first recognzed in this area in North Carolina estuaries.<br>• High incidence of ulcerative lesions (see Box 2–7) in Chesapeake Bay and Florida. Atlantic menhaden young-of-year are highly susceptible.[467] |

[a] National Fisheries Research Center, U.S. Geological Survey.

then, it has been reported in 20 species of cultured marine fishes and it has become one of the most threatening viral diseases for several of those species, such as red sea bream, yellowtail, sea bass, and Japanese parrot fish.[87]

Piscirickettsiosis, an emerging rickettsial disease of salmonid fish, is caused by infection with the rickettsia-like organism *Piscirickettsia salmonis* and has been found in four different species of salmon and in rainbow trout reared in oceanwater. This disease has also appeared in freshwater-reared coho salmon and rainbow trout.[82] Rickettsia were not recognized as important pathogens of fish prior to 1989, but that year large-scale die-offs due to *P. salmonis* occurred in coho salmon reared in seawater net pens in southern Chile. This disease was then found during 1992–1993 in salmonids on the west coasts of Canada, Norway, and Ireland,[88] and since has been found on the east coast of Canada.[86] Subsequently, several unidentified rickettsia-like organisms have also emerged as causes of fish mortality. Perhaps the most significant is the organism causing mortality in several species of tilapia in Taiwan where mortality has reached 95 percent at some sites.[88,89]

Explosive epizootics also have appeared in wild fish without an association with fish culture. Beginning in March 1995 and ending in September of that year, a mass mortality due to a herpesvirus infection spread around the coasts of Australia and New Zealand. At least 10 percent of the pilchard population in Western Australia died. No other species were affected. This epizootic was the first large-scale pilchard mortality event reported for Australian and New Zealand waters. The characteristics of the disease pattern (focal origin, high mortality, and rapid spread) are indicative of an infectious agent entering a naive host population.[90] The source of this pilchard herpesvirus epizootic is unknown. This event provides an example of the vulnerability of wild fish stocks to large-scale mortality from disease even under the unconfined conditions of the ocean environment.[91]

A substantial number of other diseases of marine finfish have been recognized in association with the expansion of species being cultured and the increasing magnitude of fish farming to meet human demands. Not addressed in the examples provided is the myriad of bacterial diseases that have appeared as expanding or previously unreported diseases of marine and freshwater finfish; nearly half of the unreported taxa involved have appeared in only two countries, Spain and the USA. The most dramatic increase of fish-pathogenic taxa is in the number of **vibrios** causing disease.[92]

Disease emergence is not an unexpected outcome of fish farming. The environmental conditions of intensive aquaculture facilitate the transmission and expression of infectious agents present within the farmed species and any infectious pathogens that enter these populations. The broad spectrum of wild and farmed fish species affected confirms the need for sound surveillance programs and aggressive management of diseases that appear.

### Turtles

**Fibropapillomas** (Fig. 2.10) have become an important emerging disease of **sea turtles** since the early 1980s. This disease was first identified in green turtles in 1938 near Key West, Florida, USA,[93] but it was rarely observed until the 1980s.[94] By late 1985, more than 50 percent of the green turtles in Florida's Indian River Lagoon had external tumors.[95] This disease has now been reported in green turtles in every major ocean where this species exists[94] and has also appeared in other species of marine turtles.[96] The prevalence of tumors in some populations sampled has exceeded 90 percent.[94,97] These tumors are believed to be of viral etiology.[98,99,573] Several other diseases also have been recently identified in marine turtles, but too little information is available to determine whether or not these are emerging as new sources of mortality.

Three bacterial diseases (ulcerative stomatitis, obstructive rhinitis, and pneumonia) and associated complexes of disease were found to cause mortality rates of up to 70 percent in farmed and oceanarium-reared 3- to 52-week-old green and loggerhead turtles. Researchers concluded that obstructive rhinitis appears to be a new disease in sea turtles as is the disease complex of the three primary conditions observed. *Vibrio alginolyticus*, *Aeromonas hydrophilia*, and *Flavobacterium* spp. were commonly isolated from turtles with ulcerative stomatitis and obstructive rhinitis, and from the trachea and bronchi of turtles with bronchopneumonia. These findings differ from those of other investigators that attributed pneumonia in farmed sea turtles to a herpesvirus infection and ulcerative stomatitis to a protozoan infection.[100]

### Marine Mammals

**Marine mammals** worldwide have been affected by emerging disease during recent years.[101–108] At least 20 species of **cetaceans** (**whales**, **dolphins**, and porpoises) and 15 species of **pinnipeds** (**seals**, sea lions, and walruses) have been victimized by more than 30 different emerging and reemerging disease agents and disease conditions.[106]

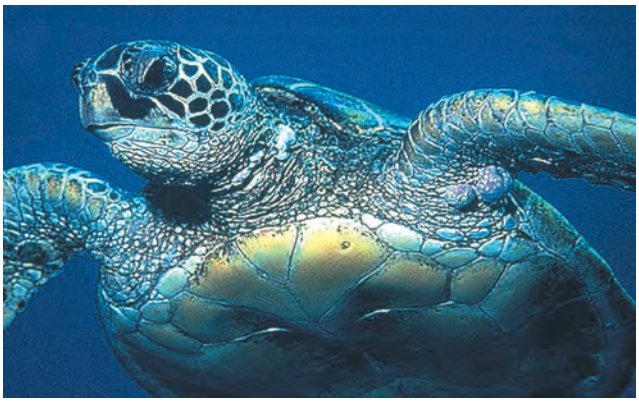


Photo by Thierry Work

**Figure 2.10** Fibropapillomas, or tumors, are an emerging disease of marine turtles.

# Box 2–8 Disease Emergence and Resurgence in Shellfish

A wide variety of marine shellfish are being affected by an equally diverse array of pathogens, many of which are the causes of emerging and reemerging diseases.[80,81] These diseases cause substantial economic impacts because of the high commercial values of mollusks and crustaceans as food products. The following examples are drawn from a more extensive list of pathogens affecting shellfish.

- MSX (Multinucleated Spore Unknown) is a protozoan disease (*Haplosporidium nelsoni*) of the eastern (American) oyster. MSX, or haplosporidiosis, was first recognized in the late 1950s as the cause of 90 to 95 percent mortality of the oysters in lower Delaware Bay (USA).[417] The initial appearance of this disease in nearby Chesapeake Bay in 1959 was followed by an epizootic killing 45 to 55 percent of the oysters on some bars for several years.[418] MSX rarely was found outside of the Delaware and Chesapeake Bay areas until the 1980s. MSX then reached the north shore of Long Island, New York in 1983, the south shore of Cape Cod, Massachusetts in 1995, the Maine-New Hampshire border in 1995, and by 1997 was found throughout Long Island Sound.[417] This disease is one of the five shellfish pathogens whose appearance is notifiable to the Office International des Epizooties (OIE) by member countries because of the high level of infectiousness and serious economic consequences associated with epizootics.

- Withering syndrome (WS) was first detected in abalones in the California Channel Islands in 1985, spread throughout those islands, and by 1992 black abalone were extirpated from six of the eight Islands. The fishery for this species was closed in 1993.[421] Mortalities of over 95 percent when water temperatures are 18–20°C are associated with this disease. The WS disease agent appears to be a rickettsial-like infection that is interactive with warm water conditions.[81,420,421] However, the role of rickettsia has not been firmly established.[420] Red abalone farms exhibited severe economic losses from WS in 1997–1998.

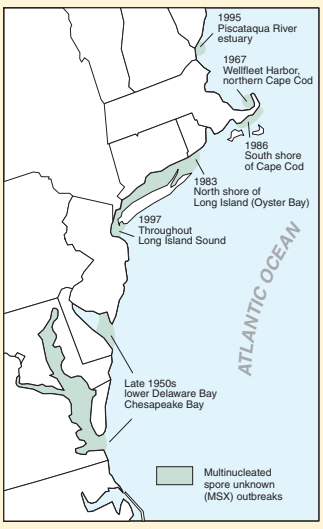
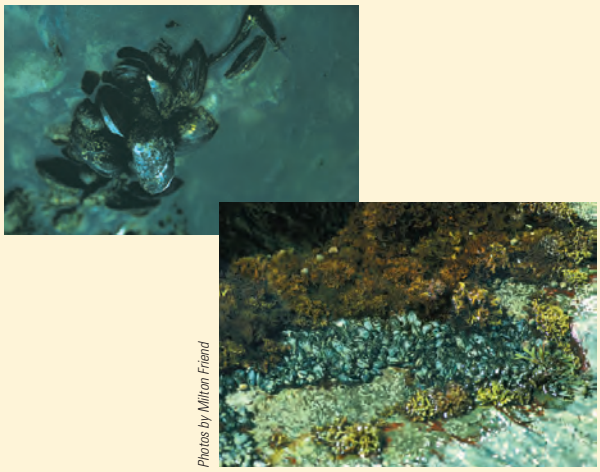




Photo by Milton Friend

- Quahog parasitic unknown (QPX) is an important new disease of **hard clams** or quahogs (quahaugs) in the Northeastern USA. The causative organism is an unnamed microscopic parasite within the subkingdom Protozoa.[422] This parasite was first reported in a limited population of clams in New Brunswick, Canada, during the early 1960s, and later that decade in a shellfish hatchery on Prince Edward Island (PEI), Canada. QPX reappeared at the PEI hatchery in 1989 and has been a persistent problem since, causing significant mortality among hard clams. The extent of hatchery losses raised concern about QPX as a mortality factor in wild populations of hard clams.[423] During 1995, QPX struck two locations on the coast of Massachusetts, causing high morbidity and mortality of hard clams. Anecdotal reports for the Provincetown site indicated nearly 90-percent mortality. However, scientific evaluations using random core samples averaged 30-percent mortality. Microscopic evaluations of nongrowing hard clams indicated 90-percent prevalence of infection. Also, retrospective analyses of archived hard clam tissues identified QPX as being present in a 1993 mortality event in Chatham, Massachusetts, and in a major mortality event in Barnegat Bay, New Jersey, in 1976.

- Juvenile oyster disease (JOD) is another major disease of eastern oysters. Since 1988, recurrent and widespread mortalities from this disease have affected nursery-reared oysters throughout the Northeastern USA. Total mortalities have ranged from 50 percent to nearly 100 percent of total nursery-reared stocks.[424] The causative agent remains elusive. Some investigators present data that a microscopic protistan parasite is the cause and



Photos by Milton Friend


reject a bacterial etiology;[425] others present data supporting a link between infection by a strain of *Vibrio* bacteria and JOD.[424]

- Disseminated neoplasia (DN) emerged during the 1980s and was first described in New England (USA) in **soft-shelled clams** and causing losses in Chesapeake Bay in 1983–1984.[426] DN has been compared to vertebrate leukemia as a disease process. The causative agent and transmission in nature are unknown.[437]

- Mikrocytosis is caused by a microcell parasite (*Mikrocytos mackini*) and is another of the five shellfish pathogens notifiable to OIE. This disease was first confirmed in the USA in New Dungess Bay, Washington, as an infection of Pacific oysters in 2002. The source of the parasite and potential impacts are unknown.[428] Mortalities of Pacific oysters in British Columbia, Canada, were reported from this disease in 1960.[429]

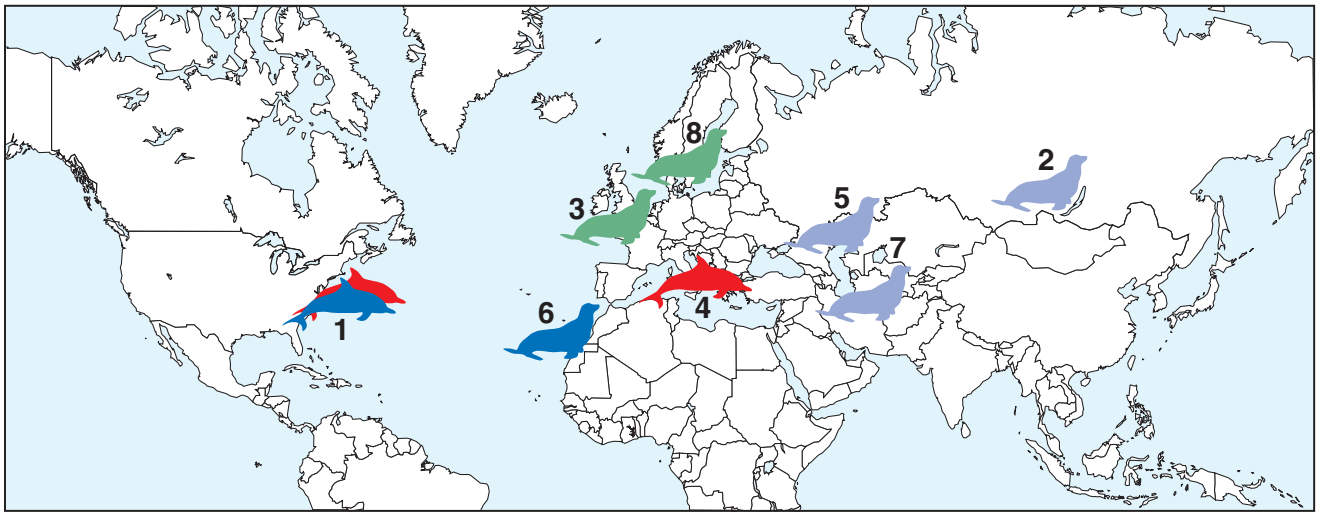
- Dermo disease is another of the five shellfish pathogens notifiable to OIE. The protozoan parasite (*Perkinsus marinus*) causing this disease is a significant pathogen of cultured and wild oysters along the Gulf Coast and East Coast of the USA. The oyster industry along the Gulf Coast of Mexico suffers 50-percent losses annually and this parasite has decimated oyster populations in Chesapeake Bay along the East Coast.[430] This pathogen has likely been present along the Gulf Coast since the early 1900s and in Chesapeake Bay since at least the 1940s. However, since the 1980s, the observed distribution of this disease has changed greatly by expanding northward along the East Coast. Major epizootics have been part of the range extension of more than 500 km north of Chesapeake Bay.[431]

- White spot syndrome virus (WSSV) emerged in 1995 as a serious disease in penaeid shrimp culture. This viral disease was first observed in East Asia in 1992–1993 and spread to the Western Hemisphere causing mass mortality in farmed shrimp in Texas and South Carolina in 1995–1997. Most recently, WSSV has erupted in the shrimp cultures of Central and South America.[432]

- Taura Syndrome disease (TS) emerged in 1991 as a major epizootic disease of panaeid shrimp and spread rapidly during 1992–1993 from Ecuador to other regions of Latin America and to Mexico. TS subsequently occurred in Hawaii and Florida and by mid-1996, had expanded its distribution to include virtually all of the shrimp farming regions of the Americas, including Texas and South Carolina in the USA. This viral disease exists in wild and farmed shrimp and causes high mortality in both. The economic impact of TS in the Americas between 1992 and 1997 exceeded US $2 billion.[79,80,433–435]

- Shell disease of lobster and several species of **crabs** emerged during the 1990s along the East Coast of the USA from Long Island, New York to Massachusetts. This disease causes erosion of chitin (the principal constituent of the animal's shell) and lesions and thinning of the carapace (the shell covering the back of the animal). Mortality is associated with incomplete sloughing of the shell during the molt and subsequent infections of the circulatory system.[436]

The examples cited attest to the diversity of species and types of infectious pathogens impacting wild and cultured shellfish populations. Toxins from algal blooms add to the impacts on these species. Clearly, disease emergence is a significant factor that is challenging the well-being of these important components of ecosystems and food-chains.




**1. Atlantic bottlenose dolphin**
1987–1988
About 2,500
Atlantic coast

**2. Baikal seal**
1987–1988
10,000 or more
Lake Baikal, Siberia

**3. Harbor seal**
1988
18,000 or more
Northwestern Europe
(Denmark, Sweden, Norway, Netherlands, Germany, UK, Ireland)

**4. Striped dolphin**
1990 first wave
1991–1992 second wave
"thousands"
Mediterranean coast
1. Spain throughout western Mediterranean Sea
2. Eastward to southern Adriatic and Ionian Seas, Sicilian Channel, southern Tyrrhenian Sea, coasts of Greece and Turkey

**5. Caspian seal**
1997
"thousands"
Caspian Sea

**6. Mediterranean monk seal**
1997
About 200
(50 percent of last remaining colony of this endangered species)
West Africa's Mauritian coast

**7. Caspian seal**
2000
About 20,000
Caspian Sea

**8. Harbor seal**
2002
About 750 initially (total unknown)
Northwestern Europe
(Denmark, Sweden, Netherlands)

**Figure 2.11** Examples of recent marine mammal mass mortality events due to infection by morbilliviruses.

Extensive mortality has been associated with some of these pathogens, especially infections caused by closely related viruses of the morbillivirus complex (Fig. 2.11).

The first recognized occurrence of a morbillivirus epizootic in marine mammals was made retrospectively during a June 1987 to May 1988 mass mortality of Atlantic bottlenose dolphins along the eastern seaboard of the USA.[109] An estimated 50 percent of the in-shore population of this species died,[110] causing an unprecedented population loss in recent history.[111] Population recovery could take up to 100 years.[112] Several months after the onset of that event, a mass mortality of Baikal seals occurred in Lake Baikal, Siberia. An estimated 10 percent of the total population of this species died.[113] The virus involved was found to be a strain of canine distemper (CDV), a morbillivirus, and might have been introduced into the seal population by **feral** or domestic dogs.[114]

Northern Europe experienced its first marine mammal morbillivirus epizootic during the spring of 1988. More than 18,000 harbor seals and a few hundred grey seals died from a newly recognized morbillivirus that was designated as phocine distemper virus.[115,116] Mortality reached 25 percent of the seal population in large areas[117] and was estimated to be as high as 60 percent in some areas.[113] This event was the first identification of morbillivirus as a cause of an active epizootic in marine mammals. The findings provided a focus for evaluation of mass mortality events that followed, and for retrospective evaluations, such as the Atlantic bottlenose dolphin mortality of the previous year.[118,119] An unusual southern movement of harp seals, possibly in response to food shortage, was thought to be the source of the virus introduced into harbor seals.[120]

The Mediterranean Sea was the next reported site of marine mammal mortality due to morbillivirus infections. More than 1,100 striped dolphin carcasses were recovered from the thousands of dolphins estimated to have died. That event began in 1990 and a second wave of mortality followed in 1991.[104,121–124] The dolphin morbillivirus (DMV) causing this event, like the porpoise morbillivirus (PMV), is a newly

recognized morbillivirus.[108] Relatively small-scale mortality events due to morbillivirus infections followed in 1993–1994 in Atlantic bottlenose dolphin in the USA portion of the Gulf of Mexico[125] and in common dolphin, during 1994, along the Crimean coast of the Black Sea.[126]

The next major epizootic occurred in the Caspian Sea of the former Soviet Union. Thousands of Caspian seals died during the spring of 1997 from a strain of CDV that was different from the strain isolated during the mass mortality event at Lake Baikal a decade earlier.[127] That same year approximately 200 of the 270 endangered Mediterranean monk seals living in a pair of caves on West Africa's Mediterranean coast died, apparently from morbillivirus infection (virus isolated but lesions absent).[128] This colony is the sole remaining population in the wild except for scattered small groups of about 20 animals each.[129] Repeat morbillivirus epizootics struck the seal populations of the Caspian Sea in 2000 and Northwestern Europe in 2002.

About 20,000 Caspian seals died from a strain of CDV that was isolated from a Caspian seal in 1997. The origin of the virus is unknown but anecdotal reports of contact between these seals and terrestrial **carnivores** of the region provide a plausible pathway for virus introduction.[127,130,131] The mass mortality in 2000 was the second major epizootic within a 5-year period and of great concern relative to the long-term survival of Caspian seals, a species identified by the World Conservation Union as being vulnerable to extinction.[131] The 2002 reappearance of PDV in harbor seals off the coasts of Denmark, the Netherlands, and Sweden is also reason for concern given the magnitude of loss experienced in 1988. Initial mortality reports for the 2002 event indicated about 750 carcasses had been found[132] but little information has been published about this event.

Other than morbilliviruses, influenza viruses are the only other viruses that have been associated with mass mortality of marine mammals. That association has been infrequent and has been limited to events along the New England (USA) coast. An estimated 600 harbor seals (at least 20 percent of the local population) died from pneumonia during 1979 along Cape Cod, Massachusetts. Influenza A virus was isolated from those animals and was attributed to be the cause for that mortality event.[101,133] Smaller scale epizootic also occurred during 1982–1983, 1991, and again during 1992.[101] Although not a cause of direct mortality, findings of papillomaviruses in Burmeister's porpoise has raised concern that the genital warts associated with venereal transmission of these viruses may reduce reproductive success and suppress population numbers.[108]

Brucellosis is the most significant emerging bacterial disease of pinnipeds.[106] Potential impacts on reproduction (i.e., abortion) rather than epizootic mortality of juveniles and/or adults is the concern. Nevertheless, until recently, brucellosis had not been reported as a cause of abortion in marine mammals. Serologic evidence for exposure to *Brucella* spp. first appeared during the early 1980s.[134] *Brucella* spp. was first isolated during 1992 from aborted fetuses from captive bottlenose dolphins at a California military facility. Those animals had been captured from Mexican waters.[135] A long list of marine mammals, including several species of whales, seals, dolphins, and porpoises, in addition to a river otter, have now been found to be exposed to *Brucella* spp.[106,134,135]

In 1970, leptospirosis emerged as a cause of epizootic mortality in California sea lions dying along the Oregon and California coast. Repeated epizootics of this bacterial disease have occurred between 1981 and 1994.[136] Several hundred animals were involved during each of the earliest events and lesser numbers since then.[137]

Numerous other emerging infectious diseases have appeared in marine mammals but have not resulted in documented mass mortality events.[106] Included are diseases of bacterial;[102,103] fungal;[103,138] rickettsial;[139] parasitic;[140] and viral origin.[105,108] A substantial number of these diseases, such as brucellosis, tuberculosis, and *Erysipelothrix*, are of zoonotic concern (Box 2–9 and Table 2.5). Some of those same diseases and others such as the marine caliciviruses are of economic concern because of their potential transfer to livestock.

Disease in the California sea otter (Box 2–10) is especially noteworthy because of the recent emergence of infectious disease as a factor inhibiting population recovery for this species.[141] Zoonoses are among the diseases found.

### Marine Birds

Globally, a wide variety of diseases have been associated with avian mortality, including birds within the marine environment. Remote areas such as Antarctica and the Galapagos Islands off the coast of Ecuador have been impacted in addition to other areas. Disease emergence is thought to be a factor in the major decline of common eider populations since the late 1980s in the Gulf of Finland.[8,142]

Mass mortality disease events on breeding colonies and other epizootics of disease along migrational routes and on wintering areas are taking a heavy toll on birds within marine environments. Avian cholera (*Pasteurella multocida*), a prominent infectious bacterial disease of **poultry** serves as an example. During the past two decades, major outbreaks of avian cholera have struck wild bird populations in marine environments of Europe, Africa, Antarctica, and North America (Table 2.6). Collectively, these events clearly illustrate the emergence of avian cholera as a mortality factor in marine birds, in addition to its impact on birds in freshwater environments.[143]

Infectious bursal disease (IBD) is a disease of domestic poultry[147] that appears to be emerging in marine birds. Exposure to IBD has been documented by the finding of antibodies to the causative viral agent (IBDV) in sera collected from Emperor and Adelie penguins in Antarctica,[144,145] from spectacled eiders nesting in a remote area of western Alaska,

**Table 2.5.** Marine mammals known to harbor pathogens that have caused disease in humans.

| Disease | Agent | Primary marine mammals affected[a] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Whales | Porpoises | Dolphins | Seals | Sea lions | Sea otters | Walrus | Polar bear | Manatee |
| Poxvirus infection | Virus | ● | ● | ● | ● | ● | ○ | ○ | ○ | ○ |
| Influenza | Virus | ● | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Calicivirus infection | Virus | ● | ○ | ● | ● | ● | ○ | ● | ○ | ○ |
| Brucellosis | Bacteria | ● | ● | ● | ● | ● | ● | ● | ● | ● |
| Erysipelothrix | Bacteria | ● | ● | ● | ● | ○ | ○ | ○ | ○ | ○ |
| Leptospirosis | Bacteria | ○ | ○ | ○ | ● | ● | ○ | ○ | ○ | ○ |
| Mycobacterial disease | Bacteria | ● | ○ | ● | ● | ● | ○ | ○ | ○ | ○ |
| Mycoplasmosis | Bacteria | ○ | ○ | ○ | ● | ● | ○ | ○ | ○ | ○ |
| Salmonellosis | Bacteria | ● | ○ | ● | ● | ● | ○ | ○ | ○ | ○ |
| Vibriosis | Bacteria | ● | ○ | ● | ● | ● | ○ | ○ | ○ | ○ |
| Q fever | Rickettsia | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Lobomycosis | Fungus | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ |
| Trichinosis | Parasite | ●[b] | ○ | ○ | ○ | ○ | ○ | ● | ● | ○ |

● Animal harbors pathogens; ○ Animal does not harbor pathogens

[a] Not all species infected have been associated with the transmission of these diseases to humans (see Box 2–8).

[b] Beluga whale meat associated with human cases in Greenland, but occurrence in whales is infrequent.[454]

## Box 2–9

## Marine Mammals and Zoonoses

Marine mammals are some of the world's most charismatic wildlife and are also an important source of food and other needs of native peoples. These factors result in direct interfaces between humans and marine mammals. Responses to stranded marine mammals, associated on-site rescues, and rehabilitation programs are common ways humans can have contact with animals disabled due to various causes, including infectious disease. Also, the rearing and maintenance of cetaceans and pinnipeds in captivity for performance behaviors and other attributes provide potential exposure by their handlers and veterinarians to infectious diseases. Subsistence and cultural uses involving the harvesting, processing, and consumption of meat from seals and other species are additional contact situations.

Nevertheless, despite the variety of human interfaces with marine mammals, the transmission of infectious disease to humans has been minimal. Differences in the strains of some of the pathogens that affect marine mammals and those that affect humans are factors. However, disease transmission does occur and infectious disease emergence and resurgence in marine mammals are accompanied by a variety of causes of human disease acquired from these species.

### Viral Diseases

To date, viral diseases of marine mammals have not been a major source for human disease. Sporadic cases of several well-established marine mammal viral diseases have occurred and will continue to do so. Whether or not these agents will become a greater source for human disease and whether or not novel viral diseases will emerge over time due to environmental change are yet to be seen.

- Poxviruses—It is generally accepted that humans may acquire parapoxvirus infections through contact with seals,[437] but little documentation exists. In one instance, isolated, self-resolving lesions appeared on the hands of two of three people handling infected grey seals.[438]
- Calicivirus—A growing body of circumstantial evidence points to the zoonotic potential of caliciviruses that are associated with handling infected animals.[439] Deep skin lesions have appeared in a laboratory worker conducting San Miguel sea lion virus studies and antibody responses to this virus have been detected among coworkers.[437] Also, there is a possible 1974 case involving "blisters on the eyes" of a biologist that handled a northern fur seal with flipper lesions suggestive of San Miguel sea lion virus.[439]

The greatest significance of marine caliciviruses is not their low zoonotic potential or their impacts on marine mammals. Instead, it is their role as a significant pathogen of terrestrial livestock. Calicivirus serotypes circulating in southern California marine populations during the 1930s to 1950s are thought to be the origin of outbreaks of vesicular exanthema of swine that swept across the USA. Eradication of this disease from the USA was accomplished by 1956 after expenditures in direct costs of $39 million.[439,440] The host range for these viruses is now known to encompass aquatic and terrestrial mammals, ocean fish, reptiles, amphibians, and insects,[439] in addition to humans.[440]

- Influenza—Harbor seals are associated with human influenza and have caused localized infection in people handling these animals. In one situation, a handler's eyes became infected following a sneeze by an infected seal; other conjunctival infections have also followed known contamination of the eyes.[441] Recent findings indicate that seals serve as reservoirs for influenza B viruses that have circulated previously in the human population.[442] Also, seals may have a role in genetic reassortment of influenza A viruses; those viruses capable of infecting and replicating in seals may be more adapted to mammalian than to avian hosts.[101]

Photo by Milton Friend

## Bacterial Diseases

The great majority of bacteria isolated from marine mammals are not a public health concern. Nevertheless, a few problem pathogens exist and many others are capable of infecting persons with compromised immune systems. Bite wounds are a common means for human infections by marine mammals,[437] and virtually dozens of potential infectious bacteria found in marine mammals have the potential to be transmitted to people by this means. Wound infections following close contact with marine mammals also broadens the potential for transmission of bacterial diseases from marine mammals to people.[103]

- Brucellosis—Beginning in the 1990s, there have been increasing reports of isolations of *Brucella* spp. from marine mammals and increased serological evidence of exposure to *Brucella*. These reports are from the United Kingdom, the USA, Canada, Norway, and Antarctica.[102] A case in a researcher working with *Brucella* strains recovered from marine mammals[443] and two community-acquired human infections with marine mammal-associated *Brucella* spp.[444] emphasize the potential zoonotic aspects of these organisms.[102] Recent isolations from stranded seals at necropsy, documented abortion in dolphins, and other findings strongly suggest the need for caution when handling animals in rehabilitation centers, working on seal rookeries, and the potential for exposure of native peoples who use seals as a food source.[102,103,106,135,437]

- Erysipelothrix—The potential for human infection by *Erysipelothrix rhusiopathiae* from infected marine mammals should not be underestimated. This organism has been isolated from the teeth or gums of elephant seals and northern fur seals, from tissues of stranded pinnipeds and cetaceans, and from many species of captive cetaceans.[102] Isolations also have been made from 12 of 116 bite wounds in handlers of marine mammals.[445]

- Leptospirosis—Veterinarians and others contacting tissues and fluids from infected animals during necropsy have become infected by *Leptospira interrogans*.[437] Transmission to humans can occur by contaminated water, urine, and tissues,[103] thereby, providing multiple potential routes for exposure at wildlife rehabilitation facilities.

- Tuberculosis—*Mycobacteria* spp. are the causative agents of several types of infections, including tuberculosis in humans and animals. Not all *Mycobacteria* cause the type of infection known as tuberculosis nor are all species within this genera of bacteria pathogenic for humans. Reports of tuberculosis in pinnipeds have increased during recent years; isolates from captive pinnipeds (1985–1986), wild pinnipeds (1989–1991), and an infected seal keeper (1988) are all identical. This is a unique strain of *M. bovis* that should be considered part of the *M. tuberculosis* complex.[564] In addition, a seal trainer had a unique strain of *M. bovis* that also was isolated from seals that died of tuberculosis.[446] Cases of cutaneous mycobacteriosis in a manatee and its handler have been attributed to *M. chelonei*.[437] Reports from the 1970s indicate that a dolphin trainer developed *M. marinum* after a dolphin bite[447] and an additional human infection developed after a seal bite.[446]

- Mycoplasmosis—"Whale finger" and "seal finger" are long-standing occupational maladies. The causative agent(s) have been elusive for decades despite the common occurrence of these conditions among whalers and sealers;[448] the Canadian Inuit and others living along coastal Canada, including the Maritime Provinces;[449] and among seal trainers.[450] A 1950 survey of a Norwegian sealing fleet disclosed over 10 percent of the individuals with cases of seal finger.[450,451]

Photo courtesy of the U.S. Fish and Wildlife Service

A 1990 case resulted in the isolation of *Mycoplasma phocacerebrale* from the front teeth of a healthy seal and from the finger of a woman bitten by the seal. These findings suggest that *Mycoplasma* is the cause of seal finger. The organism isolated in the 1990 case was first isolated in 1988 from diseased seals involved in the morbillivirus epizootic in the North and Baltic Seas. Also, a biologist contracted "seal finger" from the mouth of a sedated polar bear he handled.[449] Seals and polar bears are the only known causes of *M. phocacerebrale*.[437]

- Vibriosis—A variety of pathogenic species of vibrios that are known to cause severe or fatal infections in humans are present in the marine environment. Some of these organisms are frequently encountered in cetaceans and less commonly in pinnipeds. Human exposures to these organisms most commonly occur through the ingestion of raw shellfish and by physical contact with marine waters with elevated levels of these bacteria.[437] The greatest human risks for infection by marine mammals is through wounds and abrasions in

the skin of people handling tissues from infected animals.

- Salmonellosis—Various species of *Salmonella* have been isolated from cetaceans and pinnipeds.[103] The greatest risks for human disease appear to be associated with consumption of meat from these animals. An overall attack rate of 40 percent occurred during one event in an Alaskan Eskimo village of 265 people. Whale meat was the source of the *S. enteritidis* gastroenteritis outbreak in that village. Whale meat has been implicated in other foodborne epidemics including an event in Japan in which 172 of 178 people who ate the meat became ill from *S. enteritidis*. Other events have occurred in Alaska and in Greenland.[452]

## Rickettsial Diseases

Rickettsia are a specialized type of bacteria typically found in the gut of **lice**, fleas, ticks, and mites that vector the transmission of a variety of diseases caused by these microscopic life forms. Typically, rickettsia have not been associated with disease in marine mammals nor have marine mammals been a source for human infections.

- Q Fever—*Coxiella burnetii*, the causative agent of Q fever, was isolated in 1998 from the placenta of an adult female Pacific harbor seal at necropsy (seal died of other causes). This finding is the first record of infection by *C. burnettii* in a marine mammal and raises concern about the potential for zoonotic transmission to wildlife rehabilitation center workers that may be exposed to placental tissues or newborn seal pups.[139]

## Fungal Diseases

Direct transmission of fungal diseases from marine mammals is infrequent to rare because the vegetative stages of fungi generally found in diseased marine mammals are usually not infective for humans.[437] However, dermatomycosis (fungal infections of the skin) and some other fungal diseases may be transmitted during close contact situations.[103]

- Lobomycosis—Lobo's disease is a cutaneous-subcutaneous chronic granulomatous disease resulting from infection by the fungus *Loboa loboi* (*Lacazia loboi*). This disease has primarily been reported in people living in Central and South America, especially the Amazon region of Brazil. Infection also occurs in bottlenose dolphins from the Atlantic Ocean and the Gulf of Mexico.[453] A single instance of direct transmission from an infected dolphin to a dolphin handler was documented during the 1980s.[437]

*Photo courtesy of the U.S. Fish and Wildlife Service*

## Parasitic Diseases

In general, parasites of marine mammals are not an important source for human infections. However, native peoples and others that use marine mammals for food may become infected with several species of nematodes (roundworms), such as hookworm and anisakis. Polar bear and walrus meat are potential sources of trichinosis because these species are part of the sylvatic cycle for *Trichinella* in the Arctic.[140,454]

# Box 2–10 Infectious Disease and the Southern Sea Otter

Photo by Milton Friend

The southern sea otter is a California coastal species listed as threatened by the U.S. Fish and Wildlife Service in 1977. After experiencing steady but slow population growth since the late 1970s, in 1995 their population declined substantially, causing a reversal in plans for delisting this species from threatened status.[455] Disease emergence is an important source of mortality for this species and appears to be a factor retarding population recovery.

Sea otter mortality was investigated by the California Department of Fish and Game beginning in 1968. By 1989, nearly 1,700 carcasses had been evaluated, and more than half died from undetermined causes. Beginning in 1992, supplemental evaluations consisting of about 50 sea otter carcasses per year for 5 years were necropsied by pathologists at the National Wildlife Health Center (NWHC) and associated laboratory analyses were conducted to determine the causes of death. In contrast to the findings from 1968–1989, infectious disease was found to be the primary cause of death.[141] Nearly 40 percent of the sea otters necropsied at the NWHC died from parasitic, fungal, or bacterial infections.[455]

Acanthocephalan parasites (*Polymorphus* spp.) are the most common cause of death. Historic evaluations indicate that, in the past, the parasites causing this mortality were only found in small numbers within individual animals and that few otters were infected by these parasites. An increasing number of sea otters have now acquired large numbers of *Polymorphus* spp., along with the nonpathogenic species of acanthocephalans (*Corynosoma enhydri*). The findings of protozoal encephalitis and the fungal disease coccidioidomycosis[141] are somewhat unexpected. *Toxoplasma gondii* and *Sarcocystis neurona* are the protozoan parasites associated with the encephalitis.[456,457] Generally, both agents are associated with terrestrial rather than aquatic species.

Toxoplasmosis is typically a zoonosis associated with cats. Infections are believed to result from cat feces containing *T. gondii* oocysts that enter the marine environment through stormwater runoff. Otters eat invertebrates (i.e., mollusks) that may have ingested the oocysts.[456] This supposition is supported by the finding that otters sampled between 1997 and 2001 near areas of maximal freshwater runoff into the marine environment were about three times

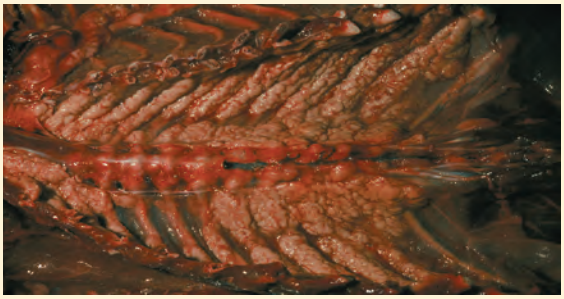


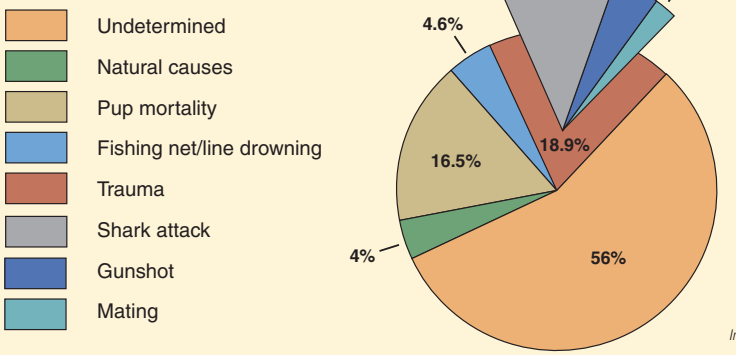
Photo by James Runningen

Coccidioidomycosis infection.

Photo by James Runningen

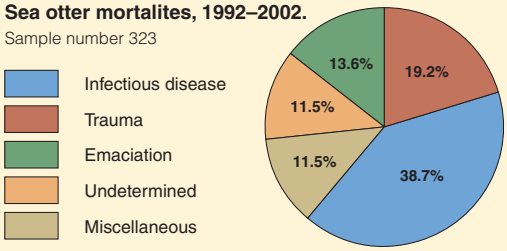
Acanthocephalan infection.

**Sea otter mortalities from 1968–1989.**[141]

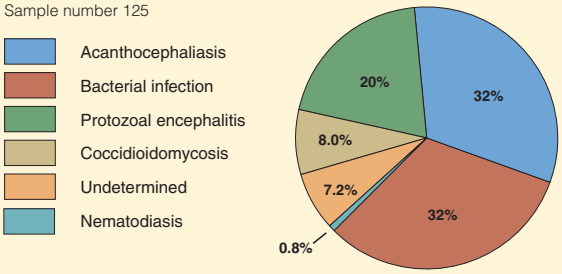



*Information from the NWHC*

**Sea otter mortalites, 1992–2002.**
Sample number 323




**Sea otter infectious disease mortalites,1992–2002**
Sample number 125




more likely to be seropositive for *T. gondii* than otters sampled in areas of low flow.[458] A similar pathway involving opossum feces containing *S. neurona* sporocysts is thought to be the route for infection of that parasite.[457,459]

Coccidioidomycosis is also a land-based disease of mammals. The causative fungus, *Coccidioides immitis*, is found in soil and is the source of San Joaquin Valley fever of humans. Only one case of this disease had previously been reported in a sea otter (1976) prior to the eight cases diagnosed during the NWHC evaluations.[141] The pathology seen is indicative of inhalation exposure and suggests that wind-borne spores from nearby land areas are the source for these infections.[460]

It is not known how much of the undiagnosed mortality during the period of 1968–1989 was due to infectious disease. Early necropsies were primarily conducted in the field by California Department of Fish and Game marine biologists. Small numbers of those animals were also evaluated under laboratory conditions by physicians and by veterinarians associated with the Monterey Bay Aquarium.[461] It is likely that those individuals would have diagnosed acanthocephalan peritonitis and coccidioidomycosis because of the severity of gross lesions associated with these diseases. Some of the miscellaneous bacterial infections encountered during the NWHC evaluations may not have been detected because of the limited amount of bacteriology done in association with those investigations. Nevertheless, it is reasonable to assume that much of the infectious disease encountered in the southern sea otter is of recent origin. Also, it is noteworthy that multiple infectious diseases are involved rather than a single disease, suggesting that environmental changes are providing new opportunities for sea otters to encounter potential disease agents.

**Table 2.6.** Examples of avian cholera *(Pasteurella multocida*) epizootics in marine environments.

| Continent/ country | Geographic area | Primary species affected | Year of inital event | Comments |
|---|---|---|---|---|
| North America | | | | |
| Canada | East coast of Quebec | Common eider | 1964 | Breeding colonies periodically experience epizootics.[324,468] |
| USA | East coast of Maine | Common eider | 1963 | Breeding colonies periodically experience epizootics.[469,470] |
| | Chesapeake Bay | Long-tailed duck (old squaw), white-winged scoter, other waterfowl | 1970 | Large-scale winter and spring epizootics killing tens of thousands of birds every few years (a 1994 epizootic killed more than 80,000). |
| South America | | | | |
| Chile | Iquique | Marine ducks | 1941 | Large-scale epizootic in unspecified marine ducks on beaches of Iquique; lesser numbers of pelicans and loons involved.[471] |
| Europe | | | | |
| The Netherlands | Vlieland | Common eider, herring gull, black-backed gull | 1977 | First occurrence in 1945; heavy mortality in winter populations from 1977 until 1980; breeding colony epizootic in 1984.[472] |
| Denmark | Coast of Hov; Island of Hov Ron, other nearby areas | Common eider, herring gull, other gulls and waterfowl, oystercatcher, cormorant | 1996 | Spring epizootic followed by mass mortalities among breeding female eiders; mortality in affected breeding colonies close to 90 percent of breeding age females.[473,474] |
| Africa | | | | |
| South Africa | Dassen Island | Black-backed gull | 1951 | *Pasteurella aviseptica* (*P. multocida*) outbreak.[475] |
| | Coast of western part of South Africa | Cape cormorants | 1991 | Large-scale mortality event that killed more than 14,500 adults; 16 percent mortality of breeders on Dassen Island and 8 percent overall for the 8 islands involved.[476] |
| Antarctica | Palmer Station | Brown skua | 1979 | See citation.[477] |
| New Zealand | Campbell Island | Rockhopper penguin | 1985 | Chicks are primary age-class impacted; avian cholera found in four separate colonies on the Island; first report of this disease in this species.[478] |

and in nesting common eiders and herring gulls in the Baltic Sea.[146] Investigators were determining the causes of mass mortalities and population declines in those species when they discovered the presence of IBDV. In chickens infected at an early age, IBDV causes severe, prolonged immunosuppression.[147] A similar host response in wild birds would enhance their susceptibility to other infectious diseases in addition to any direct mortality resulting from IBD.

The long-tailed duck is also experiencing population declines. Investigators isolated an adenovirus from dead and live ducks while investigating mortality of this species in 2000 in Alaska.[148] They found evidence of a greater frequency of exposure to the virus in live ducks at the mortality site versus those at a reference area, suggesting the virus was closely associated with the mortality event investigated. The role of the virus in relation to the decline of long-tailed ducks in Alaska since the 1970s is unknown.

The causes for some mass mortality events of marine birds remain unknown even though they are recurring and the subject of considerable investigation. Numerous examples can be found in the wildlife mortality databases of the U.S. Geological Survey's National Wildlife Health Center. For example, during the winter of 2000, several hundred Atlantic brant died along the New Jersey coast. Despite intensive field and laboratory investigations, the cause for this mortality and a similar event that followed could not be determined. Similar results have been associated with repeated **seabird** mortality off the coast of Alaska, Washington and Oregon, for loon mortality along the Florida Gulf coast, and for a number of other large-scale mortality events.[16]

### *Freshwater Aquatic Environments*

#### Amphibians

Disease emergence and reemergence in amphibian populations (Fig. 2.12) has received recent attention because of the global distribution of mass mortalities;[11,149–153] associations drawn between environmental quality for humans and amphibian health status;[12] media coverage of amphibian deformities;[154] and debate within the scientific community relative to the role of disease versus other factors in amphibian population declines.[152,155] The global distribution of diseases as a cause of amphibian mass mortalities establishes diseases as a contributing factor to population declines and to the disappearance of amphibian species. The magnitude of amphibian population declines is such that this problem has been identified as one of the most important emerging wildlife conservation issues of the latter part of the 20th century.[12]

The fungal disease chytridiomycosis and infections by ranaviruses are commonly associated with mass mortalities of amphibians[12] but are not the only emerging diseases involved (Table 2.7). Chytridiomycosis (Fig. 2.13) is caused by infection of the skin with chytrid fungi.[11] This class of fungi is ubiquitous in nature and has important functional roles in ecosystem dynamics.[149] The fungi causing disease

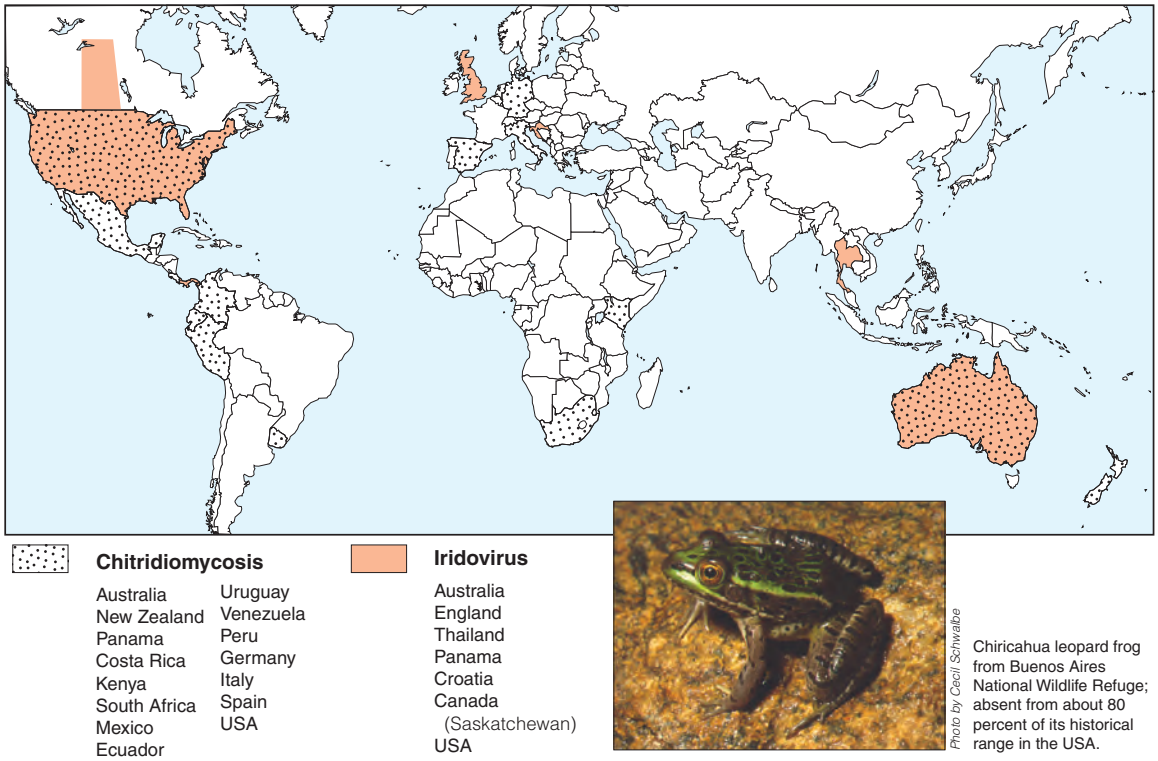



**Figure 2.12** Reported global distribution of chytridiomycosis and ranaviral (iridovirus) disease in wild amphibian populations.[12]

**Table 2.7.** Emerging and enzootic infectious diseases of amphibians (contributed by D.E. Green, U.S. Geological Survey).[a]

| Disease/agent | Type | Date first observed | Primary hosts | Geographic area | Comments |
|---|---|---|---|---|---|
| Frog virus-3 (FV-3) and tadpole edema virus (TEV) | Ranavirus | c. 1963 | Northern leopard frog, bullfrog, wood frog, spotted salamander, others | Upper Midwest and Eastern USA | • FV-3 first isolated amphibian ranavirus and type-species of the genus; isolated from cancerous renal tissue but not associated with morbidity or mortality.[479]<br>• TEV first ranavirus associated with mass mortality of amphibians;[480] numerous large die-offs of more than 10 species from Maine to Minnesota from 1995–2002.[157,565] |
| Redwood Creek ranavirus | Ranavirus | 1991 | Northern red-legged frog (tadpole), stickleback fish | California, USA | • Three virus isolates associated with sick and dying tadpoles and fish over 3 years.[481] |
| Tiger salamander viruses | Ranavirus | c. 1995 | Tiger salamanders | Western USA and Saskatchewan, Canada | • Recurring mass mortality of larval and neotenic salamanders in cattle tanks,[482] reservoirs, and lakes;[483] die-offs may kill thousands at a site.[565] |
| Bohle iridovirus (ranavirus) | Ranavirus | c. 1989 | Ornate burrowing frog | Queensland, Australia | • Die-off of young captive frogs.[484] |
| Unnamed English ranaviruses | Ranavirus | c. 1993 | Common European frog | England | • Recurring widespread die-offs of adult frogs in garden ponds.[485] |
| Pig frog ranavirus | Ranavirus | c. 2000 | American pig frog | China | • Recurring mass mortalities of captive pig frogs raised for food.[486] |
| Other ranaviruses (isolates)[b] | Ranavirus | 1996–2002 | Numerous species | USA (10 states) | • Numerous, occasionally recurring, mass mortality events in larvae of more than 10 species of true frogs, chorus frogs, mole salamanders, and newts,[157,487] often killing thousands. |
| Chytridiomycosis (chytrid fungus) | Fungus | c. 1974 | Frogs, toads | Global | • First identified in late 1990s in zoo and free-living frogs and toads in USA, Panama, and Australia; recently detected in museum animals involved in die-offs in mid-1970s.[11,151]<br>• Causes rapid population declines even at pristine sites; occurrence corresponds temporally with onset of global amphibian population declines and some amphibian extinctions.[157]<br>• Molecular studies indicate global isolates are very similar, indicating it is a newly emerged pathogen.[488] |
| Watermold infection (Saprolegniasis) | Fungus | c. 1993 | Toad eggs | Oregon, USA | • Cause of massive destruction (1 year's production) of toad eggs;[439] rapidly invades infertile and dead eggs killed by other agents. |
| *Ichthyophonus* infection (Mesomycetozoa) | Fungus | c. 1983 | Frogs, newts, salamanders | Eastern USA, Canada | • Primarily a fish disease, but causes three types of disease in amphibians: inapparent infections, swollen rumps in frogs and newts, and rarely, deaths in adult frogs.[565,566] |

**Table 2.7.** Emerging and enzootic infectious diseases of amphibians (contributed by D.E. Green, U.S. Geological Survey)[a]—Continued.

| Disease/agent | Type | Date first observed | Primary hosts | Geographic area | Comments |
|---|---|---|---|---|---|
| *Dermosporidium* infection (Mesomycetozoa) | Fungus | c. 1980 | Toads | Eastern USA and California | • Nonlethal (so far) infection of adult toads causing skin pustules; may be related to *Dermocystidium* of European amphibians.[151,565,567] |
| *Dermocystidium* infection (Mesomycetozoa) | Fungus | c. 1910s | Frogs, toads | Europe | • Apparently nonlethal infection causing skin pustules.[568] |
| Chlamydiosis | Chlamydia | 1980s? | African clawed frogs | Global? | • Lethal and occasionally nonlethal disease of captive clawed frogs associated with feeding raw beef liver; one report in free-living giant barred frog in Australia.[490] |
| *Perkinsus*-like infection (taxonomic status uncertain) | Protozoa? | 1999 | Tadpoles of true frogs | Alaska, Virginia, Minnesota, Mississippi, North Carolina, New Hampshire, USA | • Newly identified emerging lethal systemic infection of tadpoles only; often associated with massive die-offs.[565]<br>• Molecular sequencing places organisms with the oyster pathogen *Perkinsus* sp. |
| Anchorworms | Copepod | Unknown | Bullfrog tadpoles | Global? | • Primarily a parasite of fish; also an infrequent cause of morbidity and mortality in larval and adult amphibians.[158] |
| Leeches | Leech | Unknown | Frogs, toads, salamanders | Australia, Canada, Germany, USA (probably global) | • Usually innocuous, but may kill tadpoles in USA and Canada; associated with leg malformations in Germany. |
| *Ribeiroia* infection | Fluke | 1990s | Frogs, toads, salamanders | USA, Canada | • Immature stages (cercaria) kill large numbers of captive tadpoles but deaths in free-living tadpoles have not been diagnosed; major cause of leg malformations in frogs and the only known cause of extra legs.[158,492] |
| Malformations | Multiple | 1995 | Frogs, toads, salamanders | USA, Canada | • Unusually high prevalence of deformed frogs were found in most states after initial media reports in 1995.[154,565]<br>• A wide range of deformities found;[154,159] main cause in Americas is the parasite, *Ribeiroia* sp.[491,492] but ultraviolet light causes leg deformities in experiments.[493]<br>• Almost no chemicals have been properly tested experimentally to determine their role in malformations. |

[a] National Wildlife Health Center, U.S. Geological Survey.

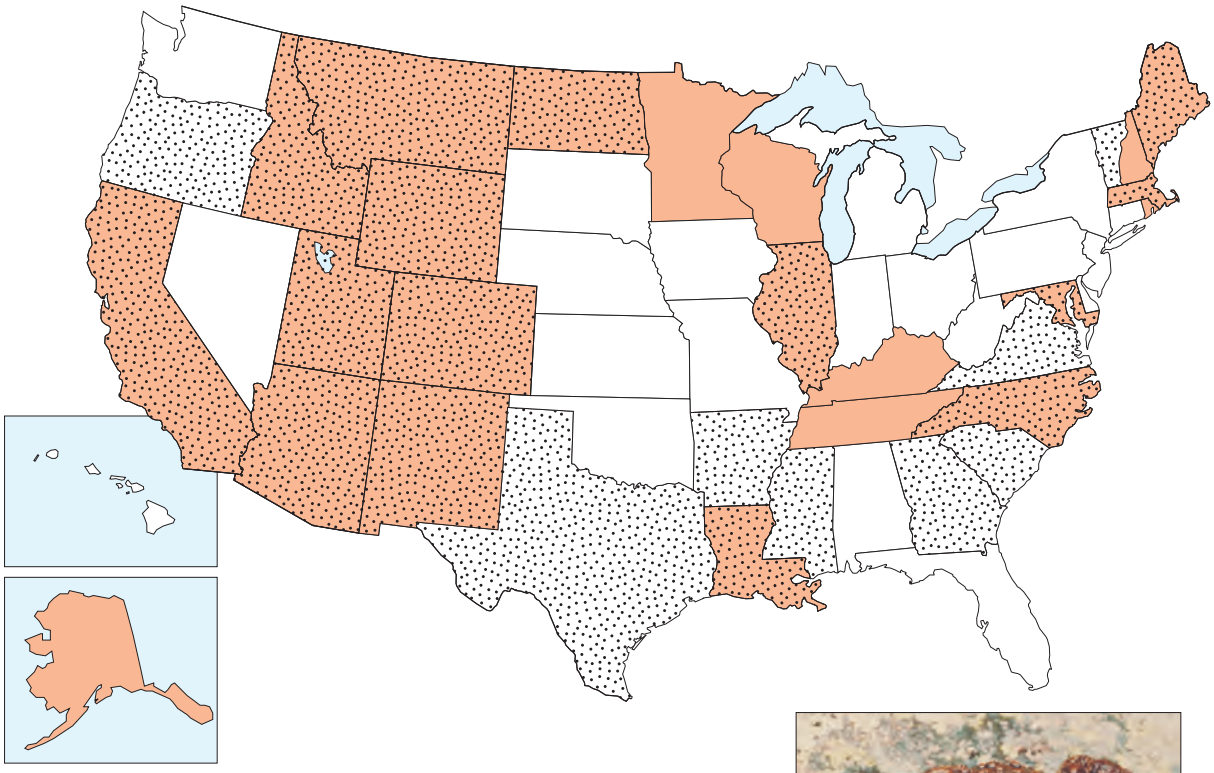
[b] Isolates differ from tiger salamander virus and FV-3 by restriction fragment length polymorphism analysis.

USGS file photo

**Figure 2.13** A tiger salamander with a skin ulcer caused by iridovirus.

in amphibians have been placed in a new genus, *Batrachochytrium*,[156] and have been responsible for mass mortalities of amphibians on several continents.

Chytrid epizootics have occurred at numerous locations within the USA (Fig. 2.14) and because of their insidious nature may easily be overlooked.[157] The causative agent, *B. dendrobatidis*, has been reported in more than 75 species of amphibians captured worldwide in the wild. Forty-seven of those species are from Australia. Other reports are from Europe, Africa, South America, Central America, and North America.[150] It is not known what has triggered epizootics of this disease; however, experimental studies have resulted in 100 percent mortality in conditions where uninfected amphibians remained healthy, thereby suggesting that predisposing immunosuppression is not a requirement for disease in individual animals or for epizootics.[149]



**Chytridiomycosis**

Massachusetts, Maine, Vermont, Maryland, Virginia, North Carolina, South Carolina, Georgia, Illinois, North Dakota, Colorado, Utah, Idaho, Wyoming, Arkansas, Texas, Louisiana, New Mexico, Arizona, Montana, California, Oregon

**Iridovirus**

Maine, New Hampshire, Massachusetts, Rhode Island, Maryland, North Carolina, Kentucky, Tennessee, Minnesota, Wisconsin, Illinois, North Dakota, Montana, Colorado, New Mexico, Utah, Idaho, Wyoming, Arizona, Louisiana, California, Alaska

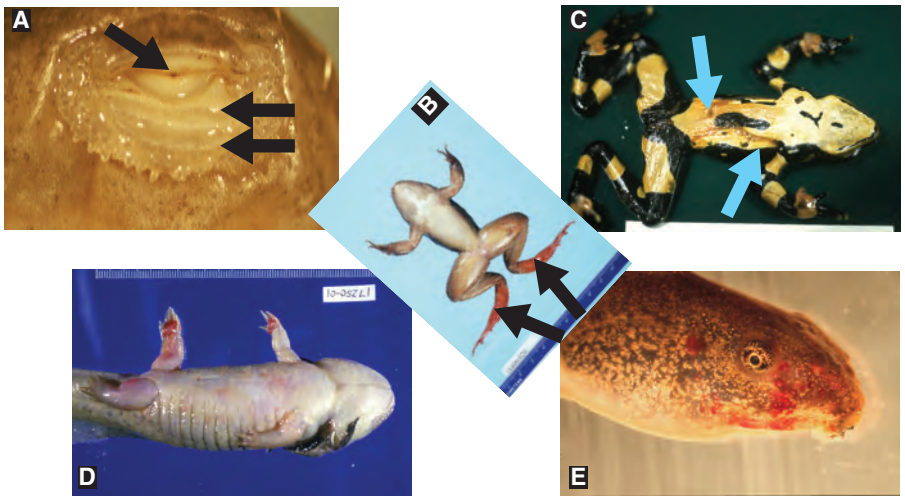
Photo by Stephen Corn

Boreal toad from Rocky Mountain National Park, Colorado; the species has undergone about an 80 percent decline in the southern Rocky Mountains.

**Figure 2.14** Reported distribution as of July 2004 of chytridiomycosis and ranaviral (iridovirus) disease in wild populations of amphibians in the United States. (USGS National Wildlife Health Center data; D.E. Green, personal communication.)

Epizootics due to infection by iridoviruses (Fig. 2.15) also have been the cause of mass mortalities of amphibians. These viruses have been isolated from amphibians from 20 states within the USA (Fig. 2.14). Viruses within the genus *Ranavirus* of the family Iridoviridae are the agents involved in these mortalities. These often highly virulent agents cause systemic infections in amphibians. Tadpoles appear to be the most susceptible developmental stage for ranavirus infection and death rates of 100 percent may occur. A variety of viruses, rather than a single agent, are involved (Table 2.7). Some of these viruses appear to be newly emerging as causes for amphibian mortality, while others have been known for some time and are reemerging. Recent **translocations** by humans of amphibians or their egg masses and larvae may be responsible for the dissemination of ranaviral disease.[12]

In addition to the diseases just noted, there is widespread recognition of amphibian malformations (Fig. 2.16). In 1995, a student field trip to a Minnesota (USA) pond disclosed numerous **frogs** with malformations. The publicity associated with those and subsequent findings and the investigations that followed resulted in 38 species of malformed frogs and 19 species of **toads** being reported from 44 states in the USA by 2000 (Fig. 2.17).[154,159] Similar findings have been reported from several Canadian provinces. Multiple factors are involved as causes for these abnormalities.[154,159] Despite this broad geographic occurrence, malformations do not appear to be a major cause for amphibian population declines.[12]




All photos by David E. Green

**Figure 2.15** (A) Oral disc of tadpole with chytrid fungus infection showing complete loss of black-pigmented keratin from tooth rows and jaw sheaths. (B) Chytrid fungus infection in adult Chiricahua leopard frog showing hyperemia of the feet. (C) Chytrid fungus infection in harlequin frog showing abnormal molting of skin. (D) Larval tiger salamander with Ranavirus infection showing reddening of ventral skin and marked hemorrhages. (E) Wood frog tadpole with Ranavirus infection showing skin hemorrhages.

All photos by Carol Meteyer

**Figure 2.16** Deformities of northern leopard frogs.

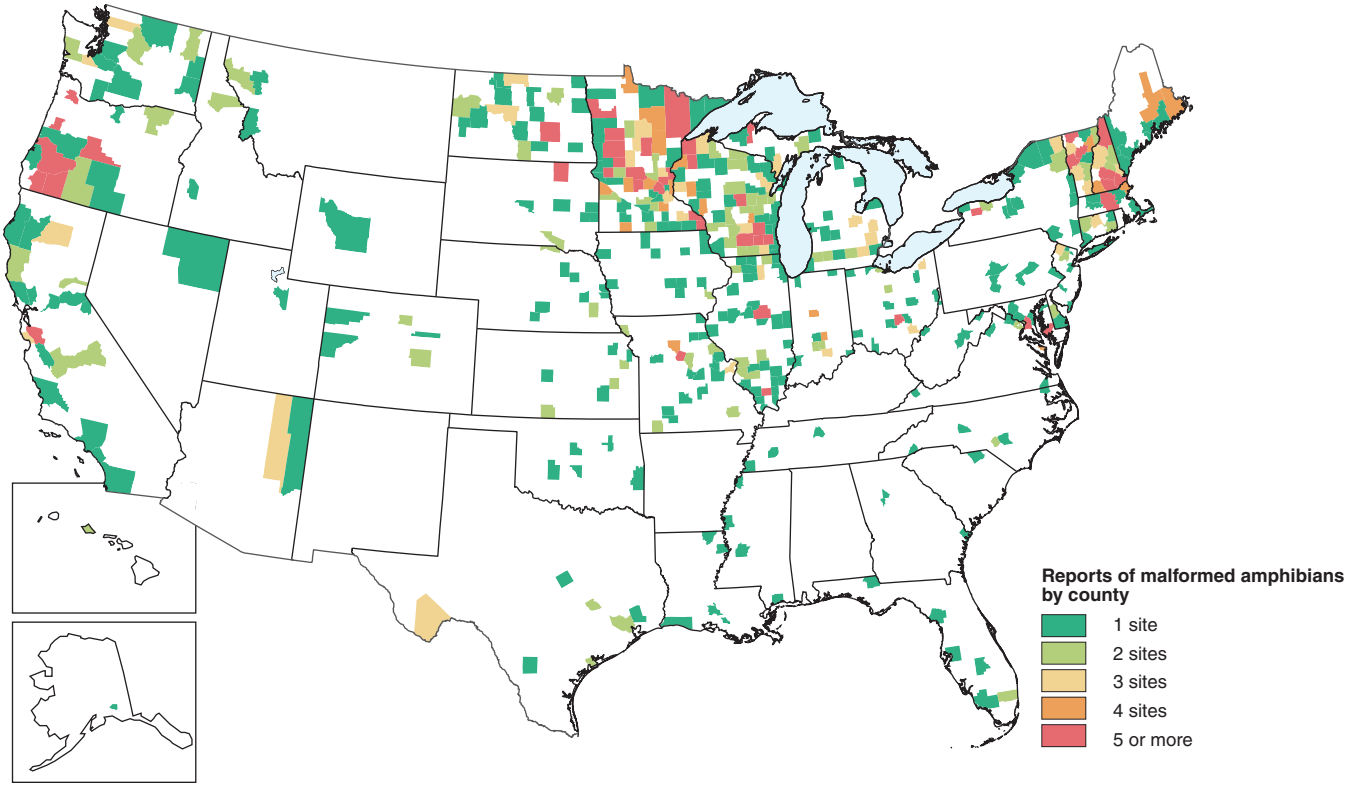



**Figure 2.17** United States counties in which amphibian malformations have been reported as of 2003 (data from North American Reporting Center for Amphibian Malformations, National Biological Information Infrastructure).

### Freshwater Fish

Disease emergence and reemergence are a continual challenge for wild and captive fish populations,[78] and disease often moves freely between both because of human actions associated with the movement of live fish, fish culture, and releases of hatchery-reared fish. For example, the protozoan *Myxobolus cerebralis* causes whirling disease in rainbow trout, but coexists with native European brown trout. Rainbow trout were introduced into Europe near the start of the 20th century[88] and the first cases of whirling disease were seen in 1903.[160] *Myxobolus cerebralis* reached the USA about 1955 in imported frozen trout shipped to Pennsylvania[161] and now affects trout and salmon in at least 21 states.[88] To combat this disease, some fish hatcheries destroy their stock because of the magnitude of losses possible and regulations that prevent the release of fish from infected hatcheries. Whirling disease can decimate wild populations of rainbow trout. In 1994 this disease was introduced into the Madison River in Montana, and the rainbow trout population plummeted from 3,500 fish per mile (1990 evaluation) to 300 per mile by 1994.[162] More recently, whirling disease appeared for the first time in a Wyoming watershed with an important rainbow trout fishery.[163]

Several other notable diseases of **freshwater fish** have also emerged within the USA (Table 2.8). For example, an epizootic disease, spring viremia of **carp**, killed about 10 tons of carp in one lake in Wisconsin.[164] Yellow perch in Wisconsin also have been affected by a new pathogen, the protozoan parasite *Heterosporis* sp.[165]

Aquaculture and hatcheries have been sources for numerous disease introductions and will continue to provide new opportunities for diseases that are brought into those facilities from wild and cultured founder stocks. These diseases accompany fish that are released or escape into the wild from those facilities. Recognition of this problem has resulted in the development of a number of fish health inspection and certification programs for the release of certain types of fishes into some watersheds with the USA, Canada, and other countries. Examples include integrated fish health management in the Great Lakes Basin[166] and the programs of the Office International des Epizooties (OIE). A primary goal for this global organization for animal health is to facilitate international trade in animals and animal products (including aquatic species) while reducing the risk of transfer of serious diseases from one country to another.

**Table 2.8.** Examples of emerging diseases of freshwater finfish within the USA (contribution of F. Panek, U.S. Geological Survey).[a]

| Disease | Type | Period of emergence | Geographic area of concern | Comments |
|---|---|---|---|---|
| Asian tapeworm (*Bothriocephalus acheilognathni*)  | Parasite (cestode) | 1980s | Continental | • Serious exotic parasite that affects many native (USA) cyprinids (minnow family), carp, and some catfish species; probably introduced with grass carp.<br>• Threat to native cyprinids in the Colorado River, including the endangered humpback chub.[494] |
| Spring viremia of carp  | Virus (rhabdovirus) | Late 1990s | North Carolina, Wisconsin, Mississippi River drainage | • Notifiable foreign animal disease to the Office International des Epizooties (OIE).<br>• Serious threat to aquaculture industry and potentially to native fish populations.<br>• North Carolina aquaculture facility first USA documentation; epizootic in wild carp from the Mississippi River in Wisconsin.[495] |
| Koi herpesvirus  | Virus (herpesvirus) | 1998 | Mid-Atlantic States and southern California | • Epizootics involving common carp and koi first appeared in mid-Atlantic area, followed by epizootics the following year (1999) in southern California.[496] |
| Largemouth bass virus  | Virus (iridovirus) | Mid-1990s | 17 southeast and southwest states | • First isolated from largemouth bass from Lake Weir, Florida[501] and linked to largemouth bass mortalities at Santee-Cooper Reservoir, South Carolina.[498]<br>• Transmissible in water and orally.[499] |
| White sturgeon iridovirus  | Virus (iridovirus) | 1988 | Oregon, Idaho, California | • Highly virulent, serious threat to cultured white sturgeon.<br>• First isolated from cultured fish in northern California.[500] |
| White sturgeon herpesvirus  | Virus (herpesvirus) | 1989 | California, Oregon | • Cause of juvenile mortality among intensively reared white sturgeon populations.[501] |
| Whirling disease (*Myxobolus cerebralis*)  | Parasite (protozoan) | 1990s | Occurs in at least 21 states | • Chronic debilitating disease that is generally considered a disease of cultured trout; several "blue ribbon" trout waters in Colorado and Montana severely impacted during 1990s.[162] |

[a] National Fisheries Research Center, U.S. Geological Survey.

**Table 2.9.** Finfish diseases notifiable to the Office International des Epizooties because of their significance as challenges to aquaculture and wild fish stocks.

| Disease | Agent type | Comments |
|---|---|---|
| Epizootic hematopoietic necrosis (EHN) | Virus | • Systemic iridovirus (ranavirus) infection caused by three similar viruses; EHN virus (EHNV) is limited to Australia and the other two viruses (European sheatfish virus, European catfish virus) to Europe.[86]<br>• EHNV remains infective for 100–200 days on dry surfaces and in frozen tissue for more than 700 days.[12]<br>• Susceptible host species for the purpose of the International Aquatic Animal Health Code are redfin perch, rainbow trout, Macquarie perch, mosquito fish, silver perch, and mountain galaxias.[502] |
| Infectious hematopoietic necrosis (IHN) | Virus | • Rhabdovirus with a historic range in western parts of North America; spread to Europe and Far East via importations of infected fish and eggs.[86]<br>• Susceptible host species for the purpose of the International Aquatic Animal Health Code are rainbow/steelhead trout, sockeye, Chinook, chum, yamame, amago, coho, and Atlantic salmon.[502] |
| *Oncorhynchus masou* disease | Virus | • Herpesvirus causing oncogenic and skin ulceration disease; present in Japan and probably coastal rivers of Eastern Asia having Pacific salmon.[86]<br>• Susceptible host species for the purpose of the International Aquatic Animal Health Code are kokanee, masou, chum, and coho salmon, and rainbow trout.[502] |
| Spring viremia of carp | Virus | • Rhabdovirus; in addition to transmission by fish, parasitic invertebrates, including leeches, can transmit this disease from diseased fish to healthy fish.[86]<br>• Until recently, confined to Europe;[502] now in USA.[164]<br>• Susceptible host species for the purpose of the International Aquatic Animal Health Code are common, grass, silver, bighead, and crucian carp, goldfish, tench, and sheatfish.[502] |
| Viral hemorrhagic septicemia | Virus | • Disease agent recently classified as a Novirhabdovirus, a new group of rhabdoviruses; until mid-1980s thought to be confined to hatcheries in Europe. Now known to occur in wild fish stocks and in marine environments of North America, part of the Pacific Ocean, North Atlantic, and the Baltic Sea; marine fish isolates from species other than salmonids.[86]<br>• Susceptible host species for the purpose of the International Aquatic Animal Health Code are rainbow trout, brown trout, grayling, white fish, pike, turbot, herring and sprat, Pacific salmon, Atlantic cod, Pacific cod, haddock, and rockling.[502] |

The Fish Diseases Commission of the OIE has developed a code and manual to guide disease prevention and within that code has developed lists of "notifiable diseases" (Table 2.9) and "other significant diseases." Notifiable aquatic animal diseases are those considered to be of socio-economic or public health importance within countries involved in the international trade of aquatic animals and products. The focus is on diseases with potential to cause serious damage to the aquaculture industries of those countries or their wild populations of fish, mollusks, and crustaceans.[167] Spring viremia of carp recently entered the USA, and is one of the OIE notifiable diseases.

In some instances, transfer of disease agents may occur via birds feeding in hatchery and wild environments. Indigenous and exotic pathogens also will continue to be introduced into fish populations by other means, such as bait fish used by fishermen, contaminated surfaces of boats, ballast water discharges, and unauthorized fish introductions. The significance of disease introduction to farmed and wild stocks of fish only becomes apparent with time[92] despite the explosive expression of disease that may appear in association with initial disease appearances in fish populations. Disease agents released into aquatic environments may not survive for a variety of reasons or they may also adapt to their new environment in unanticipated ways, sometimes as agents of mass mortality or as a source for high levels of chronic **morbidity** and mortality for species they are able to infect. Thus, disease emergence can appear in different forms and at different times following agent entry into fish populations.

Gyrodactylosis is an example of a parasitic disease on the OIE list of Other Significant Diseases that was not recognized as a problem when the causative parasite *Gyrodactylus salaris* was first noted in Swedish hatcheries at the beginning of the 1950s. More than two decades later, following introduction into Norway, this parasite was found to be highly pathogenic for wild and farmed Atlantic salmon **parr** and **smolt** and several other species of salmonids. It has been found in wild populations from rivers in Russia, Sweden, and Norway, and is present in farmed Atlantic salmon and rainbow trout in several Northern European countries.[86]

A host of infectious agents in addition to those already noted have been identified during the past 3 decades as causes of sporadic mortality or reduced body condition of freshwater fish.[82,168,169] In addition, common, well-established diseases of fish culture continue to appear in new geographic areas and to reappear in areas where the disease had been eradicated. For example, the first occurrence of bacterial kidney disease (*Renibacterium salmoninarum*) in Denmark was documented in 1997 in a rainbow trout hatchery.[170] Infectious pancreatic necrosis virus (IPNV) reappeared in Northern Ireland in 2003 for the first time since 1996.[171] IPNV is one of several emerging pathogens causing serious economic damage to aquaculture around the world, including Scotland where the increase in the prevalence of this virus was 10 percent annually at saltwater sites from 1996 to 2001, and 2 to 3 percent at freshwater sites, except for a 6.5 percent increase in Shetland.[172] Tons of hatchery fish are destroyed to combat these diseases, but concerns exist that prior to disease control actions these pathogens already may have spread to wild fish stocks in those areas.

Human alterations of the environment also provide new opportunities for fish pathogens by creating unique habitats with novel host-agent interactions. The primary fish species of the inland saline Salton Sea in California are all exotics, except for the desert pupfish, and include tilapia, typically a freshwater fish, in addition to marine fish.[173,174] The first report of the flagellate ectoparasite *Cryptobia branchialis* in a highly saline waterbody is from the Salton Sea where it causes heavy infection. The gill function of young tilapia fry is affected to the extent that this parasite may be causing significant mortality.[175] Both the parasite and the fish host are introduced species within a human altered and sustained environment.[176]

Little doubt exists about the significance of disease in wild, as well as cultured, populations of freshwater finfish and of the movement of infectious disease between these populations. Also, some serious fish pathogens, such as viral hemorrhagic septicemia, can move between freshwater and **saltwater fish** species. Greater efforts are needed to minimize the potential for such transfers because of the consequences associated with disease emergence. Disease emergence must be considered in conjunction with the development of transgenic fish, which are being proposed to help meet the growing demands for fish.[177–179]

### Waterbirds

Birds in freshwater environments, even if they are only seasonal visitors, have been affected by a broad array of emerging diseases caused by viruses, bacteria, and parasites.[8] Notable viral diseases include duck plague (DP) or duck virus enteritis (DVE) and Newcastle disease (ND). DP is caused by a herpesvirus and only affects waterfowl (ducks, **geese**, and **swans**). This European disease of **domestic ducks** first appeared in North America in 1967. An epizootic within the Long Island, New York (USA) commercial duck industry was accompanied by a small number of deaths in wild waterfowl in close proximity to the commercial duck operations.[180,181] Because of the exotic status of DP and its importance as a disease of domestic waterfowl, a major disease eradication effort was undertaken that included depopulating infected commercial duck flocks. Following an initial period when DP was thought to have been eradicated in the USA, the disease reappeared in captive and feral waterfowl and has gradually spread to many states and several Canadian provinces. The majority of this expansion has occurred during the last two decades of the 20th century (Fig. 2.18). The great majority

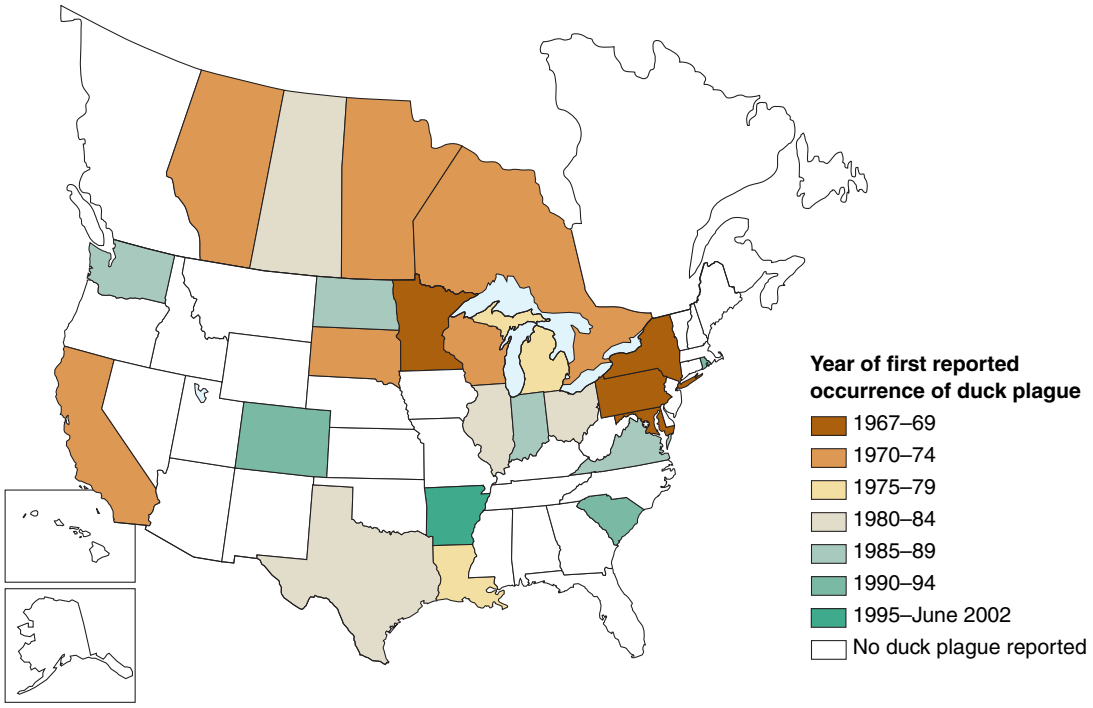



**Figure 2.18** Reported North American distribution of duck plague by period of first occurrence.

of DP outbreaks have involved urban/suburban waterfowl, many of which are semicaptive and of mixed breeding involving domestic species. On two occasions, migratory populations have experienced mass mortalities from DP (Fig. 2.19).[182,183]

Double-crested cormorants have been the primary wild bird species victimized by Newcastle disease virus (NDV).[184,334] Highly virulent forms of this virus (velogenic strains) were eradicated from the poultry industries of the USA and Canada during the early 1970s. If velogenic Newcastle disease virus (VNDV) reappears in poultry and other captive species, usually the infected flocks are killed to prevent reestablishment of these strains. A VNDV outbreak causes severe economic impacts for the poultry industry. For example, millions of commercial chickens were destroyed in infected areas of southern California along with some backyard flocks in California and Nevada during 2002 and 2003 as part of the disease eradication effort.

In 1990, ND was determined to be the cause of a mass mortality in cormorants in Canada,[334] and in 1992 a more extensive epizootic swept across the Great Lakes area of North America killing thousands of cormorants, a small number of other waterbirds, and eventually appeared in a North Dakota poultry flock (Fig. 2.20). Additional mortality events followed in cormorant flocks at other locations, including California's Salton Sea (Fig. 2.21), which marked the first mass mortalities west of the Rocky Mountains caused by ND in wild birds.[184] Mass mortalities caused by this disease are rare in wild birds, so the cormorant ND epizootics are especially noteworthy.

Type C avian botulism (*Clostridium botulinum*) (Fig. 2.22) is a bacterial disease that has greatly expanded its historic range within North America during the latter half

Photo by Milton Friend

**Figure 2.19** During the 1975 outbreak of duck plague at Lake Andes National Wildlife Refuge in South Dakota, USA, more than 40,000 mallards died.

of the 20th century and its global distribution during the past three decades (see Figs. 2.1, 2.2). Individual epizootics have killed a million or more birds.[13] Another bacterial disease, avian cholera (*Pasteurella multocida*), has become the most important infectious disease of North American waterfowl (Table 2.10). The North American geographic distribution of this disease in wild birds has expanded greatly since the 1970s[143] and is being reported with increased frequency as a cause of wild bird mortality (Fig. 2.23). Collectively, during most years, avian botulism and avian cholera kill more wild waterbirds than all other diseases combined.

Outbreaks of chlamydiosis (*Chlamydia psittaci*) among wild waterbirds also appear to be increasing.[16] Gulls, waterfowl, white pelican and double-crested cormorant are the primary species associated with recent epizootics (mid-1980s through 2002). This bacterial zoonosis has been a source for infection in biologists handling wild birds[185,186] and is a threat for those that may enter infected bird colonies.

Photo by Milton Friend

**Figure 2.21** Double-crested cormorant chicks that died on the nest from Newcastle disease.

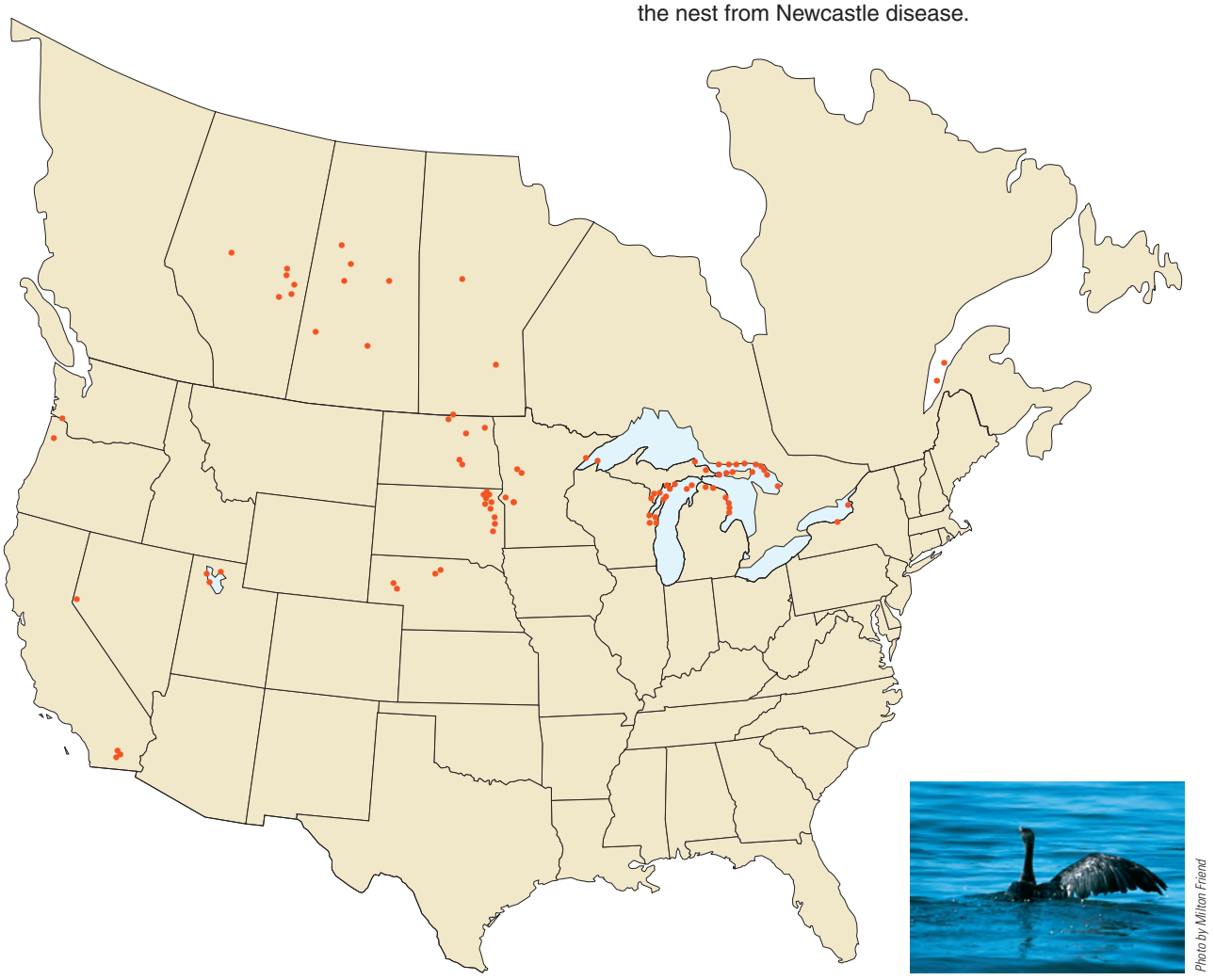


Photo by Milton Friend

**Figure 2.20** Locations in North America where Newcastle disease has caused mortality in double-crested cormorants, 1990–2000.

**Table 2.10.** Examples of wild bird mass mortalities from avian cholera (*Pasteurella multocida*) in nonmarine environments.

| Geographic area | Year | Estimated loss | Primary species affected | Comments |
|---|---|---|---|---|
| Texas Panhandle, USA | 1947 | 30,000 | Ducks | • Wintering area epizootic during winter of 1947–1948;[503] avian cholera first appeared in wild waterfowl in USA in 1944; Texas Panhandle and the San Francisco Bay area, California, were the two sites having simultaneous epizootics that winter.[143] |
| | 1956 | 60,000+ | Ducks | • Wintering area epizootic at Muleshoe National Wildlife Refuge.[504] |
| San Francisco Bay area, California, USA | 1948 | 40,000 | Ducks, geese, swans | • Wintering area epizootic; freshwater ponds and marshes sites for most mortality.[505,506] |
| Missouri, USA | 1963 | 7,000+ | Ducks, geese | • Approximately 7,000 carcasses collected during winter of 1963–1964 at Squaw Creek National Wildlife Refuge; single night mortality of 1,110 snow geese from flock of 20,000 that landed on the refuge.[507] |
| Florida Everglades, USA | 1967 | 5,000 to 6,000 | American coots | • Wintering area epizootic in Everglades National Park; small numbers of other species also died.[508] |
| Rainwater Basin, Nebraska, USA | 1975 | 25,000 | Crow, ducks, geese | • Initial avian cholera epizootic in spring migration stopover area.[509] |
| | 1980 | 30,667 | Ducks, geese | • Actual carcass pickup;[510] total mortality estimated to be 80,000. |
| | 1998 | 26,225 | Ducks, geese | • Actual carcass pickup; total mortality much greater.[16] |
| Salton Sea, California, USA | 1979 | 9,037 | Waterfowl | • Actual carcass pickup; total mortality much greater.[511] |
| Back Bay, Virginia, USA | 1976 | 25,000 | American coots | • Brackish water wintering and migration stopover area.[512] |
| Saskatchewan, Canada | 1988 | 5,000 | Redhead duck | • 4,900 carcasses of 20 species recovered; 75 percent redheads. Epizootic at major molting area; first avian cholera in ducks in Western Canada.[513] |
| Banks Island, Northwest Territories, Canada | 1995 | 30,000 | Snow goose | • Breeding colony epizootic.[514] |
| | 1996 | 20,000 | Snow goose | • Breeding colony epizootic.[514] |
| Utah, USA | 1994 | 15,000 | Ducks | • Fall migration epizootic.[16] |
| Great Salt Lake, Utah | 1998 | 44,000 | Eared grebe | • Fall migration epizootic.[16] |
| California, USA | 1990 | 12,131 | Geese, ducks | • Actual carcass pickup; total mortality much greater. Fall migration epizootic.[16] |
| | 1997 | 12,500 | American coots, waterfowl | • Fall migration epizootic.[16] |
| | 1998 | 16,062 | American coots, waterfowl | • Actual carcass pickup; total mortality much greater. Wintering area epizootic.[16] |

American coots are susceptible to a new parasitic disease for the USA. The trematode (fluke) (*Leyogonimus polyoon*) previously had only been known to exist in Europe. In 1996, a mass mortality of American coots was caused by infection by this parasite in a Wisconsin (USA) lake. American coots and common moorhen have died during additional epizootics at that location.[187]

The causes remain elusive for some mass mortalities of wild waterbirds, despite repeated occurrences with recognizable clinical signs and tissue pathology associated with those bird deaths. For example, in 1992 more than 150,000 eared grebes died at the Salton Sea from a malady that has reappeared during most years since then, killing varying numbers of eared grebes each time.[16] This disease condition is readily identifiable by a series of behavioral abnormalities seen in these birds such as coming up on land, gulping of water, and excessive preening.

A condition that causes vacuolation in myelinated central nervous system (CNS) tissue such as brain and spinal cord is affecting bald eagles and several other species of birds, including waterfowl and coots. Microscopically, the lesions of avian vacuolar myelinopathy (AVM) appear as holes in myelinated areas of CNS tissues (Fig. 2.24). About 30 of the bald eagles (65 percent) wintering at an Arkansas (USA) reservoir died from AVM during the winters of 1994–1995 and 1996–1997.[188] Following those initial events, AVM has been documented in several other locations and has caused mortality in additional species[189] (Fig. 2.25). More than 80 bald eagles have been documented to have died from AVM.

### *Terrestrial Environment*

A broad spectrum of diseases has emerged and reemerged as causes of mass mortality of wildlife in terrestrial environments (Table 2.11). The following examples illustrate the scope of diseases relative to the types of species impacted, magnitude of losses, global distribution, and types of infectious agents involved. The rapid geographic spread by several of these diseases has been unprecedented for wildlife populations. Also, their high rates of infection and severity of disease in some species are indicative of novel infectious pathogens entering naive host populations (Table 2.12).

#### Birds

West Nile fever (WNF) stands out as an emerging disease of wild birds in terrestrial environments and as an emerging viral disease that also affects mammals such as bats, horses, and humans. Following its initial appearance in 1999 in the New York City area (USA), West Nile virus (WNV) appeared

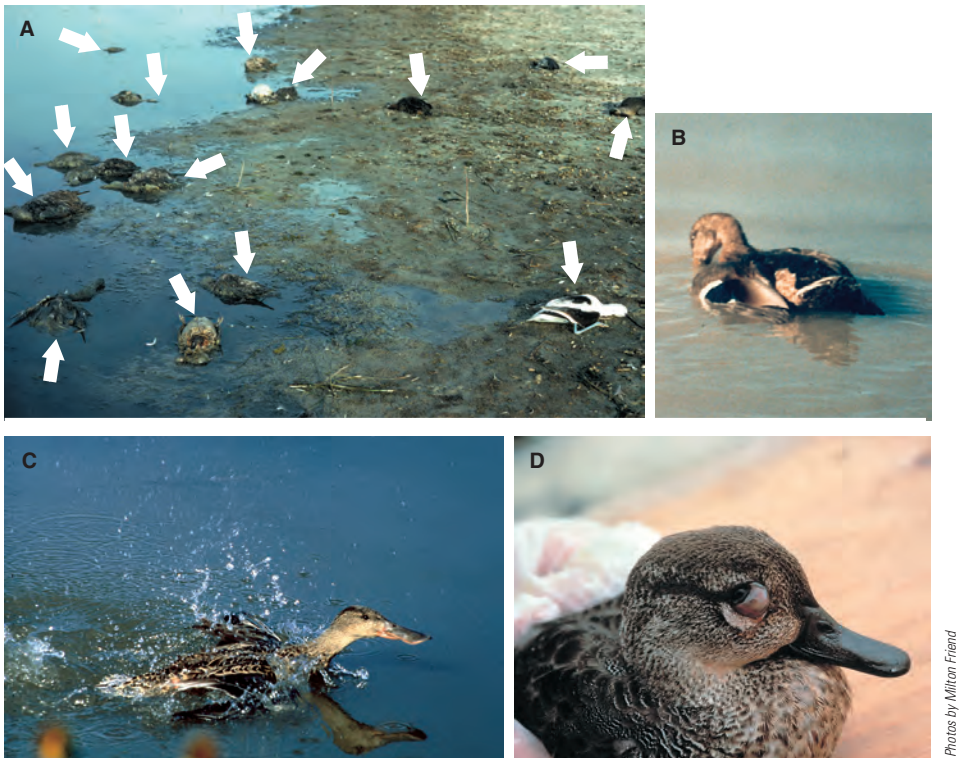



**Figure 2.22** (A) Dead birds are often found along the shore in parallel rows that represent receding water levels. (B) Botulism-intoxicated birds often exhibit "limberneck," the inability to maintain posture. (C) Botulism-intoxicated birds that have lost the power of flight and the use of their legs often attempt escape by propelling themselves across the water using their wings. (D) Paralysis of the inner eyelid is a common sign of botulism-intoxicated birds.

**Table 2.11.** Examples of wildlife mass mortality events in terrestrial environments.

| Disease | Type | Primary species | Year | Geographic area | Magnitude of mortality |
|---|---|---|---|---|---|
| Canine distemper | Virus | African lion | 1994 | Serengeti National Park, Tanzania | Approximately one-third of the 3,000 lions in the population died.[200,202,515] |
| Adenovirus hemorrhagic disease | Virus | Mule deer | 1993–1994 | California, USA | "Thousands"[516–518] |
| Plague (*Yersinia pestis*) | Bacteria | Gunnison's prairie dog | 1984–1985; 1987 | New Mexico, USA | Epizootic wave in this event covered a 100 square kilometer area and killed more than 99.5 percent of the more than 100,000 animals present.[519] |
| Anthrax (*Bacillus anthracis*) | Bacteria | White-tailed deer | 1997; 2001 | Texas, USA | Extensive losses in parts of southwest Texas; approximately 80 percent of the deer in some areas died during 1997. Epizootic in 2001 more severe than 1997 event.[520] |
| Rinderpest | Virus | Buffalo, eland, lesser kudu, giraffe | 1993–1997 | Kenya, East Africa | Epizootic wave extending over large geographic area; buffalo population in the Tsavo system declined by about 50 percent between October 1994 and July 1995; overall reductions in wild ruminants between 1991 population estimates and 1997 estimates of up to 80 percent. Losses of buffalo (29,095) were 84 percent, eland (4,279) 84 percent, and giraffe (6,936) 77 percent. Other ecosystems also lost large numbers of animals.[521] |
| Rabbit hemorrhagic disease | Virus | European rabbit | 1988 | Spain | Subsequent spread throughout Europe. Extensive mortality; has killed 50 percent or more of populations during epizootics.[522] |
| Sarcoptic mange (*Sarcoptes scabiei*) | Parasite | Many species of mammals, including marsupials (100+) | Variable | Global | Periodic epizootics that decimate populations; entry during 1970s into red fox in Sweden killed over 50 percent of population (about 90 percent in some regions). Main cause of extinction of red fox on the island of Bornholm, Denmark; most common cause of death of chamois and ibex in Europe. Disease also is threat to long-term survival of small remnant wombat populations in Australia.[523–525] |
| West Nile fever | Virus | Crow | 1999 | USA | Rapid spread of disease following introduction into New York City area; tens of thousands of birds have died.[526] |
| Trichomoniasis (*Trichomonas gallinae*) | Parasite | Band-tailed pigeon | 1988 | California, USA | More than 16,000 birds died.[16] |
| Intoxication | Drug | Vultures | 1999 | India | Populations had fallen to less than 5 percent of abundance prior to mortality events.[8,197,527] |
| Aflatoxicosis | Fungal toxin | Snow goose | 1998 | Louisiana, USA | More than 10,000.[16,528] |
| Salmonellosis (*Salmonella typhimurium*) | Bacteria | Pine siskin | 1992 | Western Canada | More than 10,000.[16] |
| Mycotoxicosis | Fungal toxin | Sandhill crane | 1985 | Texas Panhandle | About 5,000.[325] |

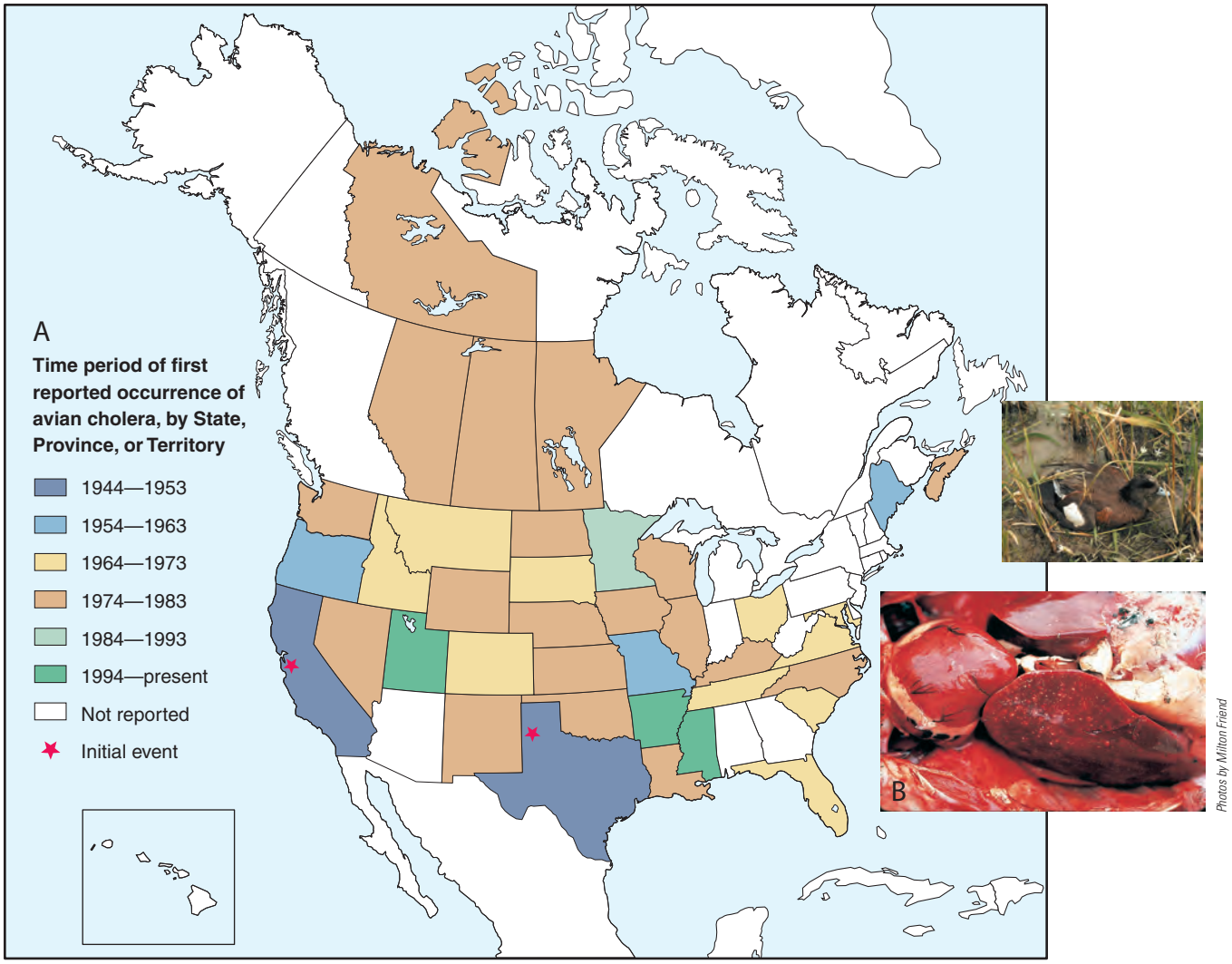



**Figure 2.23** (A) Reported occurrences of avian cholera in free-ranging birds in the United States and Canada. (B) Lesions in the liver of avian cholera-infected birds generally appear as small, discrete, yellowish spots. Pinhead-sized hemmorhages in the coronary band and heart muscle are also common.

in 44 of the contiguous states within the USA and also reached Canada within 3 years (Fig. 2.26). WNV has killed many thousands of birds, lesser numbers of other species, caused a few human deaths, and many cases of human illness. The 2002 eruption of WNF in hawks and owls created considerable concern among the wildlife conservation community because mortalities occurred in at least 12 states.[16]

Usutu virus is closely related to WNV and in 2001 emerged as the cause of bird mortality in and around Vienna, Austria. The initial epizootic killed a substantial number of free-ranging Eurasian blackbirds (a thrush species closely related to the American robin and not closely related to North American blackbirds of the Icteridae family) and several great grey owls housed at the Vienna Zoo. An epizootic among barn swallows in Upper Austria, 200 kilometers west of Vienna, followed the initial event. Retrospective analysis of archived bird samples disclosed that Usutu virus was present in Austria in 2000, even though no epizootic from this disease was diagnosed until 2001. This virus had never been observed outside of tropical and subtropical Africa nor had it been associated with severe or fatal disease in animals or humans.[190] Possibly, Usutu virus may become a recurring cause of mass mortality in birds.

House finch conjunctivitis (Fig. 2.27) is an example of an emerging disease caused by the bacterium *Mycoplasma gallisepticum* that spread rapidly following the first reported case in 1994. The initial case was seen at a bird feeder in the Washington, D.C. area. Within 3 years this disease reached

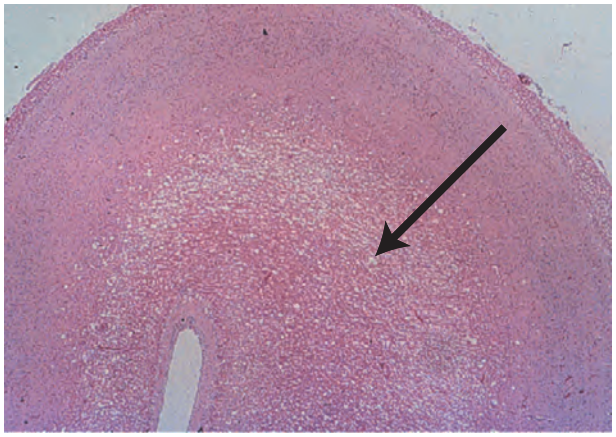


Photo from USGS files

**Figure 2.24** The most consistent pathology finding of avian vacuolar myelinopathy (AVM) is the small white dots that represent spaces between the myelin layers surrounding the nerves.

west to the Mississippi River, north to Canada, and occurred throughout the range of the eastern population of the house finch, the primary affected species (Fig. 2.28).[191,192]

Salmonellosis is another bacterial disease affecting birds. This zoonosis has long been a major disease of poultry and an important foodborne disease of humans, but epizootics involving free-ranging wild birds have been rare. However, since the mid-1980s, recurring epizootics of *Salmonella typhimurium* have taken a large toll on **songbirds** in the USA, Canada,[193] and in the United Kingdom.[194] The majority of mortalities occur at bird feeders. Other large-scale epizootics have occurred in **egret** and **heron** colonies.[16] An outbreak of *S. typhimurium* DT160 in New Zealand in the winter of 2000 that first appeared in sparrows in eastern parts of the South Island spread throughout the country killing large numbers of birds and infected other species including livestock and humans.[195] Handling dead wild birds, primarily sparrows, was associated with 13 human cases of salmonellosis; six of these cases were in children less than 5 years of age.[196]

Just as for birds utilizing freshwater and marine environments, notable diseases for which the causes remain unknown have emerged in birds within terrestrial environments (Table 2.12). Since the winter of 1994–1995, more than 50 bald eagles have died from unknown causes in Wisconsin, USA.[16] Despite intensive investigations and common pathological findings among many of these birds, a diagnosis has not been reached. These deaths differ from AVM based on the primary pathology being associated with the liver (Fig. 2.29) rather than central nervous system tissue. Also, cases have not been seen in other species or outside of Wisconsin.

### Tortoises

Emerging disease also has victimized the federally listed desert tortoises and gopher tortoises, legislatively protected species throughout their range in the Southwestern United States. Upper respiratory tract disease (URTD) has been associated with population declines in both species of tortoises. The bacterium *Mycoplasma agassizii* has been identified as the primary factor causing this disease.[203,204] Tortoises with URTD were observed in 1988 in California and a survey the following year disclosed 43 percent of 468 live desert tortoises had clinical signs of this disease.[205] The first documented large-scale mortality event from URTD in gopher tortoises occurred in 1989 in Florida. An estimated 25–50 percent of the breeding adults on Sanibel Island died during that event.[206] URTD has become a significant hurdle for conservation efforts to restore tortoise population levels, in addition to a disease causing shell necrosis. The fungus *Fusarium semitectum* has been identified as the cause of necrotizing scute disease.[207]

Tortoises on Ecuador's Galapagos Islands have not escaped recent disease. Giant tortoises at that location died in unprecedented numbers during 1979 (5), 1996 (21), and 1999 (22). Multiple mortalities are unusual for this species and a major departure from a 20-year history in which not more than one tortoise has been found dead in any year other than those noted above.[208] The cause for these mortalities has not been determined.

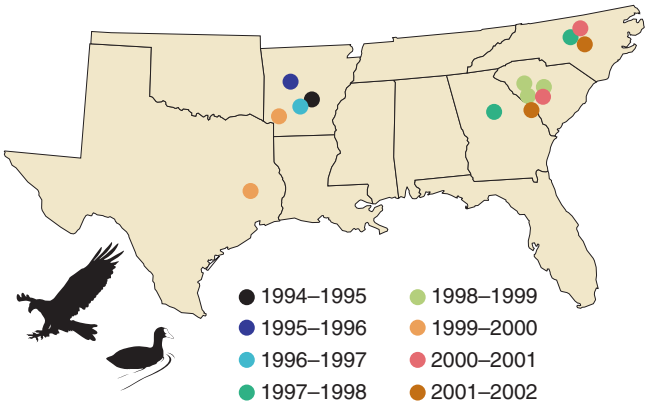



**Figure 2.25** Locations of free-ranging bird mortalities from avian vacuolar myelinopathy (AVM) in the USA, 1994–2002.

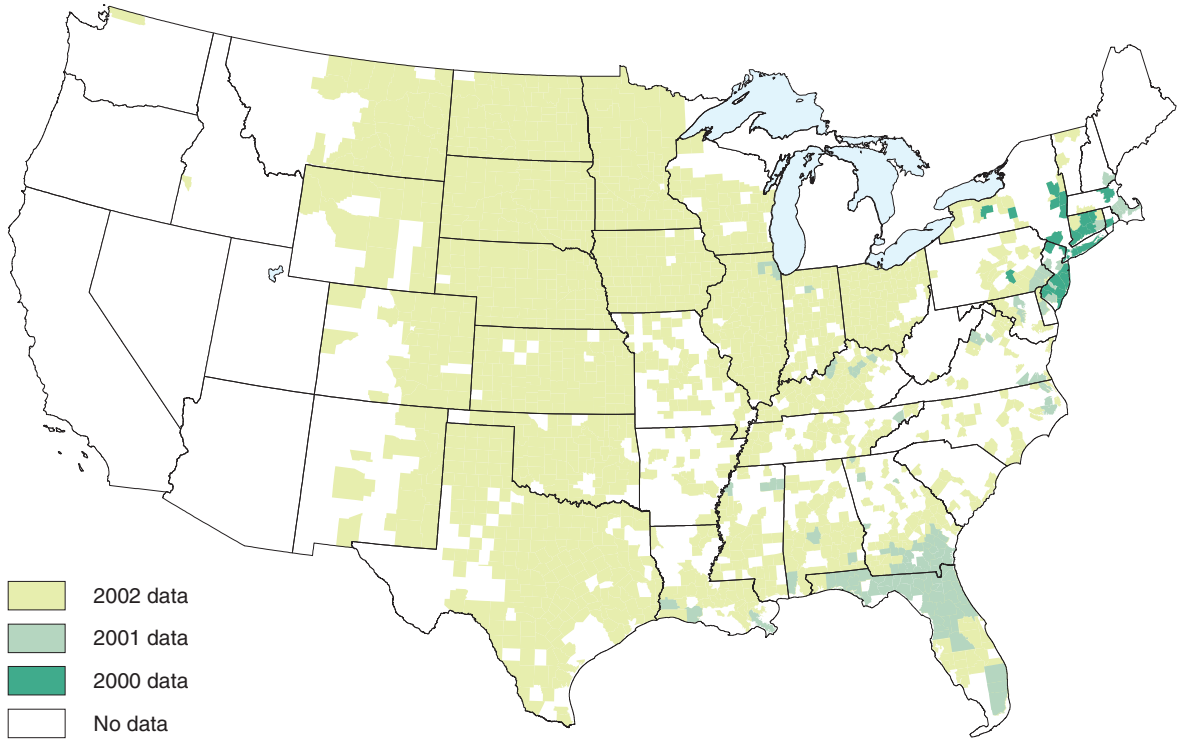



**Figure 2.26** (A) The geographic distribution of domestic animal cases of West Nile virus in the United States.

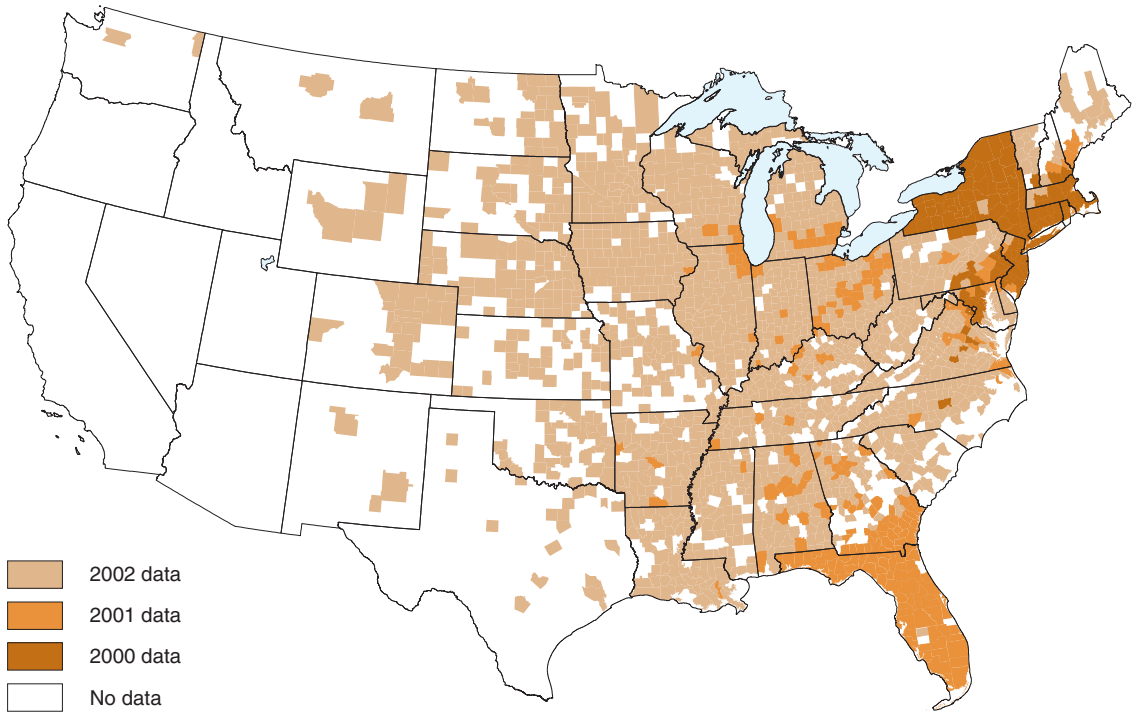



**Figure 2.26** (B) The geographic distribution of bird cases of West Nile virus in the United States.

**Table 2.12.** Examples of emerging and reemerging infectious diseases of free-ranging terrestrial wildlife populations.[a]

| Disease | Agent type | Time of emergence | Geographic area | Primary species affected | Comments |
|---|---|---|---|---|---|
| Mammals  | | | | | |
| Hog cholera (Classical swine fever) | Virus | 1980s | Europe | Wild boar | • Serious problem with multiple outbreaks involving several countries during period of 1983–2001; most occurrences during 1990s.[529] |
| Chronic wasting disease | Prion | 1980s | USA, Canada | Deer, elk | • Original foci in adjoining areas of Colorado and Wyoming; spread to wild deer in adjacent states of Nebraska and South Dakota.[357,530,531]<br>• Spread to wild deer in Wisconsin, Illinois, New Mexico, and Saskatchewan, Canada, since 2002. |
| Tuberculosis (Mycobacterium bovis) | Bacteria | 1994 | USA | White-tailed deer | • Michigan is only known USA focus in free-ranging deer.[532] |
| Tuberculosis | Bacteria | 1990s | Kruger National Park, South Africa | Lion and other species | • Spread from long-time presence of disease in African buffalo. No evidence in 1993 of spread to other species, now widespread.[533] |
| Infectious keratoconjunctivitis (Mycoplasma conjunctivae) | Bacteria | 1980s | Europe | Ibex, chamois, mouflon, thar | • First reported in wildlife in early 1900s from Austria. Mortality during recent epizootics has reached 30 percent; numerous outbreaks during 1990s.[534] |
| Adenovirus hemorrhagic disease | Virus | 1993 | USA | Mule deer | • Novel virus responsible for mortalities in 17 counties in California.[517,518] |
| Canine distemper | Virus | 1994 | Tanzania, Africa | African lion | • First epizootic of this canine virus in free-ranging large cats.[200,202,515] |
| Rabies | Virus | 1977 | Eastern USA | Raccoon | • Index case followed translocation of wild-caught raccoons from enzootic area in the southern USA; new epizootic and enzootic foci now established.[199,535–538] |
| Canine parvovirus | Virus | 1978 | Global | Canids | • Appears to have emerged in dogs in Europe; rapid worldwide spread. Infects many species of wild canids including coyote and gray wolf.[539] |
| Rabbit hemorrhagic disease | Virus | 1988 | Europe | European rabbit | • Spillover infection from domestic rabbits; rapid spread throughout much of Europe; also present in Australia and New Zealand.[522] |
| Plague (Yersinia pestis) | Bacteria | 1980s | USA | Prairie dogs | • Historic disease that has been expanding its geographic range; has caused mortality in endangered black-footed ferrets.[540,541] |

**Table 2.12.** Examples of emerging and reemerging infectious diseases of free-ranging terrestrial wildlife populations—Continued.[a]

| Disease | Agent type | Time of emergence | Geographic area | Primary species affected | Comments |
|---|---|---|---|---|---|
| Birds  | | | | | |
| Woodcock reovirus infection | Virus | 1989 | Eastern USA | American woodcock | • Novel virus causing large-scale mortality in declining eastern population of woodcock; epizootic areas are New Jersey and Virginia.[542,543] |
| West Nile fever | Virus | 1999 | USA | American crow | • Coast-to-coast spread in USA since index cases in New York City area; spread to Canada and Puerto Rico. Hundreds of species affected.[526] |
| Usutu virus infection | Virus | 2001 | Austria | Eurasian blackbird; barn swallow | • First mortality caused by this virus in any species.[190] |
| Salmonellosis (Salmonella typhimurium) | Bacteria | 1980s | USA, Canada, England | Passerine birds (songbirds) | • Widespread common disease at bird feeders.[193,194,544] |
| Mycoplasmosis (Mycoplasma gallisepticum) | Bacteria | 1994 | USA, Canada | House finch | • Rapid spread of disease throughout entire geographic range of eastern population of house finch.[191,192] |
| Intoxication | Drug | 1999 | Pakistan | Vultures | • Disease was thought to be of viral etiology and to have spread to Pakistan and Nepal; now known to be caused by an anti-inflammatory and painkiller, Diclofenac.[197,198,572] |
| Avian pox | Virus | Late 1970s | USA | Passerine birds, bald eagle | • Increasing frequency at bird feeders and factor contributing to Hawaiian forest bird mortality; numerous cases in bald eagles since species index case in 1979. Epizootics in breeding colonies of marine birds.[545] |
| Reptiles  | | | | | |
| Upper respiratory tract disease (Mycoplasma agassizii) | Bacteria | 1988 | USA | Desert tortoise, gopher tortoise | • First observed in endangered desert tortoise in California then in gopher tortoises in Florida.[205,206] |
| Ranavirus infection | Virus | 1998 | Indonesia | Green python | • First isolation of systemic infection by any ranavirus in any species of snake; detection made in Australia from illegally imported snakes collected from the wild in Indonesia.[546] |

[a] Representative examples of emerging and reemerging diseases causing mortality in wildlife; diseases such as Lyme disease that impact species other than wildlife are not included.

Photo by Terry Creekmore

**Figure 2.27** Field signs of *Mycoplasma gallisepticum* infections in house finches include eye inflammation (conjunctivitis).

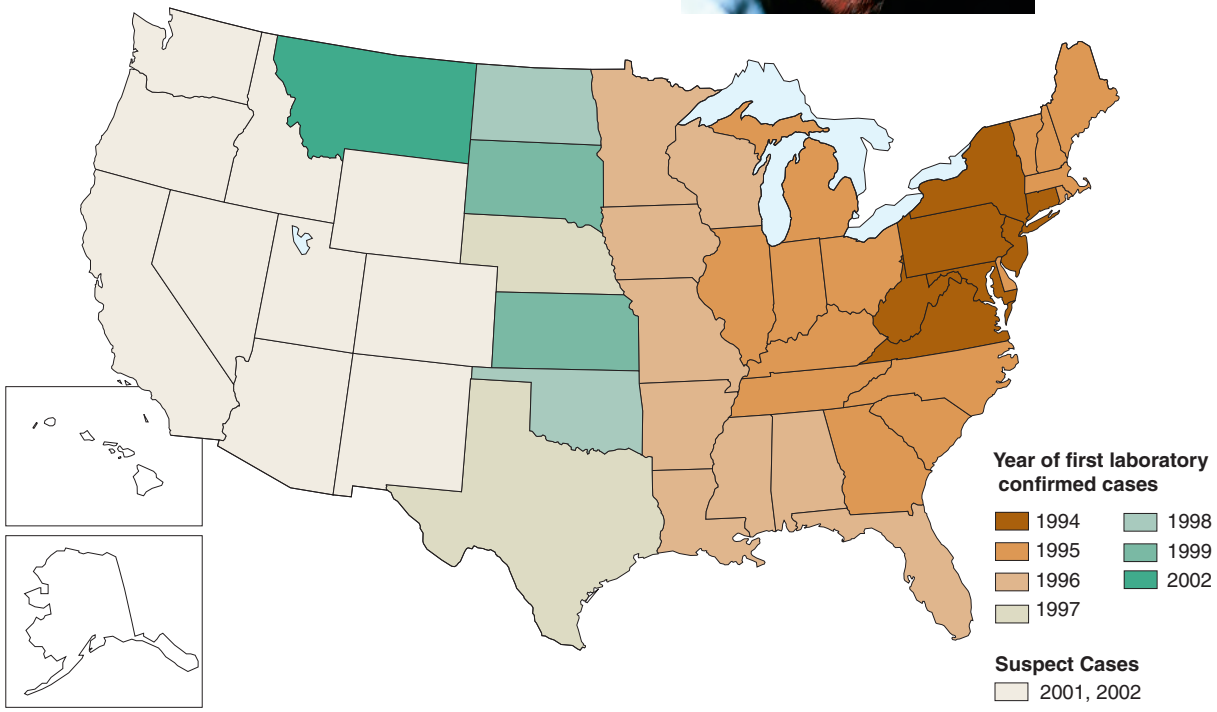



**Figure 2.28** Reported geographic spread of house finch inner eyelid inflammation (conjunctivitis) since the initial 1994 *Mycoplasma gallisepticum* observation. (Data adapted from reports in the scientific literature and personal communications between the USGS National Wildlife Health Center and other scientists. See updated data in figure 3.28.)

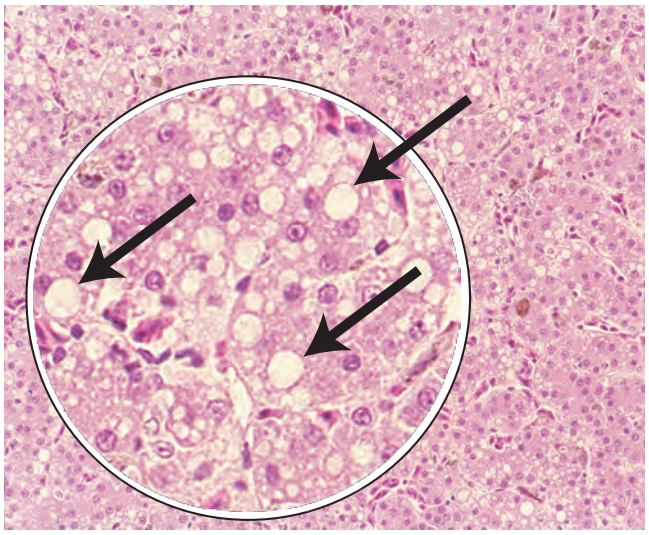


Photo by Carol Meteyer

**Figure 2.29** Numerous round, empty spaces in liver cells indicate vacuolar degeneration in an eagle that died from unknown causes in Wisconsin, USA.

### Mammals

Rabies is an example of a resurging viral zoonosis that has recently become more prominent within the Eastern USA. This age-old disease is the cause of a multistate, long-term epizootic in raccoons with spillover into other species and human exposures. In 1977, some raccoons were moved from an area where raccoon rabies is enzootic within the USA into the border areas of West Virginia and Virginia. Rabies then spread more than 400 miles (approximately 650 kilometers) northeast by 1993, killing large numbers of raccoons and a number of other species along the way.

In 1994, canine distemper, another common viral disease of **canids**, emerged as a cause of mortality in African **lions**. Canine distemper virus had previously only rarely been associated with infection in large cats and only within zoos.[200] However, distemper swept through the lion population and killed many of them at Serengeti National Park, Tanzania (Table 2.12).[201,202] A new variant of the classical canine distemper virus that emerged from local canid populations caused this mortality, and bridged the species barrier between canids and **felids**.[202]

Numerous other emerging and reemerging infectious diseases are affecting terrestrial mammals (Table 2.12). Some diseases, like tularemia and plague, are old diseases capable of causing mass mortalities in small mammals that are now appearing in new locations and under differing environmental conditions. Others, such as chronic wasting disease of deer and elk, are new diseases causing insidious impacts. The spectrum of disease and impacts also includes diseases, such as tuberculosis in deer and hydatid disease, which are of primary importance because of their impacts on other species (including humans), rather than being a cause of wildlife mass mortalities.

### Rabies and Raccoons

Historically, in the northern parts of the USA small numbers of raccoons have become infected with rabies through exposure during epizootics in other species such as **foxes** and skunks. The establishment of rabies as an enzootic disease of raccoons in several northeastern states is a dramatic example of how old diseases can exploit new opportunities. The spread of rabies in Connecticut during the epizootic wave of raccoon rabies that began in West Virginia illustrates how quickly disease status can change. The first rabid raccoon in Connecticut was detected in 1991. Prior to that time, Connecticut had been without any cases of rabies in terrestrial vertebrates for more than a decade. In less than 4 years following the 1991 **index case**, 2,500 rabies-positive animals, 80 percent of which were raccoons, were detected in Connecticut.[199]

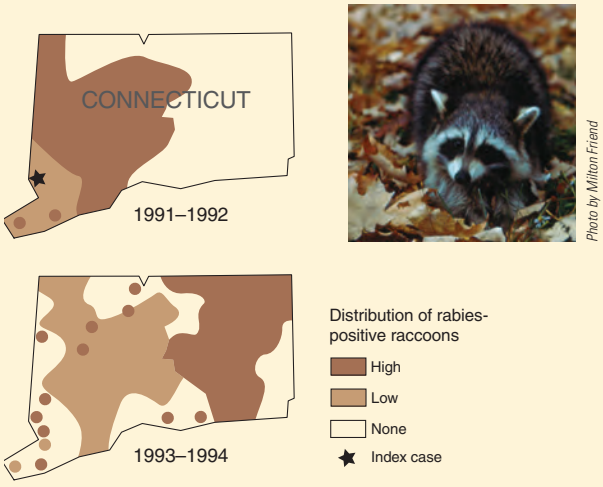




Photo by Milton Friend

### Perspective

The magnitude and complexity of emerging infectious diseases will continue to be a major challenge for the foreseeable future. The examples cited provide a cross-section of disease emergence in wildlife, rather than a holistic compendium. Clearly, disease emergence is affecting the broad spectrum of animal resources worldwide in virtually all types of environments. **Reptiles** have not been fully considered in this evaluation, and other species groups have only received moderate coverage, at best, relative to the spectrum of emerging and reemerging diseases. Listings soon become incomplete because of the dynamic nature of disease emergence. Pathogens that cross species barriers are likely to become a more frequent source for disease emergence. These events will result from new opportunities for pathogens that arise from exposure to changing environmental conditions, new species interactions, and increasing densities of potential host species as humans and animals are compressed into diminished amounts of living space. Discoveries associated with technological advances, increased investigational activities, and truly new disease events also assure a continuum of new findings. Captive-reared wildlife are an additional component of emerging diseases. Other emerging diseases are affecting native plants[209–211] and insect populations.

Disease emergence is often associated with conditions of ecosystem stress[212] caused by landscape alterations, social upheaval, and the conditions of war, which are situations that are likely to continue within different regions of the world. The increased levels of environmental stress affecting diverse systems from coral reefs to polar ice caps will be further intensified as humans attempt to provide living space, food, water, recreation opportunities, sustained economic growth and attempt to meet other societal needs.

Contact between humans and wildlife is likely to increase and may lead to more opportunities for disease emergence. Ecotourism associated with African wildlife is but one example. The close association between humans and baboons in the Kruger National Park provides a potential bridge for the transfer of tuberculosis from other Park wildlife

through infected baboons to humans.[213] Also, there is growing concern among the wildlife conservation community about ecotourists and others transferring human diseases into wildlife populations. Outbreaks of tuberculosis among mongooses and meerkats in Botswana have been attributed to humans as the source for infection.[214,215] An undiagnosed 1988 epizootic among the endangered mountain gorilla in Rwanda is thought to have been measles of human origin.[216] Also, new intestinal parasites have been found in the feces of mountain gorillas since tourists began visiting their habitat in large numbers.[214,215]

Increased globalization of society and the speed of modern transportation enhance the opportunities for disease agents to enter new geographic areas and naive host populations. Therefore, actions are needed to minimize opportunities for disease emergence in wildlife as precursors for the establishment of new zoonoses and to prevent continued escalation of zoonoses as a public health problem.

## Emerging Foodborne Diseases

**"…to speak of "foodborne disease" is to speak of many pathogens and many diseases" (Tauxe).[217]**

Foodborne transmission has been documented for more than 200 known diseases caused by a spectrum of pathogens ranging from infectious agents to biotoxins.[217,218] Viruses are the leading cause of foodborne disease in the USA, but bacteria are the most prominent causes of foodborne disease resulting in hospitalizations and deaths (Fig. 2.30). Zoonoses are associated with the great majority of those deaths in the USA (Fig. 2.31). It is estimated that one in four Americans have a significant foodborne illness each year with the majority of illness being due to pathogens yet to be identified.[217] The human health toll in the USA from these diseases is estimated in one evaluation to be 40 million cases and 9,000 deaths annually.[219] Another evaluation places this toll at 76 million cases of illness, 323,000 hospitalizations per year, and 5,000 deaths.[218]

### *Transitions and Transgressions*

The general safety of food and drinking water has long been a matter for public concern and regulatory processes. Tainted food and water are often a source for disease, which is reflected in the writings of early history and forms the basis for some of the dietary laws of various religions. The development of sanitary codes, regulatory processes, and a host of other actions focused on sources of contamination, have been created and implemented to maintain health risks at minimal practical levels consistent with technical feasibility.

Within the USA, and in many other areas, bacterial diseases such as streptococcal infections, brucellosis and tuberculosis in milk and other dairy products, and salmonellosis in poultry and eggs have been primary concerns. Trichinosis (trichinellosis) has been an important parasitic disease associated with **swine**. Pasteurization has been notable in combating brucellosis, as has mandated cooking of garbage fed to swine in combating trichinosis. Chlorination and other treatments of drinking water supplies have helped to combat a host of enteric pathogens such as *Salmonellae*. Because of these preventive measures, typhoid fever, tuberculosis, brucellosis, and septic sore throat, a zoonotic streptococcal infection, have been essentially eliminated as foodborne diseases in developed nations. Most instances of trichinosis have also been eliminated.[217]

Although many foodborne zoonoses of the past have diminished as human health problems throughout most developed nations, there has been a resurgence of foodborne zoonoses augmented by a variety of infectious pathogens that previously were not important sources of foodborne disease.[220–223] Every 2 years since 1977, a new foodborne pathogen or a pathogen newly recognized as being foodborne has appeared, many of which are zoonotic in origin.[217] The Pan American Health Organization reports that many foodborne zoonoses have increased by as much as 100 percent within recent years. The number of cases of foodborne illness in some developing countries is estimated to be as high as 10 percent of the population.[224,225] Typically, livestock and poultry have been the dominant domestic animal species involved in foodborne zoonoses. Within the USA, more than a dozen foodborne diseases have emerged during recent years (Table 2.13).

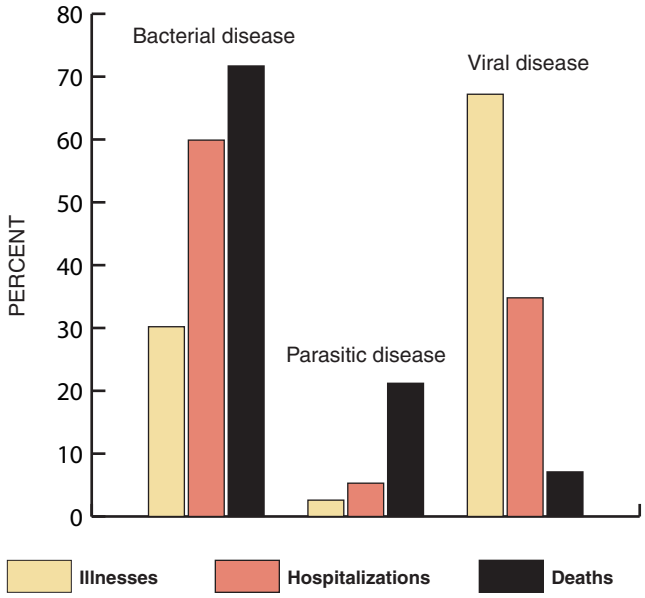



**Figure 2.30** Estimated percentage of total illnesses, hospitalizations, and deaths in the United States caused by different classes of foodborne pathogens (adapted from Mead et al.).[218]

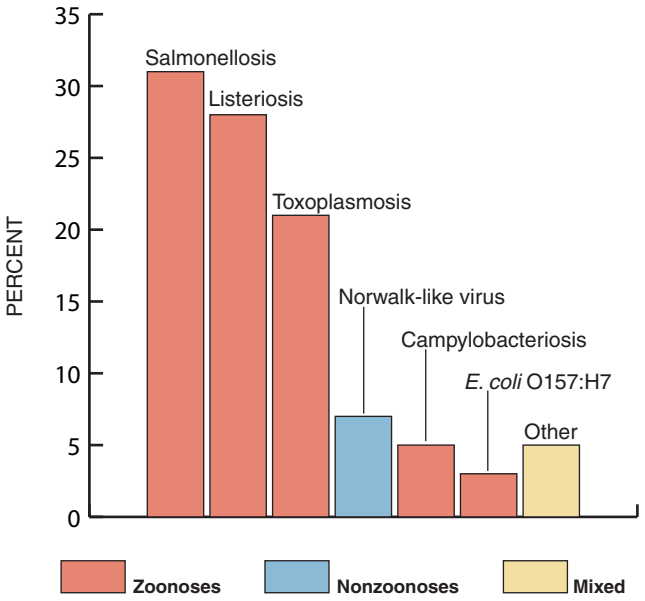



**Figure 2.31** The percentage of food-related deaths by disease in the United States (adapted from Mead et al. ).[218]

Bovine spongiform encephalopathy (BSE) is an example of not only a new foodborne zoonosis, but recognition of a new type of zoonotic agent. BSE was first diagnosed in the United Kingdom (UK) in 1986, and by August 1998 more than 174,000 cattle were reported as infected. However, it was estimated that one million cattle had probably been infected by that time and that about half of those had entered the food chain. Human infection by the **prion** causing BSE was first reported in 1995 as a new human zoonotic disease designated as new-variant Creutzfeldt-Jakob disease. By mid-1998, more than two dozen human cases of this generally fatal disease were associated with the BSE epizootic in the UK.[219]

The global dissemination of some foodborne pathogens in pandemic form is another example of the changing presentation of foodborne disease. Salmonellosis serves as an example. With the exceptions of Australia and New Zealand, strains of *Salmonella enteritidis* embarked on a global journey in the 1980s that has resulted in this cause of salmonellosis becoming the most common *Salmonella* serotype worldwide. A similar journey, with the same exceptions, began in the 1990s for antibiotic-resistant strains of *S. typhimiurium*.[217]

Associating *S. enteritidis* with eggs rather than poultry meat is a new dimension for this disease[226] and has resulted in debate relative to the roles of poultry and traditional rodent hosts in the ongoing pandemic.[226–228] Also, in 1993 an epidemic caused by *S. enteritidis* resulted in nearly a quarter of a million people in the USA becoming ill from the ingestion of ice cream.[229] Antibiotic-resistant strains of *S. typhimurium* have been linked with antibiotic use in livestock. For instance, a strain responsible for human deaths in Denmark was traced to a Danish pig herd.[230]

The bacterial diseases yersiniosis and a new serotype of *Vibrio parahaemolyticus* also have been recently identified as pandemic foodborne diseases. The strains of *Yersinia enterocolitica* involved appear to be associated with consumption of or contact with raw pork or pork products. Consuming contaminated seafood is the cause of disease from the new serotype of *V. parahaemolyticus*, which emerged in Southeast Asia in the early 1990s, then spread to Japan and the USA.[217]

*Escherichia coli* O157:H7 became a focus for concern in January 1993, with the detection of what turned out to be a multistate epizootic infecting more than 500 people, including four fatalities in children. At least 93 restaurants in a national chain were implicated, all of which obtained meat from one processing plant.[231] The first two outbreaks by this agent in the USA probably occurred in 1982 in Oregon and Michigan. Both events were associated with consumption of fast-food hamburgers. Microbial evolution is the basis for the emergence of *E. coli* O157:H7. This strain arose from a common origin to diverge genetically, causes three different forms of disease, and has become a worldwide pathogen. The variability of epidemics it causes reflects its adaptive capabilities, and its pattern of development has resulted in *E. coli* O157:H7 being categorized as a "disease of human progress."[232]

Cattle are the most significant reservoir of *E. coli* O157:H7 and other Shiga-toxin producing *E. coli*. However, the current occurrence of more foodborne outbreaks of O157:H7 linked to beef than any other single food source may not prevail over time, considering the range of foods that have been sources for human infection (Table 2.14) and species that this pathogen has been found in other than cattle and humans (e.g., birds, sheep, and deer). *E. coli* O157:H7 was first isolated in swine feces in the USA in 2001, joining Japan, Norway, and Chile, where it has been found previously.[233] Live cattle-to-human transmission, human infections from contaminated drinking water and from recreational water use, and other means of infection have been documented. Disease emergence also has been facilitated by the prolonged environmental persistence of the pathogen (several months) in water and other substrates and its unusual tolerance to environmental stresses.[232]

Campylobacteriosis is currently one of the most noteworthy foodborne diseases worldwide. Infections are acquired by consuming contaminated water, unpasteurized milk, undercooked poultry or red meat, and direct contact with infected human shedders and contaminated surfaces. Children often acquire infection from immature, diarrheic companion animals.[238] Campylobacteriosis became recognized as an emerging foodborne disease during the late 1970s.[239] In 1997, this disease accounted for approximately 14 percent of all diagnosed foodborne infections in the USA. The total

**Table 2.13.** Principal foodborne infections that have emerged during the past three decades (adapted from Tauxe).[217]

| Disease agent | Zoonoses | Primary hosts[a] |
|---|---|---|
| Viral diseases | | |
| Astrovirus | ○ | Humans |
| Norwalk-like viruses | ○ | Humans |
| Rotavirus | ○ | Humans |
| Prion diseases | | |
| Prions | ● | Cattle |
| Bacterial diseases | | |
| *Campylobacter* spp. | ● | Poultry, swine, pets, migratory birds |
| *Escherichia coli* O157:H7 | ● | Cattle |
| Enterotoxigenic *E coli.* | ● | Cattle |
| *Listeria monocytogenes* | ● | Many domestic and wildlife species |
| *Salmonella enteritidis* | ● | Poultry |
| *Vibrio* (non-cholera) | ● | Shellfish and finfish |
| *Vibrio cholerae,* toxigenic | ○ | Humans |
| *Vibrio vulnificus* | ● | Shellfish |
| *Yersinia enterocolitica* | ● | Swine, pets |
| Parasitic diseases | | |
| *Anisakis* spp. | ● | Fish |
| *Pseudoterranova* spp. | ● | Fish |
| *Cyclospora cayetanensis* | ● | Humans |

● Zoonotic infection

○ Not a zoonotic infection

[a] Primary sources for human infections; in most instances a much greater range of species may become infected and be an occasional source for human cases of disease.

estimated number of cases that year exceeded 2.5 million with 13,000 hospital admissions and 124 deaths.[238] In the UK, about 500,000 people became ill with campylobacter enteritis during 1999.[240] *Campylobacter* infections have been reported to be the most common bacterial cause of acute gastroenteritis in the industrialized world and a major cause of intestinal disease in very young children in developing countries.[240,241]

*Campylobacter jejuni* is responsible for more than 90 percent of diagnosed human cases of this disease[240] and is prevalent in all types of commercial poultry flocks worldwide.[238] An estimated 20 to 40 percent of sporadic *Campylobacter* cases may involve the consumption of chicken.[240] Wildlife also are known to harbor this organism.[242–244]

In the 1990s, cryptosporidiosis emerged as an important gastrointestinal infection transmitted by food and water contaminated by the protozoan parasite *Cryptosporidium parvum* and associated species. Human cases of this disease have been reported in more than 40 countries in 6 continents.[246] The 1993 waterborne outbreak that affected several hundred thousand people in Milwaukee, Wisconsin,[247] is well known. Analyses of that epidemic indicate that the elderly had an increased risk of severe disease, a shorter incubation period than previously reported for adults, and a higher risk of secondary person-to-person transmission.[248] The total cost of outbreak-associated illness was $96.2 million, nearly $32 million of which was medical costs and the remainder was productivity losses.[249] Cryptosporidiosis has also resulted from the consumption of contaminated apple cider, bovine and goat milk, fruits and vegetables, and other foods such as sausage, tripe, and chicken salad. Oocysts (eggs) of the parasite have also been detected in vegetables, meats, and a variety of shellfish that were not associated with human cases of disease.[246]

Seafood consumption also can cause foodborne disease. Marine and freshwater shellfish and finfish are all involved. Most seafood is safe, as are other commercial foods. However, cultural and changing food habits, such as the consumption of raw seafood and undercooking seafood, are providing increased opportunities for diseases to emerge. Parasitic

### Migratory Birds As Reservoirs For *Campylobacter*

Several species of domestic and wild animals (including birds) serve as reservoir hosts for *C. jejuni*. Migratory birds, especially waterfowl, may be the most important wildlife reservoir because of their potential to contaminate waterways and other habitat through their feces. The role of migratory birds as a reservoir for *Campylobacter* has been established by the findings of high percentages of some species (overall infection rate of 73 percent in one study) being infected with *C. jejuni*.[242–244] A far greater percentage of ducks have been found infected than Canada geese (5 percent in a Washington study) and the greatest percentage (81 percent) was found in sandhill **cranes**.[243] However, not all evaluations of wild birds have yielded positive results. No isolations of *C. jejuni* were made along the Mississippi River in Wisconsin from waterfowl, or from sediments and water where these birds roost and feed.[245] Nevertheless, the high rate of infection found in some populations of wild birds should be considered when field dressing and preparing these birds for consumption. Appropriate measures should be taken to avoid contaminating hands, surfaces, utensils, and containers used for processing other foods.

Photo by Glen Smart

zoonoses resulting from these food habits are testimony to the risks involved.[223] The consumption of raw fish has led to major increases of anisakiasis and gnathostomiasis in the USA and elsewhere.[250] Both diseases are caused by infections with different species of nematodes (roundworms). More than 90 percent of cases of seafood-borne illnesses within the USA are associated with ciguatoxin and "scombrotoxin."[251] Ciguatoxin is produced by the dinoflagellates *Gambierdiscus toxicus* and is concentrated up the food chain where it accumulates in carnivorous **reef fish** such as barracuda and popular table fish such as grouper, snapper, and sea bass. Ciguatera is the most common fishborne illness worldwide.[252] Scombroid poisoning is a result of inadequate refrigeration of fish. Bacteria (*Proteus* and *Klebsiella*) present on the surface of the fish proliferate and invade the muscle tissue where bacterial degradation processes result in a histamine-like chemical that causes human illness when ingested. Fish species most commonly associated with this food poisoning are tuna, mackerel, jacks, dolphins (mahimahi), and bluefish.[252]

Hepatitis A virus (HAV) and Norwalk-like viruses (NLVs) (*Norovirus*)[254] are the two most important viral diseases transmitted by seafood. HAV is the fourth leading cause of foodborne disease in the USA, causing 4 percent of the outbreaks and 6 percent of the cases when an etiology could be determined. Nevertheless, NLVs may be the most common cause of foodborne disease. They are the most commonly identified cause of infectious intestinal diseases in Western Europe[253] and account for greater than 95 percent of nonbacte-

**Table 2.14.** Sources of human infections by *Escherichia coli* O157:H7 (developed from Park et al.).[233]

| Country | Sources | Comments |
|---|---|---|
| USA | Hamburgers and other beef products, drinking water, lettuce, apple cider, venison, apple juice, and recreational swimming | Average incidence of 2.1 cases per 100,000 people in 1997; outbreaks occurred in 1997 and 1998 among people eating alfalfa sprouts.[547] |
| Canada | Direct contact with cattle, contaminated ground water, exposure to rural environments, undercooked ground beef | Incidence of infection ranged from 3.0 to 5.3 cases per 100,000 people from 1991 to 1996. |
| Japan | White radish sprouts | About 6,000 people, mostly children, infected in 1996 from luncheon containing radish sprouts; a second outbreak the following year infected 126 people who ate white radish sprouts.[547] |
| United Kingdom | Hamburger and other beef products | Laboratory confirmed cases increased from 1 in 1982 to 1,039 in 1995; isolated from 18.7 percent of cattle feces tested. |
| Germany | Goat milk, cheese, swimming in lakes, person-to-person transmission | Hamburgers and other beef products not common sources for infection. |
| Scotland | Ground beef | 1996 outbreak of 496 cases with 20 deaths. |

rial outbreaks in Denmark, England, Wales, Finland, France, and Sweden. The percentage is slightly lower (84 percent) in the Netherlands.[254] Human contamination by food handlers is the primary source for these diseases.[221] Multiple outbreaks of gastroenteritis associated with norovirus on cruise ships entering USA ports occurred during 2001 and 2002.[255] Fecal contamination is the source for HAV and NLVs, but shellfish are often a vehicle for human exposure to these viruses[256] (Table 2.15). Shellfish also are a source of bacterial infections. In the USA, *Vibrio* spp., and in particular *V. vulnificus*, account for the second highest number of infectious disease cases associated with shellfish (behind viral agents) and 95 percent of all shellfish-related deaths.[257,258]

In Canada, seafood is the source of about 7 percent of all outbreaks of foodborne disease and 4 percent of all reported cases. About 60 percent of cases are due to microorganisms, 31 percent to seafood toxins, and 9 percent to other chemical agents. Between 1973 and 1987, there were multiple seafood-related disease outbreaks involving infectious agents, several resulted from home food processing (canning and smoking) (Table 2.16).[259]

Foodborne diseases associated with fruits and vegetables are increasing. The mean number of reported outbreaks associated with produce more than doubled from the period 1973 to 1987 to the period 1988 to 1991 (from 4.3 outbreaks per year to 9.75 per year, respectively). The number of human cases of produce-related illness rose from 242 per year to 614 per year when comparing these same time periods.[261] International in scope,[260] this problem is not entirely independent of pathogens in animals, because some of the disease agents involved have animal hosts (Table 2.17). The use of improperly composted manure, contaminated water, and contact with products of animal origin are all factors contributing to the increasing incidence of human illness associated with the consumption of uncooked fruits and vegetables.[262]

Many diseases acquired as foodborne infections also may be directly acquired as waterborne infections and some of the cited nonfoodborne diseases use water as a pathogen-delivery system for infection. The route for disease transmission as foodborne or waterborne may be altered by changing environmental conditions. For example, giardiasis and cryptosporidiosis are more likely to be transmitted through water than food, while the reverse has been true for toxoplasmosis. Nevertheless, giardiasis and cryptosporidiosis are emerging foodborne diseases and toxoplasmosis is an emerging waterborne disease. A recent study of waterborne toxoplasmosis in Brazil disclosed that 84 percent of a subset (lower socioeconomic group) of nearly 1,500 people along a continuum of socioeconomic status in a serological survey had antibody to *T. gondii*, as did 62 percent and 23 percent of the people in middle and upper socioeconomic groups, respectively. Those findings reflect the importance of oocyst transmission by water and the risks for exposure from drinking unfiltered water.[263] It is likely that increases in waterborne zoonotic disease will continue as an outcome of degrading water quality associated with increasing human populations.

The specter of waterborne zoonotic disease extends to bottled drinking water. Recent epidemiological investigations have resulted in the three species of *Campylobacter* that cause disease being identified with different types of exposure; *C. coli* infections were most frequently associated with patients consuming bottled water. The rapidly expanding bottled water industry (5 billion gallons consumed in the USA in 2000; 7.3 billion gallons predicted for 2005) coupled with *C. coli* findings suggest the possible need for enhanced bottled water standards to protect human health from campylobacteriosis.[240]

Wild game meat also may be a source for human disease. Tularemia acquired from rabbits and hares, and toxoplasmo-

**Table 2.15.** Examples of outbreaks of foodborne viral diseases from the consumption of contaminated shellfish.

| Disease agent | Food item | Human cases | Location | Year |
|---|---|---|---|---|
| Norovirus | Raw clams | 813 | New York, USA | 1982[252] |
| Norovirus | Raw oysters | 204 | New York, USA | 1982[252] |
| Hepatitis A virus | Raw clams | 300,000 | Shanghai, China | 1988[256] |
| Hepatitis A virus | Raw oysters | 61 | Alabama, Georgia, Florida, Tennessee, Hawaii, USA | 1988[256] |
| Norovirus | Oysters | 175+ | Eastern Canada | 1991[259] |
| Gastrointestinal virus[a] | Raw/steamed oysters | 180 | Louisiana, Maryland, Mississippi, Florida, North Carolina, USA | 1993[256] |

[a]Small round-structured gastrointestinal viruses related to noroviruses.

**Table 2.16** Sources of infectious foodborne illness from seafood in Canada, 1973 to 1987 (adapted from Todd).[259]

| Disease agent | Number of incidents | Primary sources of infection |
|---|---|---|
| *Staphylococcus aureus* | 28 | Commercial and home-canned finfish, primarily salmon (12 events); crab (4 events) |
| *Salmonella* spp. | 17 | Tuna, salmon, lobster, and crab |
| *Bacillus cereus* | 15 | Shrimp, lobster chowder, various crab products, clams, scallops |
| *Clostridium botulinum* | 11 | Home-fermented salmon eggs; home-smoked salmon, trout, or char |
| *Clostridium perfringens* | 5 | Fish |
| Norovirus | 1 | New Brunswick oysters (~175 people infected) |
| Anisakiasis (parasite not reported) | 1 | Sushi from an unidentified species of fish; a case the previous year was reported from eating cod cooked on a campstove |

**Table 2.17.** Foodborne infections from produce in the USA that have emerged during the past 3 decades (adapted from Tauxe,[217] with additions from Millar et al.[246]).

| Produce type | Pathogens[a] | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Viral | | Bacterial | | | | | | | | Parasitic | |
| | V1 | V2 | B1 | B2 | B3 | B4 | B5 | B6 | B7 | B8 | P1 | P2 |
| Lettuce/cabbage/greens | ● | ● | ○ | ○ | ○ | ● | ● | ○ | ● | ○ | ○ | ○ |
| Carrots/celery/scallions | ○ | ● | ○ | ○ | ● | ○ | ○ | ○ | ● | ○ | ● | ○ |
| Sprouts | ○ | ○ | ● | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ |
| Tomatoes | ● | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ |
| Melons | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ |
| Raspberries/strawberries | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● |
| Fruit/vegetables (unspecified) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ |
| Chopped garlic | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Apple cider | ○ | ○ | ○ | ○ | ○ | ● | ○ | ● | ○ | ○ | ● | ○ |
| Orange juice | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ |
| Coconut milk | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ |

● Pathogen found in produce type ○ Pathogen not found in produce type

[a] V1—Hepatitis A
V2—Noroviruses
B1—*Bacillus cereus*
B2—*Clostridium botulinum*
B3—Enterotoxigenic *Escherichia coli*
B4—*E. coli O157:H7*
B5—*Listeria monocytogenes*
B6—*Salmonella* spp.
B7—*Shigella* spp.
B8—Toxigenic *Vibrio cholerae* 01
P1—*Cryptosporidium parvum*
P2—*Cyclospora*

All of these pathogens, except V1, V2, and P2, are zoonotic

sis acquired from birds and other species, are examples of diseases more commonly acquired within developed nations when wildlife is a common component of the foodbase. The transition from wildlife sources to domesticated sources of food resulted in more foodborne zoonoses being acquired from domestic animals than from wildlife. However, the wildlife component of foodborne disease is sustained in subsistence cultures and to a lesser degree among sportsmen. In the USA, from 1981 to 1996, nearly 40 percent of all cases of human trichinosis were from eating game meat.[264] Wildlife also are a disease dimension associated with aquaculture, ecotourism, and changes in human lifestyles and food habits.

### *"Going Native"*

The tourism industry is the world's largest employer with nearly 200 million jobs or about 10 percent of the jobs globally.[265] More than 663 million tourists traveled internationally in 1999 and spent more than US$453 billion in the pursuit of their activities. Ecotourism has become a major component of international travel, increasing annually at 10 to 30 percent.[266]

People travel the globe each year to visit exotic places and experience the wildlife and cultures of the area (Fig. 2.32). These sojourns generally provide new experiences, including new types of food and beverages. Wild game, native fruits, and other local items often are the major foods for people in remote areas and in cultures that are closely tied to nature. Tourists often consume these foods as part of their trip experiences. In many situations, those food items are locally harvested and may have minimal to no external oversight relative to health standards. Therefore, it is prudent to obtain basic knowledge of zoonoses that are commonly transmitted through food and water in areas to be visited. Advance knowledge provides a foundation for choices on what one consumes when in those areas and how that food is prepared. These considerations extend to raw fruits and vegetables, as well as to cooked meats, dairy products, and other food items.

Millions of people in the mainstream of industrialized society also "return to nature" through transient personal harvests of shellfish, birds, mammals, and other types of wildlife (Fig. 2.33). These harvests, which generally are devoid of any external food safety evaluations, supplement people's

Photo by Milton Friend

**Figure 2.33** Harvested wildlife.

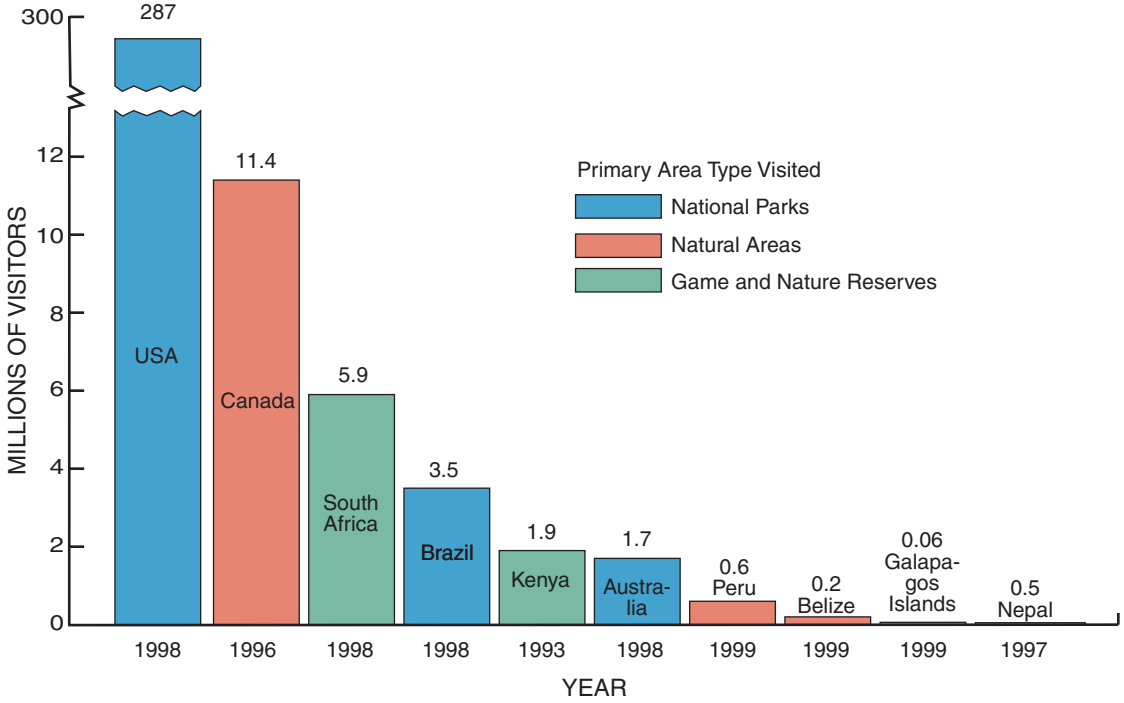



**Figure 2.32** Global examples of nature tourism during the 1990s. [266]

foodbase in various ways from basic staples to novel food items. Some tangential protection is provided by regulatory agencies through health advisories issued by government agencies to alert those harvesting wildlife for personal and family consumption about certain types of risks that may be present in specific areas.

However, because there are no programs that routinely and continuously monitor free-ranging wildlife populations for foodborne infectious pathogens, consumers of wildlife are primarily left to their own judgments relative to what to eat and how to prepare it. Experience gained over time in the proper handling and preparation of wild game in ways that minimize risks for foodborne illness is useful, but foodborne illnesses may be obtained from wildlife (and other animals) with no overt clinical signs or gross lesions of disease. Therefore, local knowledge of diseases present in the pursued wildlife also needs to be considered. This is a cumulative learning process that becomes strengthened by becoming familiar with the areas where wildlife are harvested.

The common sense of most people, coupled with the general good health of most wildlife, are major factors why wildlife, from shellfish to mammals, provide wholesome food for millions of people (Fig. 2.34). Nevertheless, problems do occur. Within the USA, home preparation of novel foods often is the cause for the infrequent occurrence of foodborne illness from wildlife. For example, during 1995, **cougar** jerky was the source for 10 cases of trichinosis in Idaho. All of those cases involved jerky made from a single animal taken by a hunter. The meat was not sufficiently heated during the smoking process to kill the parasites present. In North America, wildlife-associated cases of trichinosis most commonly result from the consumption of insufficiently cooked **bear**, wild boar, and walrus meat.[267] Wild boar is an important species associated with trichinosis in Europe, where this disease appears to be an emerging zoonosis.[268] Wildlife also are sources for this disease in South America and Asia.[269]

### *Perspective*

Information provided here and elsewhere clearly illustrates that the nature of foodborne diseases has changed greatly in the USA and globally during the 20th century. A substantial number of the pathogens of greatest concern today have only recently (within the past 25 years) been recognized as causes of foodborne illness.[218] In part, improved technology has helped scientists detect and study the pathogens involved, especially enteric viruses.[221] Nevertheless, the threat from emerging foodborne disease is more a product of our global marketplace and mobile society, than it is a result of advanced technology. Consider for example that the USA is the world's second largest importer, as well as the second largest exporter, of seafood.[251]

The consequences for human health following infection by foodborne diseases often extends beyond initial illness by causing chronic sequelae or long-term disability (Table 2.18).[218,220] The large number of foodborne diseases involving pathogens of animal origin indicates that consideration should be given to disease emergence in wildlife and other animals as factors influencing foodborne and waterborne diseases.

### Deer and *Escherichia coli* O157:H7

Annually, about 10 million Americans hunt deer.[234] Venison supplements many larders and is an important staple for some. Preparation of deer meat includes venison summer sausage, jerky, and fondue, in addition to grilling, roasting, and other common ways for cooking beef and other meats. Mule deer (black-tailed deer) and white-tailed deer both have been the source of *E. coli* O157:H7 infections in humans. In 1995, an outbreak involving 11 human cases was traced to the consumption of jerky made from the meat of a mule deer killed the previous week in Oregon.[235] A spontaneous human case was also diagnosed following the consumption of venison from a white-tailed deer killed in Vermont. The meat had been grilled and served rare.[236] Both events were the first documented for the deer species involved. The mule deer event was the first time jerky had been documented as a source for infection.

Deer previously had been linked to human cases of *E. coli* O157:H7 but proof is lacking for those reports. In 1987, the organism was recovered from venison that caused an isolated human case in Washington State. However, cross-contamination from beef butchered at the same facility may have been the source of the organism.[235] In another situation, contamination of an apple orchard by deer feces was hypothesized to be the source for an *E. coli* O157:H7 outbreak caused by unpasteurized apple cider.[237]

Photo courtesy of the U.S. Fish and Wildlife Service



Photos by Milton Friend

**Figure 2.34** (A) Outdoor recreation experiences often involve the harvest of wildlife and (B) consumption of some of the harvest under field conditions.

**Table 2.18.** Examples of chronic sequelae or disability associated with foodborne disease.

| Disease | Sequelae/disability | Comments[a] |
|---|---|---|
| Campylobacteriosis | Guillain-Barré syndrome (GBS) | One of the most common causes of flacid paralysis in the USA. An estimated 1,360 cases in 1997 were associated with *Campylobacter* infections; about 30 percent of infections are followed by GBS. |
| *Escherichia coli* infections | Hemolytic uremic syndrome (HUS) | *E. coli* O157:H7 is a leading cause of HUS, the most common cause of acute kidney failure in children in USA; about 4 percent of all reported cases develop HUS. In Canada, non-O157 cases of *E. coli* contribute to at least 7 percent and perhaps 20 percent of the HUS cases. |
| Listeriosis | Miscarriages, meningitis | Cases of meningitis are associated with patients with chronic diseases. |
| Cryptosporidiosis | Diarrhea | Persons with AIDS generally have a severe protracted course of diarrhea. |
| Toxoplasmosis | Congenital malformation; retinitis, encephalitis | Involved in mental and physical retardation cases in Korea.[548] In USA, 1:10,000 births results in congenital toxoplasmosis; 300 to 2,100 ocular cases estimated annually. Each year an estimated 4,000 AIDS patients develop *Toxoplasma* encephalitis. |
| Trichinosis | Chronic illness | In 10 to 20 percent of cases, neurological or cardiac symptoms develop, many are severe and may lead to chronic illness. |
| Salmonellosis | Arthritis | Infection may cause invasive disease or reactive arthritis. |

[a]Information from Altekruse et al., [220] and Mead et al.,[218] unless otherwise noted.

## Disease Emergence and Companion Animals

**"More than half the households in the English speaking world keep a pet. The most common pets are cats and dogs" (Riordon and Tarlow).[270]**

Nearly 60 percent of all households within the USA own either a dog or a cat.[250] Results from a recent survey indicate an estimated 68 million dogs and 73 million cats among the 63 million USA households that own pets.[271] Of the households that owned a pet, the 2000 USA census reports that 36 percent had a dog and 32 percent had a cat.[271] Over 98 million other types of pets, from fish and reptiles to horses, also are part of 20.6 million USA households.[272] The estimated numbers of animals involved are 19 million birds, 19 million small animals of various species, 9 million reptiles, 159 million freshwater fish, and 6 million saltwater fish.[271] An estimated 15 to 20 percent of American households have pet birds and 20 million households have aquariums.[273] Many other countries also have a high percentage of households with pet ownership (Fig. 2.35). Pets can contribute to the physical, social, and emotional health of many, especially enhancing the development of children and the well-being of the elderly.[274] The popularity of companion animals is likely to continue to increase, as is the increase in different species, other than dogs and cats, kept as pets. For example, the number of **iguanas** imported into the USA rose from about 28,000 in 1986 to nearly 800,000 in 1993.[275]

People are generally aware of health hazards associated with pet ownership such as animal bites, allergies, and high-profile diseases like rabies in dogs. However, most individuals are unfamiliar with the diversity of other diseases transmissible to humans that pets may harbor (Table 2.19). There is even less appreciation of emerging diseases as a component of pet ownership.[274,276]

### *Risk Factors*

Health hazards associated with pet ownership can be classified into three classes of disease.[277] Allergic response, asthma, and/or hypersensitivity pneumonitis are immunologic conditions and are not considered here. Bites and/or scratches may induce infections by microbes present in the saliva and on the mouth parts of the pet. For venomous species, toxins may be injected into the body. In the USA, there are an estimated 1 to 2 million dog bites and 400,000 cat bites each year, many of which result in bacterial contamination.[277] Infections occur in about 5 percent of dog bites and 16 to 35 percent of cat bites.[270] A mixture of microorganisms frequently causes these infections. Those most commonly involved include *Staphylococcus* spp., *Streptococcus* spp., *Corynebacterium* spp., *Pasteurella multocida*, *Capnocytophaga canimorsus* (formerly called DF–2), and a variety of anaerobes.[278] The third health-hazard category is transmission of infectious diseases.

The risks of acquiring zoonotic diseases from companion animals differ among groups of people and animal species kept as pets. Factors influencing disease risk include age and source of the animals, type of environment within which the animals are maintained, and physiological status and age of the pet owners. Investigations involving these factors have disclosed that many new dogs and cats are acquired as puppies and kittens. Typically, these young animals have a higher prevalence of parasitism and, if untreated, provide more risk for infection of household members. Young children tend to have a great deal of close contact with those animals and are at increased risk. Pets acquired from animal shelters and pet stores often have greater parasite burdens than pets in personal ownership.[274]

Diseases involving wildlife as companion animals are addressed elsewhere. Here the focus is on dogs and cats, and on providing a conceptual awareness of disease aspects associated with pet ownership. Numerous evaluations of zoonoses transmitted by dogs and cats have been published[270,277–279] including the recent book, "Dogs, Zoonoses and Public Health."[280] Those publications include specific information about infections acquired from dogs and cats.

Dogs and cats confined to the home and those that have controlled outdoor excursions within urban environments have less opportunity to acquire pathogens from wildlife than pets living in rural settings and most hunting dogs that are allowed to roam in adjacent fields and wooded areas. Hunting dogs are generally controlled during their field activities. Dogs and cats that kill and consume small rodents, feed on carrion encountered in the field, or are fed viscera and other waste from animals harvested and processed by humans for food are at risk of acquiring infectious agents. Often the dog or cat does not become infected by the pathogen. Instead, its mouth and claws become contaminated by the pathogen.

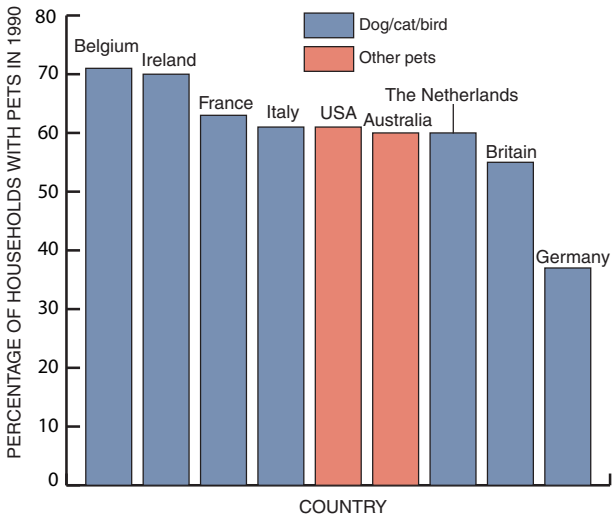



**Figure 2.35** The percentage of households with pets, 1990.

**Table 2.19.** Examples of infectious diseases transmitted to humans by domesticated species of household pets in the USA (adapted from Plaut et al.).[277]

| Disease | Pet animals | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Dog | Cat | Ferret | Rabbit | Hamster | Other rodents | Horse | Parrot | Pigeon | Other birds | Turtle | Fish |
| **VIRAL DISEASES** | | | | | | | | | | | | |
| Rabies[a] | ● | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Lymphocytic choriomeningitis | ○ | ○ | ○ | ○ | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ |
| **BACTERIAL DISEASES** | | | | | | | | | | | | |
| Campylobacteriosis | ● | ● | ○ | ○ | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| *Capnocytophaga canimorsus* (DF-2) | ● | ◐ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Leptospirosis | ● | ◐ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ |
| Lyme disease | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Melioidosis | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ◐ |
| *Mycobacterium marinum* | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● |
| *Pasteurella multocida* | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Plague | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Rat-bite fever | ○ | ◐ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Salmonellosis (not *S. typhi*) | ◐ | ◐ | ● | ● | ● | ● | ○ | ● | ● | ● | ● | ○ |
| Tetanus | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Tularemia | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Yersiniosis | ● | ● | ○ | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ |
| Chlamydial and rickettsial diseases | | | | | | | | | | | | |
| Cat scratch fever | ◐ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Chlamydiosis | ○ | ◐ | ○ | ○ | ○ | ○ | ○ | ● | ● | ● | ○ | ○ |
| Rocky Mountain spotted fever | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

| Disease | Pet animals | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Dog | Cat | Ferret | Rabbit | Hamster | Other rodents | Horse | Parrot | Pigeon | Other birds | Turtle | Fish |
| |  |  |  |  |  |  |  |  |  |  |  |  |
| **FUNGAL DISEASES** | | | | | | | | | | | | |
| Cryptococcosis | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ● | ● | ◐ | ○ |
| Ringworm | ● | ● | ● | ● | ○ | ● | ● | ● | ○ | ○ | ○ | ○ |
| Sporotrichosis | ◉ | ● | ○ | ○ | ○ | ◐ | ○ | ○ | ○ | ○ | ○ | ○ |
| **PARASITIC DISEASES** | | | | | | | | | | | | |
| Cryptosporidiosis | ◉ | ◉ | ◉ | ○ | ○ | ◉ | ○ | ○ | ○ | ○ | ○ | ○ |
| Cutaneous larva migrans | ◉ | ◉ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Visceral larva migrans | ● | ◉ | ◐ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Echinococcosis | ◉ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Scabies and *Cheyletiella* | ◉ | ◉ | ○ | ◉ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Toxoplasmosis | ◐ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Giardiasis | ◉ | ◉ | ◉ | ○ | ○ | ◉ | ○ | ○ | ○ | ○ | ○ | ○ |

●, frequent; ◉, common; ◐, rare; ○, unreported

[a]Rabies is a rare human disease in USA.

Transmission of disease agents by pets to humans often occurs during play and other close contact. Typically, this results from dogs licking the skin of people and through scratches inflicted to skin surfaces by dogs and cats. Persistence of pathogens in the mouths of pets is much more prolonged than that on the feet. For example, *Pasteurella multocida* has been isolated from the mouths of 50–70 percent of healthy cats. Typically this bacterium causes localized infections in association with bite wounds[279] but more severe outcomes can occur. This same organism has caused meningitis in infants following their faces being licked by dogs.[281] Cat scratch disease, or bartonellosis, is an example of a disease associated with young kittens that is transmitted by scratches, and less frequently by being licked on the face.[270,277]

Dogs and cats may have, or can acquire, ticks and biting insects that are either infected or contaminated with infectious agents. Those arthropods may be transferred within the home environment to members of the household. Infection of humans occurs when the arthropod feeds on the human. Examples include ticks that transmit tularemia, Rocky Mountain spotted fever, and Lyme disease, and **fleas** that transmit plague, bacillary angiomatosis, scabies, and *Cheyletiella* infections. The use of tick and flea collars to prevent attachment of these arthropods to pet animals along with the timely inspection and careful removal of ticks from pets (tularemia has been transferred to humans by crushing infected ticks during removal) can minimize this potential source for human infections.

The probability of dogs being infected with *Giardia* and the chance for acquiring this protozoan parasite is greatest in households with multiple dogs. This is true for a number of parasitic zoonoses where the eggs of these parasites are shed in feces and may persist for some time. Home environments can become heavily contaminated by parasites transmitted by fecal-oral routes if feces are not regularly removed from yards and if cat litter boxes are not cleaned often.[282] Toxoplasmosis, hookworms, and roundworms are examples of these situations. Cats are the major source for transmission of toxoplasmosis and because it takes 1–5 days for the oocysts to become infective, cat litter should be disposed of daily.[270] Dog and cat hookworms are the cause of cutaneous larva migrans and dog and cat roundworms of the genus *Toxocara* are the most common causes of visceral larva migrans. Both types of infection are acquired from soils contaminated by pet feces.

### *Immunocompetency as a Factor*

The potential for humans to become infected by disease agents associated with their companion animals is often related to the physiological condition of the person. Disease emergence and reemergence have been a hallmark of AIDS because of the immunosuppression associated with this disease. For example, the average annual incidence of salmonellosis among AIDS patients is 19.2 times greater than the population without AIDS, that for campylobacteriosis 39 times greater, and between 30 percent and 50 percent of AIDS patients have disseminated tuberculosis caused by the *Mycobacterium avium* complex,[60] organisms that seldom cause disease in humans. Similarly, infectious diseases are often complications for patients whose immune systems have been suppressed by treatments associated with organ transplants, cancer therapy, and by other conditions. When the degree of immunosuppression is great, organisms that typically are unable to cause disease or only minor illness in healthy persons may cause serious disease in immunosuppressed individuals. Similarly, the very young also have increased vulnerability until their immune system becomes fully developed. Vulnerability of the fetus is a factor in protecting pregnant women from disease agents such as *Toxoplasma gondii* that can invade the fetus. This parasite can cross the placenta and cause chorioretinitis and severe brain damage in the fetus.[270] These conditions should be considered in contacts between humans and their companion animals and appropriate steps taken to minimize health risks.

The aging human population is another aspect of reduced immunocompetency that, like AIDS and organ transplants, is an emerging component of modern society. A consequence of aging relative to disease emergence is the potential waning of immunocompetency. In the United States, 2.6 percent of the population was 74 or older in 1950. By 1995, that percentage had more than doubled to 5.6 percent and represented 14.7 million persons versus 3.8 million in 1950.[283] Currently, 20 percent of the USA population is comprised of the very young, the elderly, pregnant women, and **immunocompromised** individuals. This percentage is expected to increase substantially.[60] The increasing percentage of senior citizens in society has been projected for the near term by the Bureau of Census and potentially indicates a greater pool of human hosts with increased susceptibility to pet-transmissible zoonoses. They report that at the beginning of the 20th century less than 5 percent of the United States population was over 65, but by the year 2040, more than 25 percent of the population will be that age or older.[284]

The aging human population is an important consideration relative to the role of companion animals in disease emergence because of current trends to incorporate animals within the environment of nursing homes. These animals provide companionship and other attributes that are important benefits for improving the quality of life for many of the elderly confined to these facilities. The aggregation of elderly within the space limitations of nursing homes provides a potential for epidemics of zoonotic disease transmissible by companion animals. Therefore, it is important that adequate health maintenance be provided for animals maintained within nursing homes and that informed decisions are made on species acquisitions and the sources of animals brought into those facilities.

*Pets and Human Wellness*

In general, humans benefit from pet ownership. Dogs and cats are a source of great pleasure for humans and significantly contribute to the physical and emotional well being of the elderly, as well as to their safety.[59] For example, a European evaluation disclosed that, in general, pet owners have lower blood pressure and cholesterol levels than non-pet owners and use fewer medications.[285] A study of AIDS patients disclosed less depression among patients who owned pets than for patients who did not.[286] Nevertheless, the potential health risk to humans from enteric parasites harbored by pet dogs and cats is a significant problem throughout the world[276] and the elderly are among those at greatest risk.[287] As noted above, dogs and cats also are sources for diseases caused by a variety of microbes. The challenge is to maximize human benefits from pet ownership by minimizing any associated risks from disease. To do this, there is a need to fully appreciate the nature of the disease risks and how those diseases are transmitted. That information provides the foundation for strategies and actions needed.[60] Public education is an important component of those strategies and actions.[274,276]

## Factors Contributing to Disease Emergence

**"We have the met the enemy and he is us" (Pogo).**

The emergence of infectious disease can be viewed as a two-step process. First, the pathogen is introduced into a new host population; the pathogen then becomes established and is further disseminated within that population.[288] Disease expansion to other populations often follows. Numerous examples have been provided regarding the introduction of pathogens into new wildlife host populations. The steep mortality curves and relatively short duration of the epizootic stage of many wildlife disease events (Fig. 2.36) are typical of "virgin soil epidemics" that occur in naive human populations. World history has documented many such past events having profound impacts on human populations.[1,291–293] The AIDS pandemic and the recent reintroduction of cholera (*Vibrio cholerae* O1) into the Americas are current examples of significant pathogens introduced into human populations.

Pathogens are introduced by numerous means, but these introductions do not necessarily result in disease establishment, further dissemination of disease within the population, or further dissemination of the pathogen to other populations and geographic areas. Numerous novel pathogens and disease conditions have been observed as isolated events, and commonly appear in scientific journals as brief case reports to document the occurrence of the pathogen or disease condition in wildlife and to alert others. In some instances, diseases encountered may be zoonoses.[292–294] For example, a wildlife biologist acquired an isolated case of an exotic fungal disease (streptothricosis) (Fig. 2.37) while checking hunter-killed

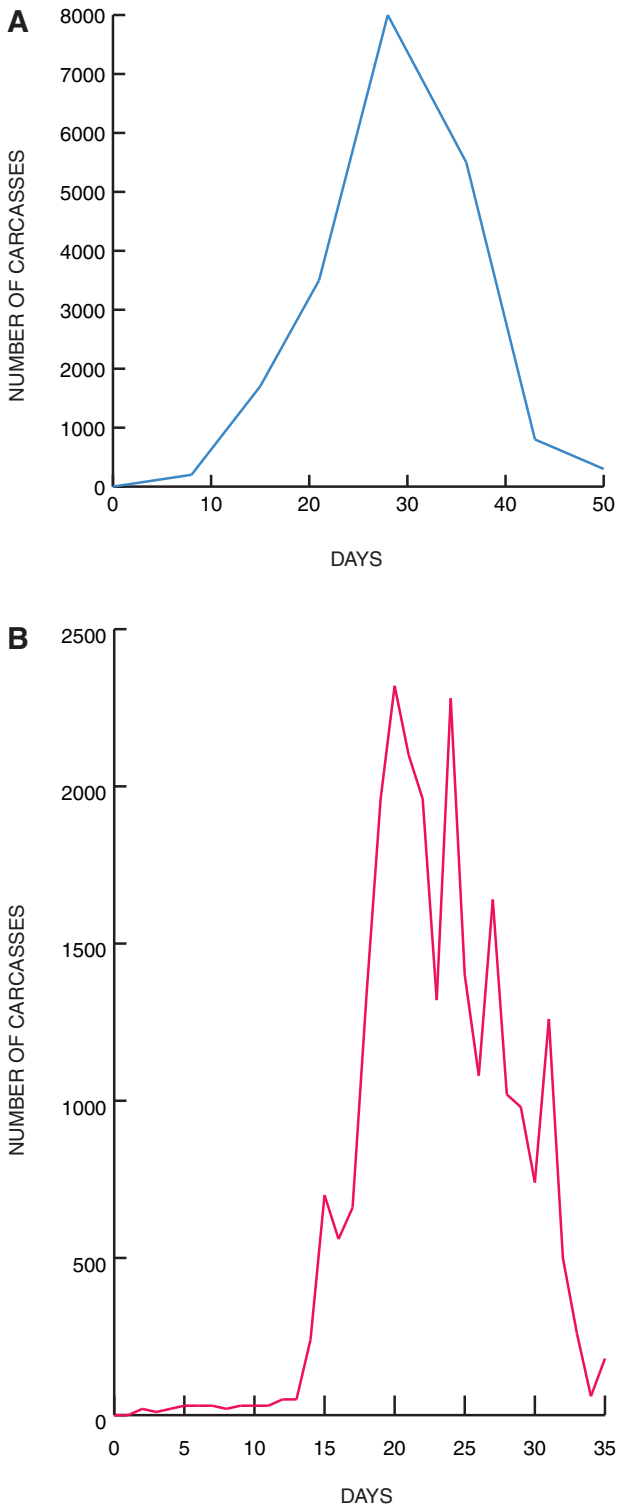



**Figure 2.36** Epizootics can cause many wild bird mortalities in a short time period. (A) Mallard mortalities due to duck plague at the Lake Andes National Wildlife Refuge, South Dakota, USA, in 1973. (B) Wild bird mortalities due to avian cholera in the Rainwater Basin, Nebraska, USA, 1975.

deer, a routine activity conducted by hundreds of biologists and technicians every year. Intensive follow-up investigations failed to disclose additional cases.[292]

In many instances, it is the interactions among several factors that facilitate disease establishment within new host populations and the subsequent outcomes of disease **endemicity** and geographic spread. Lists of the primary factors involved have been developed for emerging and reemerging infectious diseases of humans (Table 2.20). Those concepts have also been extended to disease emergence affecting other species.[3–6,28,39,43,46,49,283,295–301]

Different factors are more important for some classes of disease agents (e.g., viruses and protozoan parasites) than others. For example, the absence of a suitable intermediate host for completion of a parasite life cycle can prevent the establishment of a highly pathogenic parasite following its introduction into a susceptible host population. In contrast, if a parasite or microbe that does not require an intermediate host is directly introduced into that same host population, the pathogen could become established and spread to additional populations and geographic areas. Other pathogens may require arthropod vectors for their development, transmission, or maintenance in nature. For example, the introduction of appropriate species of ticks can result in those ticks becoming reservoir hosts that sustain an indigenous pathogen between periods of disease epizootics/epidemics. Also, pathogen numbers may be amplified through biological multiplication within the body of the infected tick. When they take a blood meal, these ticks then transmit the pathogen to susceptible hosts.

Hawaii serves as a classic example of wildlife disease resulting from introduced arthropod vectors. **Mosquitoes** were introduced and became established during the early 1800s into these previously mosquito-free islands. They provided the vectors needed to sustain two important pathogens, avian poxvirus and the protozoan parasite responsible for avian malaria (*Plasmodium relictum*). Both pathogens became established in the Hawaiian Islands after the introduction of mosquito vectors. Pox was present by the late 1800s,[302] but malaria did not reach epizootic status until the early 1900s. Introduced exotic bird species, especially those from Asia, were the probable source for the malaria parasite.[303] Within native forest bird habitat, mosquitoes have become established because introduced pigs create mosquito-breeding habitat in the base of tree ferns that the pigs uproot.[304]

New York Department of Environmental Conservation file photo

**Figure 2.37** Hand lesions caused by streptothricosis.

Mosquitoes are mechanical vectors for avian pox. Conceptually, they are a "flying syringe" that transfers the virus taken in by the mosquito when they previously fed on infected birds. The same species of mosquitoes are biological vectors for avian malaria. In this situation, the mosquito is a required component for the development and multiplication of the malaria parasite and also is a vector for disease transmission. Avian malaria and, to a far lesser extent, avian pox have become limiting factors for populations of native birds on the islands of Hawaii.[302,303,305]

### *Pathogen Factors*

The development of pathogen genotypes better adapted for infection of humans and genotypes or phenotypes associated with a specific pathogenic capacity are factors in disease emergence and reemergence.[306] These attributes of the disease agent are not independently adequate for disease emergence to occur. The predictability of disease emergence in humans based on evaluations of only pathogenic agents is complicated by inadequate knowledge of the ecology of many known diseases and other factors. For example, the routes for transmission of over 200 human pathogens are unknown. Nevertheless, it has been shown that emerging diseases of humans are not caused by a random selection of pathogens. Zoonotic pathogens are overall twice as likely to be associated with emerging diseases than nonzoonotic pathogens. Also, viruses and protozoan parasites are especially likely to emerge as diseases of humans, while helminthes (parasitic worms) are very unlikely to emerge, regardless of their transmission routes or zoonotic status. The reasons for these outcomes have not been adequately determined. Genetic diversity, generation time, and existence of a reservoir for maintenance of the pathogen between periods of disease outbreaks are among the salient factors.[46]

#### Life as a Pathogen

The biologically relevant endpoints for pathogens are survival, proliferation, and transmission. These endpoints drive pathogen adaptation to their environment. Microbes generally have greater capacity to rapidly adjust to environmental changes than helminth parasites because of their greater genetic capabilities and much shorter generation times. Those capabilities and other attributes also provide microbes with evolutionary advantages over humans and other species that

**Table 2.20.** Primary factors associated with disease emergence and reemergence in humans.

| Category/factors[a] | Comments |
|---|---|
| **AGENT (PATHOGEN)** | |
| Microbial adaptation and change | • Includes selective pressures, mutations, evolution, and associated changes.[3–5,28,549] |
| Ability to cross species boundaries | • Invasion and establishment in nontraditional hosts of dissimilar species.[48,307,550] |
| Transmissibility, pathogenesis, and virulence | • Ability to invade hosts, cause disease, and be transmitted to new hosts.[28] |
| Survival and maintenance | • Environmental persistence during periods of disease quiescence. |
| **ENVIRONMENT** | |
| Ecological change | • Includes climate change and natural processes such as vegetation succession, seismic activity, fires, major flood events, and other weather related events that cause large-scale landscape impacts.[3,28,549] |
| Animal migration | • Natural cycles of animal movements such as seasonal movements of migratory birds, pursuit of water in arid regions, and movement to calving areas by large mammals.[549] |
| **HUMANS** | |
| Population | • Includes growth, distribution (changes in demography) and density (crowding).[4,5,549] |
| Behavior | • Includes sexual (e.g., AIDS), social, cultural, and other behaviors as well as attitudinal perspectives and actions.[3–5,549] |
| Urbanization | • Movement of people from rural to larger communities.[4,549] |
| Modern travel and commerce | • Movement of goods and people associated with international travel, ecotourism, and the global marketplace.[4,5,301,549–551] |
| Changes in agriculture and food practices | • Includes cropping patterns, methods of rearing animals for food production, aquaculture development, and food processing and packaging.[3,28,549,550] |
| Modern medicine | • Includes organ transplants, antibiotics, increasing longevity of human population, and other aspects of health care.[3,4,28,549] |
| Breakdown in public health infrastructure and measures | • Includes reductions in arbovirus surveillance and other activities and shifting emphasis away from infectious disease.[3–5,549] |
| Animal relocations | • Includes introductions of exotic species, human movements of agricultural species and wildlife (including fish), and commerce in companion animals (domestic and wild species).[5,549,550] |
| Environmental change | • Land-use impacts due to human actions such as deforestation, dam construction, large-scale agriculture, urban development, and the development of recreation areas.[3–5,28,550] |
| Societal events | • Includes war or civil conflict, urban decay, day care for children, and political actions that degrade standards of living.[3,28] |
| Technology and industry | • Increased speed of transportation (jet aircraft), water reclamation, medical capabilities, air conditioning, and other beneficial products and capabilities that have “side-effects” relative to disease emergence.[4,5,28,301] |

[a] Considerable overlap exists among factors within categories and categories are interactive with one another.

**Table 2.21.** Examples of emerging and reemerging infectious diseases of humans that have crossed species barriers.

| Disease | Type | Comments |
|---|---|---|
| Influenza | Virus | • Human infections involve viruses composed of a reassortment of genetic material from viruses infecting birds and domestic animals.[552–554] |
| | | • The 1957 and 1968 influenza pandemics contained genes derived from avian influenza viruses; the 1997 locally lethal occurrence of Hong Kong Flu and the 1999 Hong Kong isolates from two severe human cases of disease had all their eight gene segments of avian origin.[48] H5N1 avian influenza has killed poultry in nine Asian nations since its appearance in the late 1990s. Twenty-six people, domestic and large zoo cats, and swine also have died.[570] This virus could potentially evolve into one that can spread amongst humans, causing a pandemic.[571] |
| | | • Influenza B viruses that circulate among the human population have now been isolated from infected marine mammals (seals).[442] |
| AIDS | Virus | • About 35 million people worldwide have been infected by HIV-1 virus that originated in the chimpanzee; the sooty mangabey is the source of HIV-2 virus.[48] |
| B-virus (*Cercopithecine herpesvirus* 1) | Virus | • Only 40 human cases have been documented since the 1932 index case, but the case-fatality rate prior to the availability of antiviral therapy was greater than 70 percent.[555] |
| | | • Rhesus macaque and cynomolgus macaque are commonly found with B-virus infection and are commonly used in AIDS and other biomedical research. Other species also found infected.[555] |
| Marburg hemorrhagic fever | Virus | • Infrequently occurring deadly hemorrhagic fever first seen in 1967 among laboratory workers in Germany and Yugoslavia; all had handled tissues from African green monkeys.[556] |
| Ebola hemorrhagic fever | Virus | • First occurrence in 1976 in Zaire followed by epidemics elsewhere in 1995 and 1996; case-fatality rate reached 88 percent in initial event (280 deaths).[48,556] |
| | | • As with Marburg hemorrhagic fever, primates are associated with Ebola fever in humans, but the reservoir hosts are unknown.[48] |
| Hendra virus infection | Virus | • First appeared in Australia in 1994; fatalities in horses and a horse trainer. Reappearances in 1995 and 1996; fruit bats appear to be reservoir hosts.[557] |
| Nipah virus infection | Virus | • First appeared in Malaysia during 1998 to 1999; up to 40 percent case-fatality rate in people having close contact with sick pigs. Dogs and cats also died; fruit bats appear to be the reservoir hosts.[48,557] |
| Bovine spongiform encephalopathy (BSE or "mad cow disease") | Prion | • Documented in the United Kingdom in 1985 as a fatal disease of cattle; several species of zoo animals and cats died in 1990s following consumption of food containing material from infected cattle. First human case documented in 1995.[48] |

**Table 2.21.** Examples of emerging and reemerging infectious diseases of humans that have crossed species barriers—Continued.

| Disease | Type | Comments |
|---|---|---|
| Monkeypox | Virus | • First identified in 1970 in the Democratic Republic of the Congo (DRC); probably existed before but it was confused with smallpox. Only 14 cases in DRC from 1987 to 1992, none from 1993 to 1995; major resurgence of human cases since 1996 (more than 500).[558] The disease appeared in North America for the first time in 2003. |
| Hantaviruses | Virus | • Initial US event in 1993 among Native Americans in the Southwest. Deer mouse is the natural host and reservoir for the virus, which is shed in their urine and feces. Human infection is often fatal.[559,560] |
| Ehrlichiosis | Rickettsia | • Human monocytic ehrlichiosis (HME) first identified in USA in 1986, mainly occurs in southwestern and south central USA. Tick transmitted. White-tailed deer are primary definitive hosts for tick vectors.[561] |
| | | • Human granulocytic ehrlichiosis (HGE) first identified in USA in 1995, mainly occurs in Northeast and northern Midwest. Tick transmitted. White-footed mouse is primary reservoir host. |
| | | • White-tailed deer may be an important reservoir host for *Ehrlichia ewingii*, one of the several causative agents of ehrlichiosis.[562] |
| Leptospirosis | Bacteria | • Contaminated recreational waters becoming an increasing source for human infections. Reservioir hosts range from rodents to large mammals to marine species, wild and domestic.[48] |
| Babesiosis | Parasite | • Distinct species first observed in Eastern and Western USA in 1968; more than 200 cases in eastern USA since 1982 where white-footed mouse is primary reservoir host and white-tailed deer the definitive host for the tick vectors; blood transfusions also can transmit disease.[563] |
| | | • The western species (WAI-type *Babesia*) reappeared during the early 1990s; isolates from human cases from California are indistinguishable from those from mule deer and suggest large ungulates as the primary reservoir hosts.[563] |
| Tuberculosis | Bacteria | • Avian and fish strains (*Mycobacterium avium* and *M. marinum*) of the tuberculosis complex are generally of low virulence for humans, however, these strains can cause mortality in people with AIDS.[277] |

are increasingly being expressed as emerging diseases.[31] Those capabilities also have converted challenges for pathogen survival posed by antibiotics into opportunities for the emergence of antibiotic-resistant bacteria. Human actions that have resulted in the ubiquity of antimicrobials in the environment have been instrumental in facilitating the resulting evolutionary lessons that continue to occur on microbial adaptation and the power of natural selection in species with the population dynamics and genetic capabilities of microbes.[5,25,31]

The ability of pathogens to cross species boundaries is another important biological aspect of disease emergence. Free-ranging wildlife populations and humans have been victimized by such events.[307] Notable examples of pathogens crossing species boundaries include diseases in humans caused by viruses, rickettsia and other bacteria (Table 2.21), and parasites. The factors that influence the ability of each infectious agent to effectively cross the species barriers are poorly understood. However, human actions can create opportunities for species boundaries to be bridged.[48] In essence, humans set the table at which microbes and parasites feed.

### *The Human Factor*

In 1992, the Institute of Medicine published an insightful evaluation of disease emergence titled, "Emerging Infections: Microbial Threats to Health in the United States."[4] That evaluation addressed the primary factors driving infectious disease emergence. Human actions clearly are an important component in each of the six primary factors identified. To a large extent, these same factors apply to disease emergence in wildlife populations. Understanding and addressing this interconnectivity is important in combating zoonotic diseases and for minimizing the potential for the emergence of these types of diseases.

#### Human Demographics and Behavior

Population growth, density, and distribution have changed significantly in a manner that facilitates the transmission and maintenance of infectious disease within human populations. Worldwide, less than 1.7 percent of people lived in urban communities in 1800 compared to more than one-third by 1970 and one-half by 2000. This shift in demographics is also accompanied by increased population density in urban communities because of population growth; 225 cities reached population levels of over 1 million in 1985, and 445 cities reached that level by 2000. Twenty-five cities have populations exceeding 11 million people. Often, the infrastructure and economy of these large urban areas is insufficient to provide adequate living space, sanitation, and clean water for many of the inhabitants. Associated conditions of overcrowding, poor sanitation, and degraded environmental conditions facilitate the emergence of various pathogens and disease vectors such as mosquitoes. These factors have facilitated the emergence of dengue fever in the Americas.[4]

A somewhat analogous situation exists for North American waterfowl populations. The millions of ducks, geese, and swans that constitute this biological resource are typically migratory and gregarious species. Population maintenance is accomplished through annual cycles that involve breeding in northern areas followed by seasonal movements along general geographic corridors (referred to as "flyways") to wintering areas and then back to the breeding areas. The greatest numbers of waterfowl are found in the Pacific Flyway, where millions of these birds begin to move southward each fall. Historically, there was an abundance of wetlands available to provide for resting, feeding, and wintering areas along this annual journey. However, between the 1780s and the mid-1980s, 22 of the conterminous 48 states within the USA drained, filled, or otherwise destroyed more than 50 percent of their wetlands. California leads the nation with a loss of 91 percent of its historic wetlands.[308] The significant degradation of an additional 4 percent of the remaining wetlands results in an effective loss of 95 percent of the historic habitat base for **migratory birds** dependent on wetlands. More than one-half of the 7 million waterfowl wintering in the Pacific Flyway depended on California wetlands during 2000.[309]

Wetland loss has resulted in dense aggregations of waterfowl on the remaining habitat for prolonged periods of time (Fig. 2.38). Fecal contamination from these birds is extensive and degrades the water quality of their habitat.[310] These situations facilitate the transmission of infectious agents shed in the feces. Waterfowl also can be exposed to infectious agents present in wastewater that has not been adequately treated. Birds make heavy use of sewage lagoons and other wastewater sites as feeding and loafing areas. In addition, historic migratory patterns have been altered to the extent that Canada geese and some other species have established nonmigratory urban/suburban populations that make continuous use of the same water bodies (Fig. 2.39). These altered environmental conditions have substantially contributed to the unprecedented occurrence of infectious disease as a major mortality factor in migratory birds. Prior to the 1970s, infectious disease was infrequently observed among free-ranging waterfowl populations and seldom accounted for large-scale epizootics, such as those that now commonly kill thousands to tens of thousands of birds per event.[8]

Photo by Milton Friend

**Figure 2.38** Waterfowl often gather in dense groups.

Photo by Milton Friend

**Figure 2.39** Wild waterfowl are becoming increasingly urbanized.

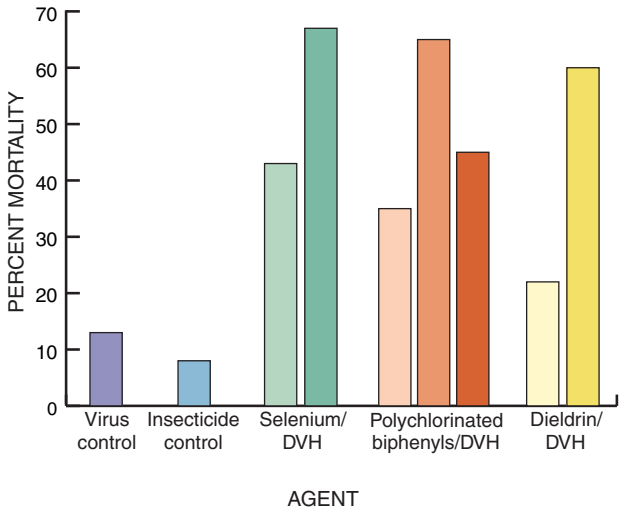



**Figure 2.40** Pre-exposure to chemical agents increases susceptibility of mallard ducklings to duck virus hepatitis (DVH). Darker shades reflect higher dosage of agent.

Immunosuppression is another factor that influences the occurrence of infectious disease in humans.[4] For waterfowl, malnutrition associated with altered diets and the potential immunological effects of pesticide exposure threaten their populations. Replacement of wetlands with large-scale agriculture has resulted in a shift to cereal grains as a primary food source for some waterfowl species. These high-energy foods do not meet the full dietary requirements for waterfowl and this inadequacy can increase susceptibility to disease agents. A classic situation is lead poisoning. The toxic effects of ingested lead shot are increased in birds feeding on corn rather than natural foods.[311]

Exposure to pesticides by waterfowl and other wildlife is well documented and often postulated to be an interactive factor with microbes that enhances wildlife susceptibility to mortality. The potential for such occurrences have been demonstrated experimentally by using a duck virus hepatitis-mallard duck model (Fig. 2.40).[312–316] Concurrent disease is another condition that occurs in wildlife as well as humans. Aspergillosis (*Aspergillus fumigatus*) is a common cause of death in Canada geese and swans (Fig. 2.41) that have been incapacitated by lead poisoning.[317]

### Technology and Industry

Modern medicine, food processing and handling, and water treatment are primary components contributing to disease emergence in humans as a result of technology and industry. **Nosocomial** (hospital acquired) infections are a major "side effect" of modern medicine that results in an estimated 2 million cases and 20,000 deaths annually in the USA.[4] Antibiotic resistance is a major part of this problem. Technological changes in agriculture, food processing, and food handling to provide greater yields, operational efficiencies, and other benefits have also had emerging disease "side-effects." Feedlots, large-scale poultry operations, and the growth of aquaculture have all provided new environmental opportunities for human pathogens. Hamburger and *Escherichia coli* infections and poultry and *Salmonella enteritidis* are examples of disease emergence associated with the food industry.[4]

*Wildlife Rehabilitation*—Public values associated with the well-being of wildlife have resulted in a large number of independent, largely private sector, wildlife rescue and rehabilitation programs where oiled, injured, and other afflicted wildlife are brought for treatment. Wildlife with infectious disease are commonly among the animals submitted. The opportunity for wildlife to acquire "nosocomial infections" in these facilities is substantial because of the physical limitations and other inadequacies often present relative to disease **containment**. The return of infected, but clinically inapparent wildlife, to nature may be a source for disease introductions and epizootics.

*Antibiotic Resistance*—The role of wildlife as contributors to the development of antibiotic-resistant pathogens has not been seriously explored and questions remain. Wildlife are exposed to antibiotic use in wildlife rehabilitation programs and in nature. Antibiotics enter waters that are feeding areas for wildlife and also may be present in poultry and other domestic animal wastes spread on fields where wildlife feed. Some species of birds actively feed on materials present in fresh feces of cattle and some feed among livestock in feedlot operations (Fig. 2.42). Therefore, considerable opportunity exists for some species of wildlife to become exposed to antibiotics. These same wildlife may be involved in the maintenance and transmission of disease agents that affect domestic animals and humans. The effects of antibiotic exposures of wildlife on the pathogenicity of the myriad of microbes and parasites that wildlife share with other species are unknown.

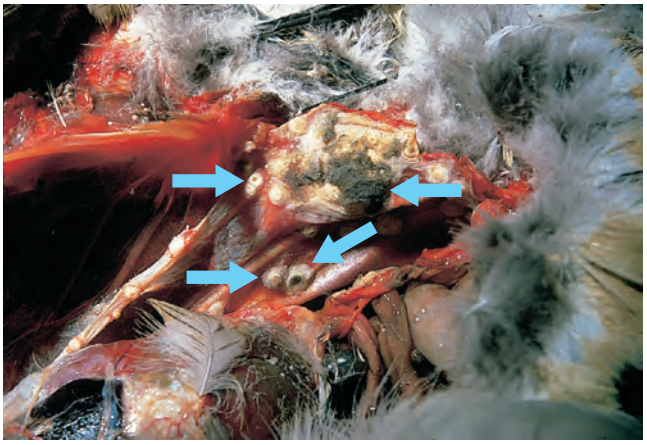


Photo by Milton Friend

**Figure 2.41** "Cheesy" plaques and fungal growth in the lungs and air sacs of a bird with aspergillosis.


Photos by Milton Friend

**Figure 2.42** (A) Wild birds, such as sandhill cranes, often feed among cattle. (B) Wild birds also congregate near cattle feedlots, may feed among the cattle, or feed in adjacent habitat receiving waste runoff from the feedlot.

*Aquaculture and Disease Emergence*—Aquaculture has become an increasing source of the human foodbase because of shifting dietary preferences and the inability of natural stocks of finfish and shellfish to supply the needs of the growing human population. In the USA there is large-volume fish consumption, with prices, in some cases, exceeding those for beef, even for species that a half-century ago were considered "trash fish" eaten only by those of low economic status. The growth of aquaculture as a greater component of world food supplies has been stimulated by increased product value, allowing enhanced investments by that industry. These investments lead to technological advances providing an increasing array of shellfish and finfish at marketable volumes that provide adequate economic returns to stimulate further production. Aquaculture contributes up to 15 percent of the seafood utilized in the USA[251] and supplies one-third of the seafood consumed worldwide.[318]

The continued growth of the aquaculture industry is expected, as are emerging diseases as a component of this form of agriculture.[251] For example, in 1987 several hundred persons in the Montreal area of Quebec, Canada, became ill and several died from eating cultured blue mussels. This malady was termed "neurovisceral toxic syndrome" and was found to be a domoic acid toxin associated with the pennate **diatom** *Nitzschia pungens*. That event was the first recognized occurrence of this shellfish-induced toxic syndrome.[319,320]

The most significant diseases of mollusks in cultured and wild stocks have been linked to the introduction and transfer of infectious agents. During recent years, many disease problems in these species appear to be related to changing culture techniques and the diversification of species under culture.[81] Similarly, in less than 30 years, the **penaeid shrimp** culture industries of the world have grown from an experimental beginning into major businesses.[79] Shrimp aquaculture grew by 430 percent between 1985 to 1994 and a high rate of growth continues.[80] This growth has been accompanied by recognition of a host of previously unknown infectious disease agents.[79] Fortunately, for human health, few of these pathogens are zoonoses; however, their emergence commonly impacts wild stocks of the same or similar species. Ecological damage resulting from these diseases can be substantial with staggering economic losses.[79]

Farming of the Chinese or soft-shelled turtle has also developed rapidly in recent years and has been accompanied by an increasing number of diseases. A new iridovirus with the proposed name of soft-shelled turtle iridovirus was found to be the cause of an epizootic of "red neck disease" on a turtle farm in China.[321]

### Economic Development and Land Use

Changes in land use commonly are associated with settlement of wild lands or economic development activities. The resulting landscape changes alter the habitat base for **vertebrates** and invertebrates as well as species interactions. For example, the geographic prevalence of tularemia within the USA has shifted greatly during recent decades, in part due to landscape changes that have altered the habitat base for the mammalian hosts and tick vectors for this disease.

Dam building and reforestation provide examples of landscape changes resulting in the emergence of diseases impacting humans. Mosquito-borne Rift Valley fever (RVF) is primarily a disease of sheep and cattle. This disease had only been known to occur in Africa south of the Sahara. In 1977, following the completion of the Aswan Dam, RVF caused an estimated 200,000 human cases of clinical illness and nearly 600 deaths in Egypt. In 1987, following the completion of the Diama Dam, RVF caused more than 1,200 cases of severe

illness and nearly 250 deaths in the Senegal River Basin. Sheep and cattle were also affected during both events. In both situations, dam building contributed to the emergence of RVF by providing breeding habitat for the mosquitoes that transmit this disease.[4]

Lyme disease (*Borrelia burgdorferi*) is transmitted by several species of *Ixodes* ticks. In the Midwestern and Eastern USA, the white-footed mouse is the reservoir host for the causative bacterium and white-tailed deer are definitive hosts for the tick vectors. Early development in areas east of the Mississippi River resulted in extensive deforestation and the demise of deer due to the loss of their woodland and forest habitat. Changes in land use that followed the movement of much of agriculture westward, resulted in dramatic reforestation that exceeds the forest cover at the time of settlement. Changes in lifestyles have resulted in the development of suburban communities within these reforested areas. High deer and mouse populations have accompanied the reforestation. The close proximity of human hosts (living in those reforested areas) to the ticks that transmit Lyme disease add to this problem. Lyme disease has become the most common **vector-borne disease** in the USA and it has been reported in all 50 states.[4]

Disease emergence impacting free-ranging wildlife populations has also occurred from economic development and land use. Agriculture practices have directly contributed to disease emergence in wildlife as a result of cropping patterns and the replacement of natural foods with grain as a primary wildlife foodbase. Agricultural crop-related diseases of wild birds include aflatoxicosis,[322] castor bean poisoning,[323] and enterotoxemia due to *Clostridium perfringens*.[324] A wintering area for sandhill cranes in the Texas/Oklahoma panhandles of the USA was developed for peanut farming, which resulted in the emergence of mycotoxicosis as a cause of mortality for these birds. The climate of that area is conducive to freeze-thaw cycles, a condition required for growth of the causative fungi and production of toxin. Waste peanuts left from harvest provide the growth medium for the fungi and cranes then eat the toxic peanuts (Fig. 2.43). Approximately 10,000 sandhill cranes died during the initial epizootics.[325]

The development of livestock and poultry operations has provided various interfaces that have facilitated the transfer and emergence of infectious diseases of commercially raised animals into free-ranging wildlife populations. Avian cholera (*Pasteurella multocida*) in wild birds and brucellosis (*Brucella abortus*) in bison and elk are only two of many diseases that can be cited. Aquaculture has provided a host of diseases from finfish and shellfish culture that have emerged in wild populations of these species, such as whirling diseases (*Myxobolus cerebralis*) in rainbow trout and Taura syndrome virus in shrimp.

Agriculture also affects wetland water quality. Nutrient loading from fertilizer residuals contributes to **eutrophication** that results in algal blooms, some of which have been associated with wildlife toxicity.[62,326–328] Water quality is closely associated with the increased geographic distribution of type C avian botulism (*Clostridium botulinum*) within the USA and globally.[329,330] Wastewater treatment ponds, stormwater runoff into wetlands, and wastewater discharges from the processing of agricultural commodities have all been associated with avian botulism epizootics.

### International Travel and Commerce

The emergence of infectious disease as a "by-product" of the movement of people and goods from one region to another is well documented throughout human history. The introduction of smallpox into the New World and syphilis into the Old World are classic diseases associated with human travel. The movement of infected animals and arthropod vectors of disease into new regions has occurred through commerce, often as hitchhikers present in the transport vessels.[4] For example, introduction of the Asian tiger mosquito into the USA occurred in used tires, rats infected with disease have entered various ports from ships that began their voyages in other parts of the world, and the discharge of ballast water is thought to be the source of the cholera (*Vibrio cholerae*) outbreak in the Americas that began in 1991. Also, the speed of modern transportation facilitates the movement of diseases between continents by travelers who are incubating serious infections, such as **SARS**. Contacts with people along the way at the time when infected individuals are shedding disease agents further enhance the potential for disease distribution.[301]

Wildlife also are moved regionally and internationally by human actions. The raccoon rabies epizootic of the eastern USA is the result of a translocation of raccoons for sporting purposes.[537] Inclusion body disease of cranes was likely brought into the USA in zoological collections or in cranes needed for captive breeding programs. Diseases of finfish and shellfish have been moved between continents in founder stocks for aquaculture and by trade in aquarium fish. Bovine tuberculosis and malignant catarrhal fever have jeopardized captive-breeding populations of endangered spe-

Photo by Ronald Windingstad

**Figure 2.43** Sandhill crane that died from eating peanuts contaminated with mycotoxins.

cies of wild **ruminants** following the international movement of infected animals into those breeding populations. Other disease introductions and the emergence of novel diseases for captive and free-ranging wildlife populations also have occurred by this means.

### Microbial Adaptation and Change

The adaptation of microbes to their environment is highly complex, involving such components as natural variation or mutation by microbes (e.g., influenza A virus), and selective pressure and the development of resistance in known infectious agents (e.g., multidrug-resistant tuberculosis); and microbes acting as cofactors in chronic disease (e.g., *Chlamydia pneumoniae* and atherosclerosis).[4] It is reasonable to assume that similar adaptations are occurring within wildlife populations. Examples have been cited within this chapter of classic pathogens (e.g., canine distemper virus emerging as a cause of disease in new hosts, such as lions and marine mammals). Historic diseases such as rabies are now known to have strain variants that are adapted to specific groups of animals. Continual opportunities for adaptation are provided to microbes by the plethora of introductions of exotic species of vertebrates and invertebrates associated with human induced landscape changes and other actions. New species interactions including the potential for transfer of microbes and parasites between naive hosts and the involvement of new arthropod vectors are potential outcomes from these introductions. Human engineered, newly created major ecosystems appear within short time frames around the globe as a result of dams, deforestation, and urban development. In addition, alteration of the gene pool for some vertebrate species as a result of large-scale releases of captive-reared animals is another potential opportunity for microbes to exploit.

### Breakdown of Public Health Measures

The reappearance of diseases such as cholera (*Vibrio cholerae*) due to inadequate sanitation, measles due to complacency towards immunization, and a host of other diseases associated with the conditions of war and postwar periods are components of the public health infrastructure factor.[4] A recent vivid example is the devastating diphtheria epidemic that occurred in Russia following the breakdown of the Soviet Union and its transition to other forms of government. When the epidemic began in 1990, reported diphtheria (*Corynebacterium diphtheriae*) cases increased from 603 in 1989 to 47,802 in 1994 causing 746 deaths that year.[333] More than 80,000 people were infected and more than 2,000 died by early 1995.[332] In addition, several major outbreaks of tularemia have occurred in postwar Bosnia and other nearby areas associated with that conflict. Contamination of water supplies by diseased animals is thought to be the source for those events.[331] The 1993 cryptosporidiosis outbreak in Milwaukee, Wisconsin, USA due to failure of water treatment, resulted in 403,000 human cases of this emerging disease.[247,336]

Unlike human and domestic animal health, there is no comparable infrastructure for wildlife health. Short-term crisis response is the general action the wildlife conservation community takes. As a result, infectious diseases may be able to establish a foundation for their perpetuation before they become recognized as emerging infections. During recent decades, several multidimensional programs devoted to addressing disease in free-ranging wildlife populations have been developed within government agencies and the university community (Table 2.22). These programs complement other programs within wildlife conservation agencies that respond at various levels to wildlife health issues. While these programs have continually demonstrated their value, they remain few in number, small in size, and isolated from the much larger programs developed to address infectious diseases in humans and domestic animals. To help combat wildlife disease, there is a need to enhance infrastructure and wildlife programs to provide essential information on wildlife diseases. The need for enhanced coordination and integration of efforts is evident from the high percentage of zoonoses that are of wildlife origin and the increasing interface between humans and other species that will continue to occur as a result of human population growth and associated landscape changes.

*Milton Friend*

**Table 2.22.** Major North American wildlife disease programs (free-ranging wildlife).[a]

| Program | Type | Comments |
|---|---|---|
| Canadian Cooperative Wildlife Health Center | Cooperative | • Four regional programs operate out of the Schools of Veterinary Medicine, University of Prince Edward Island; University of Guelph; Université de Montréal; and University of Saskatchewan.<br>• Program support and interfaces with Environment Canada (federal), Provincial and Territorial wildlife departments, other government agencies, and private sector organizations.<br>• Conduct laboratory and field investigations to determine causes of wildlife mortality; research to resolve the ecology of various diseases; public outreach and education; and train students. |
| National Wildlife Health Center | Federal | • Federally funded science program of the U.S. Geological Survey; headquarters in Madison, Wisconsin, with a field station in Honolulu, Hawaii.<br>• Government owned and operated site with biosecurity level-3 facilities for both laboratory and live animal investigations.<br>• Conduct laboratory and field investigations to determine causes of wildlife mortality; research to resolve the ecology of various diseases; public outreach and education; and train students. |
| Northeastern Research Center for Wildlife Diseases | Cooperative | • Established within the Department of Pathobiology, University of Connecticut, Storrs, Connecticut.<br>• Program support provided by numerous sources, including member state wildlife agencies.<br>• Conduct laboratory and field investigations to determine causes of wildlife mortality; research to resolve the ecology of various diseases; public outreach and education; and train students. |
| Southeastern Cooperative Wildlife Disease Study | Cooperative | • Established within the School of Veterinary Medicine, University of Georgia, Athens, Georgia.<br>• Program support provided by numerous sources, including southern and eastern member state wildlife agencies.<br>• Conduct laboratory and field investigations to determine causes of wildlife mortality; research to resolve the ecology of various diseases; public outreach and education; and train students. |

[a]Does not include programs devoted solely to fish disease or small scale wildlife disease programs such as those carried out by a number of state wildlife agencies.

## Literature Cited


1. McNeill, W.H., 1976, Plagues and peoples: New York, Anchor Press, 369 p.
2. Meslin, F.X., Stohr, K., and Heymann, D., 2000, Public health implications of emerging zoonoses: Revue Scientifique et Technique, Office International des Epizooties, v. 19, p. 310–317.
3. Centers for Disease Control and Prevention, 1994, Addressing emerging infectious diseases threats: a prevention strategy for the United States: Atlanta, Ga., CDC, National Center for Infectious Diseases, 46 p.
4. Lederberg, J., Shope, R.E., and Oaks, S.C., Jr., eds., 1992, Emerging infections: microbial threats to health in the United States: Institute of Medicine, Washington, D.C., National Academy Press, 294 p.
5. Morse, S.S., 1995, Factors in the emergence of infectious diseases: Emerging Infectious Diseases, v. 1, p. 7–15.
6. Daszak, 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.
7. Friend, M., 1995, Increased avian diseases with habitat change, *in* LaRoe, E.T., and other eds., Our living resources—a report to the nation on the distribution, abundance, and health of U.S. plants, animals, ecosystems: Washington, D.C., U.S. Department of the Interior, National Biological Service, p. 401–404.
8. Friend, M., McLean, R.G., and Dein, J.F., 2001, Disease emergence in birds: challenges for the twenty-first century: The Auk, v. 118, p. 290–303.
9. Guralinik, D.B, ed., 1982, Webster's new world dictionary (2nd college ed.): New York, Simon and Schuster, 540 p.
10. Bradley, G.A., Rosen, P.C., Sredl, M.J., Jones, T.R., and Longcore, J.E., 2002, Chytridiomycosis in native Arizona frogs: Journal of Wildlife Diseases, v. 38, p. 206–212.
11. Berger, L., Speare, R., Daszak, P., Green, D.E., Cunningham, A.A., Goggin, C.L., Slocombe, R., Ragan, M.A., Hyatt, A.D., McDonald, K.R., Hines, H.B., Lips, K.R., Marantelli, G., and Parkes, H., 1998, Chytridiomycosis causes amphibian mortality associated with population declines in the rainforests of Australia and Central America: Proceedings of the National Academy of Sciences, v. 95, p. 9031–9036.
12. Daszak, P., Berger, L., Cunningham, A.A., Hyatt, A.D., Green, D.E., and Speare, R., 1999, Emerging infectious diseases and amphibian population declines: Emerging Infectious Diseases, v. 5, p. 735–748.
13. Rocke, T.E., and Friend, M., 1999, Avian botulism, *in* Friend, M., and Franson, J. C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 271–281.
14. 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, *in* Shuford, W.D., and Molina, K.C., eds., Ecology and conservation of birds of the Salton Sink: an endangered ecosystem: Studies in Avian Biology, v. 27, p. 136–140.
15. Dolman, C.E., and Iida, H., 1963, Type E botulism: its epidemiology, prevention and specific treatment: Canadian Journal of Public Health, v. 54, p. 293–307.
16. National Wildlife Health Center, 2003, Epizootiological database: Madison, Wis., U.S. Geological Survey.
17. Brand, C.J., Duncan, R.M., Garrow, S.P., Olson, D., and Schumann, L.E., 1983, Waterbird mortality from botulism type E in Lake Michigan: an update: Wilson Bulletin, v. 95, p. 269–275.
18. Brand, C.J., Schmitt, S.M., Duncan, R.M., and Cooley, T.M., 1988, An outbreak of type E botulism among common loons (*Gavia immer*) in Michigan's Upper Peninsula: Journal of Wildlife Diseases, v. 24, p. 471–476.
19. Dowell, V.R., Jr., Gangarosa, E.J., and Armstrong, R.W., 1970, Clinical and epidemiological aspects of botulism in the United States, in Herzber, M., ed., Proceedings First U.S.-Japan Conference on Toxic Micro-organisms, Mycotoxins, Botulism: Washington, D.C., U.S.-Japan Cooperative Program in Natural Resources and U.S. Department of the Interior, p. 360–364.
20. Sugiyama, H., Bott, T.L., and Foster, E.M., 1970, *Clostridium botulinum* type E in an inland bay (Green Bay of Lake Michigan), *in* Herzberg, M., ed., Proceedings of the First U.S.-Japan Conference on Toxic Micro-organisms, Mycotoxins, Botulism:Washington, D.C., U.S.-Japan Cooperative Program in Natural Resources and U.S. Department of the Interior, p. 287–291.
21. Domske, H.M., Lichtkoppler, F.R., and Obert, E.C., 2002, Botulism in Lake Erie, Workshop Proceedings: Buffalo, N.Y., Feb. 2002, New York Sea Grant, 65 p.
22. Lederberg, J., 2000, Infectious history: Science, v. 288, p. 287–293.
23. Dubos, R., 1965, Man adapting: New Haven, Conn., Yale University Press, 527 p.
24. Ewald, P.W., 1994, Evolution of infectious disease: New York, Oxford University Press, 298 p.
25. Lederberg, J., 1998, Emerging infections: an evolutionary perspective: Emerging Infectious Diseases, v. 4, p. 366–371.
26. Fenner, F., and Ratcliffe, F.N., eds., 1965, Myxomatosis: Cambridge, UK, Cambridge University Press, 379 p.
27. Fenner, F., 1953, Changes in the mortality rate due to myxomatosis in the Australian wild rabbit: Nature, v. 172, p. 228.
28. Murphy, F.A., 1994, New, emerging, and reemerging infectious diseases: Advances in Virus Research, v. 43, p. 1–52.
29. Fenner, F., and Myers, K., 1978, Myxoma virus and myxomatosis in retrospect: the first quarter century of a new disease, *in* Kurstak, E., and Maramorosch, K., eds., Viruses and environment: New York, Academic Press p. 539–570.
30. Mutze, G., Bird, P., Kovaliski, J., Peacock, D., Jennings, S., and Cooke, B., 2002, Emerging epidemiological patterns in rabbit haemorrhagic disease, its interaction with myxomatosis, and their effects on rabbit populations in South Australia: Wildlife Research, v. 29, p. 577–590.
31. Lederberg, J., 1997, Infectious disease as an evolutionary paradigm: Emerging Infectious Diseases, v. 3, p. 417–423.
32. Palumbi, S.R., 2001, Humans as the world's greatest evolutionary force: Science, v. 293, p. 1786–1790.
33. Ayensu, E., Claasen, D.V.R., Collins, M., Dearing, A., Fresco, L., Gadgil, M., Gitay, H., Glaser, G., Juma, C., Krebs, J., Lenton, R., Lubchenco, J., McNeely, J.A., Mooney, H.A., Pinstrup-Andersen, P., Ramos, M., Raven,

P., Reid, W.V., Samper, C., Sarukhán, J., Schei, P., Tundisi, J.G., Watson, R. T., Guanhua, X., and Zakri, A.H., 1999, International ecosystem assessment: Science, v. 286, p. 685.
34. Vitousek, P.M., D'Antonio, C.M., Loope, L.L., Rejmanek, M., and Westbrooks, R., 1997, Introduced species: a significant component of human-caused global change: New Zealand Journal of Ecology, v. 21, p. 1–16.
35. Cohen, M.L., 2000, Changing patterns of infectious disease: Nature, v. 406, p. 762–767.
36. Nathanson, N., and Fine, P., 2002, Poliomyelitis eradication—a dangerous endgame: Science, v. 296, p. 269–270.
37. Armstrong, G.L., Conn, L.A., and Pinner, R.W., 1999, Trends in infectious disease mortality in the United States during the 20th century: Journal of American Medical Association, v. 281, p. 61–66.
38. DaSilva, E., and Iaccarino, M., 1999, Emerging diseases: a global threat (a review): Biotechnology Advances, v. 17, p. 363–384.
39. Levins, R., Awerbuch, T., Brinkmann, U., Eckardt, I., Epstein, P., Makhoul, N., Albuquerque de Possas, C., Puccia, C., Spielman, A., and Wilson, M.E., 1994, The emergence of new disease: lessons learned from the emergence of new diseases and the resurgence of old ones may help us prepare for future epidemics: American Scientist, v. 82, p. 52–60.
40. Murphy, F.A., and Nathanson, N., 1994, The emergence of new virus diseases: an overview: Seminars in Virology, v. 5, p. 87–102.
41. Hughes, J.M., 1998, Addressing emerging infectious disease threats—accomplishments and future plans: Emerging Infectious Diseases, v. 4, p. 360–361.
42. Dubos, R., 1959, Mirage of health: utopias, progress, and biological change, *in* Anshon, R.N., ed., World perspectives: New York, Harper & Brothers, v. 22, 236 p.
43. Daszak, P., Cunningham, A.A., and Hyatt, A.D., 2001, Anthropogenic environmental change and the emergence of infectious diseases in wildlife: Acta Tropica, v. 78, p. 103–116.
44. Patz, J.A., Graczyk, T.K., Geller, N., and Vittor, A.Y., 2000, Effects of environmental change on emerging parasitic diseases: International Journal for Parasitology, v. 30, p. 1395–1405.
45. Conover, M., ed., 2002, Zoonoses, *in* Resolving human-wildlife conflicts: Boca Raton, Fla., Lewis Publishers, p. 67–89.
46. 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.
47. Thompson, R.C.A., 2000, Emerging parasite zoonoses: International Journal of Parasitology, v. 30, p. iv.
48. 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.
49. Cleaveland, S., Laurensen, 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, v. 356, p. 991–999.
50. Rocke, T.E., and Friend, M., 2003, Wildlife disease in the Colorado Delta as an indicator of ecosystem health, *in* Rapport, D.J., and others, eds., Managing for healthy ecosystems: Boca Raton, Fla., Lewis Publishers, p. 1111–1124.
51. Centers for Disease Control and Prevention, 1996, Invasive infection with *Streptococcus iniae*, Ontario, 1995–1996: Morbidity and Mortality Weekly Report, v. 45, p. 650–653.
52. Cooper, J.E., 1990, Birds and zoonoses: Ibis, v. 132, p. 181–191.
53. Keymer, I.F., 1958, A survey and review of the causes of mortality in British birds and the significance of wild birds as disseminators of disease—part I: The Veterinary Record, v. 69, p. 713–720.
54. Keymer, I.F., 1958, A survey and review of the causes of mortality in British birds and the significance of wild birds as disseminators of disease—part II: The Veterinary Record, v. 70, p. 736–740.
55. Nice, C.S., 1994, The dissemination of human infectious disease by birds: Reviews in Medical Microbiology, v. 5, p. 191–198.
56. Cooper, J.E., 1985, Medico-veterinary collaboration, a review of its importance and relevance, especially in the tropics: Tropical Doctor, v. 15, p. 187–197.
57. Ampel, N.M., 1996, Emerging disease issue and fungal pathogens associated with HIV infection: Emerging Infectious Diseases, v. 2, p. 109–116.
58. Miller, R.F., 2000, Clinical presentation and significance of opportunistic infections: Journal of Eukaryotic Microbiology, v. 47, p. 21–23.
59. Thompson, R.C.A., 2001, The future impact of societal and cultural factors on parasitic disease—some emerging issues (a review): International Journal of Parasitology, v. 31, p. 949–959.
60. Robinson, R.A., 2000, Zoonoses and immunosuppressed populations, *in* Macpherson, C.N.L., and others, eds., 2000, Dogs, zoonoses and public health: Oxon, UK, CBI publishing, p. 273–298.
61. Sinderman, C.J., 1963, Disease in marine populations: Transactions of the North American Wildlife and Natural Resources Conference, v. 28, p. 336–356.
62. Epstein, P., ed., 1998, Marine ecosystems: emerging diseases as indicators of change: Year of the Ocean Special Report On Health of the Oceans from Labrador to Venezuela, Harvard Medical and the Global Environment, Boston, Mass., December 1998, 85 p.
63. Porter, J.W., ed., 2001, Preface: Hydrobiologia, v. 460, p. ix–xvi.
64. Robblee, M., Barber, T.R., Carlson, P.R., Jr., Durako, M.J., Fourqurean, J.W., Muehlstein, L.K., Porter, D., Yarbro, L.A., Zieman, R.T., and Zieman, J.C., 1991, Mass mortality of the tropical sea grass *Thalassia testudinum* in Florida Bay (USA): Marine Ecology Progress Series, v. 71, p. 297–299.
65. Short, F.T., Muehlstein, L.K., and Porter, D., 1987, Eelgrass wasting disease: cause and recurrence of a marine epidemic: Biology Bulletin, v. 173, p. 557–562.
66. Spalding, M.D., Ravilious, C., and Green, E.P., 2001, World atlas of coral reefs: Berkeley, Calif., University of California Press, 424 p.
67. Field, M.E., Cochran, S.A., and Evans, K.R., 2002, U.S.

coral reefs—imperiled national treasures: U.S. Geological Survey Fact Sheet 025–02.
68. Stokstad, E., 2001, Humans to blame for coral loss: Science, v. 293, p. 593.
69. Aronson, R.B., Precht, W.F., Macintyre, I.G., and Murdoch, T.J.T., 2000, Coral bleach-out in Belize: Nature, v. 405, p. 36.
70. Aronson, R.B., and Precht, W.F., 2001, Evolutionary paleoecology of Caribbean coral reefs, *in* Allmon, W.D., and Bottjer, D.J., eds., Evolutionary paleoecology: the ecological context of macroevolutionary change: New York, Columbia University Press, p. 171–233.
71. Hughes, T.P., 1994, Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef: Science, v. 265, p. 1547–1551.
72. Hayes, R.L., and Goreau, N.I., 1998, The significance of emerging diseases in the tropical coral reef ecosystem: Revista de Biología Tropical, v. 46, p. 173–185.
73. Littler, M.M., and Littler, D.S., 1995, Impact of CLOD pathogen on Pacific coral reefs: Science, v. 267, p. 1356–1359.
74. Brown, B., 1997, Coral bleaching: causes and consequences: Proceedings of the International Coral Reef Symposium, v. 8, p. 65–74.
75. Woodley, J.D., Bone, D., Buchan, K., Bush, P., De Meyer, K., Garzon-Ferreira, J., Gayle, P., Gerace, D.T., Grober, L., Klein, E., Koltes, K., Losada, F., McFeild, M.D., McGrath, T., Mendes, J.M., Nagelkerken, I., Ostrander, G., Pors, L.P.J.J., Rodriquez, A., Rodriquez, R., Ruiz-Renteria, F., Smith, G., Tschirky, J., Alcolado, P., Bonair, K., Garcia, J.R., Geraldes, F.X., Guzman, H., Parker, C., and Smith, S.R., 1997, Studies on Caribbean coral bleaching, 1995–96: Caribbean Coastal Marine Productivity Programme, Proceedings of the International Coal Reef Symposium, v. 8, p. 673–678.
76. Harvell, D., Kim, K., Quirolo, C., Weir, J., and Smith, G., 2001, Coral bleaching and disease: contributors to 1998 mass mortality in *Briareum asbestinum* (Octocorallia, Gorgonacea): Hydrobiologia, v. 460, p. 97–104.
77. Normile, D., 2000, Reef migrations, bleaching effects stir the air in Bali: Science, v. 290, p. 1282–1283.
78. Hedrick, R.P., 1998 Emerging diseases of fish: Proceedings of the International Symposium on Aquatic Animal Health, v. 3., September, Baltimore, Md.
79. Lightner, D.V., 1996, Epizootiology, distribution and the impact on international trade of two penaeid shrimp viruses in the Americas: Revue Scientifique et Technique, Office International des Epizooties, v. 15, p. 579–601.
80. Lightner, D.V., and Redman, R.M., 1998, Emerging crustacean diseases: Proceedings of the International Symposium on Aquatic Animal Health, v. 3, September, Baltimore, Md.
81. McGladdery, S.E., 1998, Emerging molluscan disease: Proceedings of the International Symposium on Aquatic Animal Health, v. 3, September, Baltimore, Md.
82. Fryer, J.L., and Mauel, M.J., 1997, The rickettsia—an emerging group of pathogens in fish: Emerging Infectious Diseases, v. 3, p. 137–144.
83. Meyers, T.R., and Winton, J.R., 1995, Viral haemorrhagic septicaemia virus in North America: Annual Review of Fish Diseases, v. 5, p. 3–24.
84. Ross, K., McCarthy, U., Huntly, P.J., Wood, B.P., Stuart, D., Rough, E.I., Smail, D.A., and Bruno, D.W., 1994, An outbreak of viral haemorrhagic septicaemia (VHS) in turbot (*Scophthalmus maximus)* in Scotland: Bulletin of the European Association of Fish Pathologists, v. 14, p. 213–214.
85. Dixon, P.F., Feist, S., Kehoe, E., Parry, L., Stone, D.M., and Way, K., 1997, Isolation of viral haemorrhagic septicaemia virus from Atlantic herring *Clupea harengus* from the English Channel: Diseases of Aquatic Organisms, v. 30, p. 81–89.
86. Office International des Epizooties, 2000, Diagnostic manual for aquatic animal diseases (3rd ed.): Paris, Office International des Epizooties, 237 p.
87. Nakajima, K., Maeno, Y., Kurita, J., and Inui, Y., 1997, Vaccination against red sea bream iridoviral disease in red sea bream: Fish Pathology, v. 32, p. 205–209.
88. Fryer, J.L, and Bartholomew, J.L., 1996, Established and emerging infectious diseases of fish: as fish move, infections move with them: ASM News, v. 62, p. 592–594.
89. Chern, R.S., and Chao, C.B., 1994, Outbreaks of a disease caused by rickettsia-like organism in cultured tilapias in Taiwan: Fish Pathology, v. 29, p. 61–71.
90. Hyatt, A.D., Hine, P.M., Jones, J.B., Whittington, R.J., Kearns, C., Wise, T.G., Crane, M.S., and Williams, L.M., 1997, Epizootic mortality in the pilchard (*Sardinops sagax neopilchardus*) in Australia and New Zealand in 1995. II. Identification of a herpesvirus within gill epithelium: Diseases of Aquatic Organisms, v. 28, p. 17–29.
91. Whittington, R.J., Jones, J.B., Hine, P.M., and Hyatt, A.D., 1997, Epizootic mortality in the pilchard (*Sardinops sagax neopilchardus*) in Australia and New Zealand in 1995. I. Pathology and epizootiology: Diseases of Aquatic Organisms, v. 28, p. 1–15.
92. Austin, B., 1999, Emerging bacterial fish pathogens: Bulletin of the European Association of Fish Pathologists, v. 19, p. 231–234.
93. Smith, G.M., and Coates, C.W., 1938, Fibro-epithelial growth of the skin in large marine turtles, *Chelonia mydas* (Linnaeus): Zoologica, v. 23, p. 93–98.
94. Herbst, L.H., 1994, Fibropapillomatosis of marine turtles: Annual Review of Fish Diseases, v. 4, p. 389–425.
95. Williams, E.H., Jr., Bunkley-Williams, L., Peters, E.C., Pinto Rodriguez, B., Matos-Morales, R., Mignucci-Giannoni, A.A., Hall, K.V., Rueda-Almonacid, J.V., Sybesma, J., de Calventi, I.B., and Boulon, R.H., 1994, An epizootic of cutaneous fibropapillomas in green turtles *Chelonia mydas* of the Caribbean: part of a panzootic?: Journal of Aquatic Animal Health, v. 6, p. 70–78.
96. Williams, E.H., Jr., Bunkley-Williams, L., Peters, E.C., Harshbarger, J.C., Pinto-Rodriguez, B., Matos-Morales, R., Mignucci-Giannoni, A.A., Hall, K.J., Sybesma, J., De Calverti, I.B., and Boulon, R.H., 1991, Fibropapillomas in Caribbean greean turtles: part of a widespread disturbance?: Association of Marine Laboratories of the Caribbean, v. 24.
97. Balazs, G.H., and Pooley, S.G., 1991, Research plan for marine turtle fibropapilloma: U.S. Department of Commerce, NOAA, National Marine Fisheries Service, National Oceanic and Atmospheric Administration–TM–NMFS–SWFSC–156.
98. Herbst, L.H., Jacobson, E.R., Moretti, R., Brown, T., Sund-

berg, J.P., and Klein, P.A., 1995, Experimental transmission of green turtle fibropapillomatosis using cell-free tumor extracts: Diseases of Aquatic Organisms, v. 22, p. 1–12.
99. Quackenbush, S.L., Work, T.M., Balazs, G.H., Casey, R.N., Rovnak, J., Chaves, A., duToit, L., Baines, J.D., Parrish, C.R., Bower, P.R., and Casey, J.W., 1998, Three closely related herpesviruses are associated with fibropapillomatosis in marine turtles: Virology, v. 246, p. 392–399.
100. Galzenbrook, J.S., Campbell, R.S.F., and Thomas, A.T., 1993, Studies on an ulcerative stomatitis—obstructive rhinitis—pneumonia disease complex in hatchling and juvenile sea turtles *Chelonia mydas* and *Caretta caretta*: Diseases of Aquatic Organisms, v. 16, p. 133–147.
101. Callan, R.J., Early, G., Kida, H., and Hinshaw, V.S., 1995, The appearance of H3 influenza virus in seals: Journal of General Virology, v. 76, p. 199–203.
102. Dunn, J.L., Buck, J.D., and Robeck, T.R., 2001, Bacterial diseases of cetaceans and pinnipeds, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 309–335.
103. Higgins, R., 2000, Bacteria and fungi of marine mammals–a review: Canadian Veterinary Journal, v. 41, p. 105–116.
104. Kennedy, S., 1998, Morbillivirus infections in aquatic mammals (a review): Journal of Comparative Pathology v. 119, p. 201–225.
105. Kennedy-Stoskopf, S., 2001, Viral diseases, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 285–307.
106. Miller, D.L., Ewing, R.Y., and Bossart, G.D., 2001, Emerging and resurging diseases, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 15–30.
107. Scholin, C.A., Gulland, F., Doucette, G.J., Benson, S., Busman, M., Chavez, F.P., Cordaro, J., DeLong, R., De Vogelaere, A., Harvey, J., Haulena, M., Lefebvre, K., Lipscomb, T., Loscutoff, S., Lowenstine, L.J., Marin, R., Miller, P.E., McLellan, W.A., Moeller, P.D.R., Powell, C.L., Rowles, T., Silvagni, P., Silver, M., Spraker, T., Trainer, V., and Van Dolah, F.M., 2000, Mortality of sea lions along the central California coast linked to a toxic diatom bloom: Nature, v. 403, p. 80–84.
108. Van Bressem, M.F., Van Waerebeek, K., and Raga, J.A., 1999, A review of virus infections of cetaceans and the potential impact of morbilliviruses, poxviruses, and papillomaviruses on host population dynamics: Diseases of Aquatic Organisms, v. 38, p. 53–65.
109. Lipscomb, T.P., Schulman, F.Y., Moffett, D., and Kennedy, S., 1994, Morbilliviral disease in Atlantic bottlenose dolphins (*Tursiops truncatus*) from 1987–1988 epizootic: Journal of Wildlife Diseases, v. 30, p. 567–571.
110. Federal Register, 1993, Taking and importing of marine mammals; depletion of the coastal-migratory stock of bottlenose dolphins along the U.S. Mid-Atlantic coast: Washington, D.C., U.S. Government Printing Office, v. 58, no. 64, p. 17,789–17,791.
111. Schulman, F.Y., Lipscomb, T.P., Moffett, D., Krafft, A.E., Lichy, J.H., Tsai, M.M., Taubenberger, J.K., and Kennedy, S., 1997, Histologic, immunohistochemicall, and polymerase chain reaction studies of bottlenose dolphins from the 1987–1988 United States Atlantic coast epizootic:: Veterinary Pathology, v. 34, p. 288–295.
112. Scott, G.P., Burn, D.M., and Hansen, L.J., 1988, The dolphin die-off: long-term effects and recovery of the population, *in* Proceedings of the Oceans 1988 Conference: Baltimore, Md, p. 819–823.
113. Dietz, R., Heide-Jorgensen, M.P., and Harkonen, T., 1989, Mass deaths of harbor seals (*Phoca vitulina*) in Europe: Ambio, v. 18, p. 258–264.
114. Mamaev, L.V., Visser, I., Belikov, S., Denikina, N.N., Harder, T., Goatley, L., Rima, B., Edginton, B., Osterhaus, A., and Barrett, T., 1996, Canine distemper virus in Lake Baikal seals (*Phoca sibirica*): Veterinary Record, v. 138, p. 437–439.
115. Mahy, B.W.J., Barrett, T., Evans, S., Anderson, E.C., and Bostrock, C.J., 1988, Characterization of a seal morbillivirus: Nature, v. 336, p. 115.
116. Osterhaus, A., Groen, J., Spijkers, H., Broeders, H., Uytde-Haag, F., de Vries, P., Teppema, J.S., Visser, I.K.G., van de Bildt, M.W.G., and Vedder, E.J., 1990, Mass mortality in seals caused by a newly discovered morbillivirus: Veterinary Microbiology, v. 23, p. 343–350.
117. Heide-Jorgensen, M., Harkonen, T., Dietz, R., and Thompson, P., 1992, Retrospective of the 1988 European seal epizootic: Diseases of Aquatic Organisms, v. 13, p. 37–62.
118. Kennedy, S., Smyth, J.A., Cush, P.F., McCullough, S.J., Allan, G.M., and McQuaid, S., 1988, Viral distemper now found in porpoises: Nature, v. 336, p. 21.
119. Osterhaus, A.D.M.E., and Vedder, E.J., 1988, Identification of virus causing recent seal deaths: Nature, v. 335, p. 20.
120. Goodhart, C.B., 1988, Did virus transfer from harp seals to common seals?: Nature, v. 336, p. 21.
121. Aguilar, A., and Raga, J., 1993, The striped dolphin epizootic in the Mediterranean Sea: Ambio, v. 22, p. 524–528.
122. Domingo, M., Ferrer, L, Pumarola, M., Marco, A., Plana, J., Kennedy, S., McAliskey, M., and Rima, B.K., 1990, Morbillivirus in dolphins: Nature, v. 348, p. 21.
123. Domingo, M., Visa, J., Pumarola, M., Marco, A., Ferrer, L., Rabanal, R., and Kennedy, S., 1992, Pathological and immunocytochemical studies of morbillivirus infection in striped dolphins (*Stenella coeruleoalba*): Veterinary Pathology, v. 29, p. 1–10.
124. Duignan, P., Geraci, J., Raga, J., and Calzada, N., 1992, Pathology of morbillivirus infection in striped dolphins (*Stenella coeruleoalba*) from Valencia and Murcia Spain: Canadian Journal of Veterinary Research, v. 56, p. 242–248.
125. Lipscomb, T., Kennedy, S., Moffett, D., Krafft, A., Klaunberg, B., Lichy, J., Regan, G., Worthy, G., and Taubenberger, J., 1996, Morbilliviral epizootic in bottlenose dolphins of the Gulf of Mexico: Journal of Veterinary Diagnostic Investigation, v. 8, p. 283–290.
126. Birkun, A., Kuiken, T., Krivokhizhn, S., Haines, D.M., Osterhaus, A.D.M.E., van de Bildt, M.W.G., Joiris, C.R., and Siebert, U., 1999, Epizootic of morbilliviral disease in common dolphins (*Delphinus delphis ponticus*) from the Black Sea: Veterinary Record, v. 144, p. 85–92.
127. Forsyth, M.A., Kennedy, S., Wilson, S., Eybatov, T., and Barrett, T., 1998, Canine distemper virus in a Caspian seal

(*Phoca caspica*): Veterinary Record, v. 143, p. 662–664.
128. Osterhaus, A., Groen, J., Niesters, H., van de Bildt, M., Martina, B., Vedder, L., Vos, J., van Egmond, H., Abou Sidi, B., and Barham, M.E.O., 1997, Morbillivirus in monk seal mass mortality: Nature, v. 388, p. 838–839.
129. Raloff, J., 1997, Endangered seals suffer massive die-off: Science News, v. 152, p. 134.
130. Kennedy, S., Kuiken, T., Jepson, P.D., Deaville, R., Forsyth, M., Barrett, T., van de Bildt, M.W.G., Osterhaus, A., Eybatov, T., Duck, C., Kydyrmanov, A., Mitrofanov, I., and Wilson, S., 2000, Mass die-off of Caspian seals caused by canine distemper virus: Emerging Infectious Diseases, v. 6, p. 637–639.
131. Stone, R., 2000, Canine virus blamed in Caspian seal death: Science, v. 289, p. 2017–2018.
132. Jensen, T., van de Bildt, M., Dietz, H.H., Andersen, T.H., Hammer, A.S., Kuiken, T., and Osterhaus, A., 2002, Another phocine distemper outbreak in Europe: Science, v. 297, p. 209.
133. Webster, R.G., Hinshaw, V.S., Bean, W.J., van Wyke, K.L., Geraci, J.R., St. Aubin, D.J., and Petursson, G., 1981, Characterization of an influenza A virus from seals: Virology, v. 113, p. 712–724.
134. Rhyan, J.C., 2000, Brucellosis in terrestrial wildlife and marine mammals, *in* Brown, C., and Bolin, C., eds., Emerging diseases of animals: Washington, D.C., ASM Press, p. 161–184.
135. Miller, W.G., Adams, L.G., Ficht, T.A., Chevible, N.F., Payeur, J.P., Harley, D.R., House, C., and Ridgeway, S.H., 1999, *Brucella*-induced abortions and infection in bottlenose dolphins (*Tursiops truncatus*): Journal of Zoo and Wildlife Medicine, v. 30, p. 100–110.
136. Gulland, F.M.D., 2001, Leptospirosis in marine mammals, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 469–471.
137. Harwood, J., and Hall, A., 1990, Mass mortality in marine mammals: its implications for population dynamics and genetics: Trends in Ecology and Evolution, v. 5, p. 254–257.
138. Reidarson, T.H., McBain, J., Dalton, L.M., and Rinaldi, M.G., 2001, Mycotic Diseases, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 337–355.
139. Lapointe, J.M., Gulland, F.M.D., Haines, D.M., Barr, B.C., and Duignan, P.J., 1999, Placentitis due to *Coxiella burnetii* in a Pacific harbor seal (*Phoca vitulina richardsi*): Journal of Veterinary Diagnostic Investigation, v. 11, p. 541–543.
140. Dailey, M.D., 2001, Parasitic diseases, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 357–379.
141. Thomas, N.J., and Cole, R.A., 1996, The risk of disease and threats to the wild population: Conservation and management of the southern sea otter, Endangered Species Update, v. 13, p. 23–27.
142. Hollmén, T., 2002, Biomarkers of health and disease in common eiders (*Somateria mollissima*) in Finland: Helsinki University Biomedical Dissertations, no. 12, Department of Basic Veterinary Sciences, Academic Dissertation, Helsinki, 59 p. and attachments.
143. Friend, M., 1999, Avian cholera, *in* Friend, M., and Franson, J. C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 75–92.
144. Gardner, H., Brouwer, S., Gleeson, L., Kerry, K., and Riddle, M., 1997, Poultry virus infection found in Antarctic penguins: Penguin Conservation, v. 10, p. 8–21.
145. Gardner, H., Kerry, K., Riddle, M., Brouwer, S., and Gleeson, L., 1997, Poultry virus infection in Antarctic penguins: Nature, v. 387, p. 245.
146. Hollmén, T., Franson, J.C., Docherty, D.E., Kilpi, M., Hario, M., Creekmore, L.H., and Petersen, M.R., 2000, Infectious bursal disease virus antibodies in eider ducks and herring gulls: Condor, v. 102, p. 688–691.
147. Lukert, P.D., and Saif, Y.M.,1991, Infectious bursal disease, *in* Calnek, B.W., and others eds., Diseases of poultry (9th ed.): London, Wolfe Publishing Ltd., p. 648–663.
148. Hollmén, T.E., Franson, J.C., Flint, P.L., Grand, J.B., Lanctot, R.B., Docherty, D.E., and Wilson, H.M., 2003, An adenovirus linked to mortality and disease in long-tailed ducks (*Clangula hyemalis*) in Alaska: Avian Diseases, v. 47, p. 1434–1440.
149. Berger, L., Speare, R., Thomas, A., and Hyatt, A., 2001, Mucocutaneous fungal disease in tadpoles of *Bufo marinus* in Australia: Journal of Herpetology, v. 35, p. 330–335.
150. Fellers, G.M., Green, D.E., and Longcore, J.E., 2001, Oral chytridiomycosis in the mountain yellow-legged frog (*Rana muscosa*): Copeia, v. 4, p. 945–953.
151. Green, D.E., and Sherman, C. Kagarise, 2001, Diagnostic histological findings in Yosemite toads (*Bufo canorus*) from a die-off in the 1970s: Journal of Herpetology, v. 35, p. 92–103.
152. Laurance, W.F., McDonald, K.R., and Speare, R., 1996, Epidemic disease and the catastrophic decline of Australian rainforest frogs: Conservation Biology, v. 77, p. 203–212.
153. Lips, K.R., 1999, Mass mortality and population decline of anurans at an upland site in western Panama: Conservation Biology, v. 13, p. 117–125.
154. Meteyer, C.U., 2000, Field guide to malformations of frogs and toads with radiographic interpretations: U.S. Geological Survey, Biological Science Report 2000–0005, 16 p.
155. Corn, P.S., 1994, What we know and don't know about amphibian declines in the West, *in* Covington, W.W., DeBano, L.F., eds., Sustainable ecological systems: implementing an ecological approach to land management: U.S. Forest Service, General Technical Report RM–247, p. 59–67.
156. Longcore, J.E., Pessier, A.P., and Nichols, D.K., 1999, *Batrachochytrium dendrobatidis* gen. et sp. nov., a chytrid pathogenic to amphibians: Mycologia, v. 91, p. 219–227.
157. Green, D.E., Converse, K.A., and Schrader, A.K., 2002, Epizootiology of sixty-four amphibian morbidity and mortality events in the USA, 1996–2001: Annals New York Academy of Sciences, v. 969, p. 323–339.
158. Flynn, R.J., 1973, Parasites of laboratory animals: Ames, Iowa State University Press, 884 p.
159. Meteyer, C.U., Loeffler, I.K., Fallon, J.F., Converse, K.A.,

Green, E., Helgen, J.C., Kersten, S., Levey, R., Eaton-Poole, L., and Burkhart, J.G., 2000, Hind limb malformations in free-living northern leopard frogs (*Rana pipiens*) from Maine, Minnesota, and Vermont suggest multiple etiologies: Teratology, v. 62, p. 151–171.
160. Markiw, M.E., 1992, Salmonid whirling disease: U.S. Fish and Widlife Service, Fish and Wildlife Leaflet 17, 11 p.
161. Hoffman, G.L., 1990, *Myxobolus cerebralis*, a worldwide cause of salmonid whirling disease: Journal of Aquatic Animal Health, v. 2, p. 30–37.
162. Potera, C., 1997, Fishing for answers to whirling disease: Parasitology, v. 278, p. 225–226.
163. ProMED-mail, 2003, Whirling disease detected in Clark's Fork River (Wyoming), Feb. 27, 2003: 20030228.0495, accessed Feb. 28, 2003 at URL http://www.promedmail.org.
164. Wisconsin State Journal, September 16, 2002, Virus cited in huge carp die-off.
165. Wisconsin Department of Natural Resources, 2002: New yellow perch parasite, PUB–FH–726–6/00, accessed April 25, 2003, at URL http://www.dnr.state.wi.us/org/water/fhp/fish/health/disease.htm.
166. Meyer, F.P., Warren, J.W., and Carey, T.G., eds., 1983, A guide to integrated fish health management in the Great Lakes Basin: Great Lakes Fishery Commission, Special Publication no. 83–2., 272 p.
167. Hill, B.J., 1998, Update on fish pathogens listed in the OIE Aquatic Animal Health Code: Proceedings of the International Symposium on Aquatic Animal Health, v. 3, September, Baltimore, Md.
168. Austin, B., and Austin, D.A., 1999, Bacterial fish pathogens: disease of farmed and wild fish (3rd ed.): Chichester, UK, Springer Praxis Publishing, 457 p.
169. Wolf, K., 1988, Fish viruses and fish viral diseases: Ithaca, N.Y., Cornell University Press, p. 476.
170. Lorenzen, E., Olesen, N.J., Korsholm, H., Hever, O.E., and Evensen, A., 1997, First demonstration of *Renibacterium salmoninarum*/BKD in Denmark: Bulletin of European Association of Fish Pathologists, v. 17, p. 140–144.
171. ProMED-mail, 2003, Infectious pancreatic necrosis, trout—UK (Northern Ireland): Feb. 27, 2003: 20030227.0493, accessed Feb. 27, 2003 at URL http://www.promedmail.org.
172. Murray, A.G., Busby, C.D., and Bruno, D.W., 2003, Infectious pancreatic necrosis virus in Scottish Atlantic salmon farms, 1996–2001: Emerging Infectious Diseases, v. 9, p. 455–460.
173. Riedel, R., Caskey, L., and Costa-Pierce, B.A., 2002, Fish biology and fisheries ecology of the Salton Sea, California: Hydrobiologia, v. 473, p. 229–244.
174. Sutton, R.J., 2002, Summer movements of desert pupfish among habitats at the Salton Sea: Hydrobiologia, v. 473, p. 223–228.
175. Kuperman, B.I., Matey, V.E., and Barlow, S.B., 2002, Flagellate *Cryptobia branchialis* (*Bodonida: kinetoplastida*), ectoparasite of tilapia from the Salton Sea: Hydrobiologia, v. 473, p. 93–102.
176. Friend, M., 2002, Preface: Hydrobiologia, v. 473, p. vii–xii.
177. Ramseyer, L.J., 2002, Transgenic fish—a boon or threat: Science, v. 298, p. 1715.
178. Stokstad, E., 2002, Engineered fish: friend or foe of the environment?: Science, v. 297, p. 1797–1799.
179. Weaver, D., 2002, Dealing with the risks of transgenic fish: Science, v. 298, p. 1715.
180. Leibovitz, L., and Hwang, J., 1968, Duck plague on the American continent: Avian Diseases, v. 12, p. 361–378.
181. Leibovitz, L., and Hwang, J., 1968, Duck plague in American Anseriformes: Bulletin of the Wildlife Disease Association, v. 4, p. 13–14.
182. Converse, K.A., and Kidd, G.A., 2001, Duck plague epizootics in the United States, 1967–1995: Journal of Wildlife Diseases, v. 37, p. 347–357.
183. Friend, M., 1999, Duck plague, *in* Friend, M., and Franson, J. C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 141–151.
184. Docherty, D. E., and Friend, M., 1999, Newcastle disease, *in* Friend, M., and Franson, J.C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 175–179.
185. Wobeser, G.A., 1981, Diseases of wild waterfowl: New York, Plenum Press, 300 p.
186. Wobeser, G., and Brand, C.J., 1982, Chlamydiosis in two biologists investigating disease occurrences in wild waterfowl: Wildlife Society Bulletin, v. 10, p. 170–172.
187. Cole, R.A., and Friend, M., 1999, Miscellaneous parasitic diseases, *in* Friend, M., and Franson, J. C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 249–258.
188. Thomas, N.J., Meteyer, C.U., and Sileo, L., 1998, Epizootic vacuolar myelinopathy of the central nervous system of bald eagles (*Haliaeetus leucocephalus*) and American coots (*Fulica americana*): Veterinary Pathology, v. 35, p. 479–487.
189. Fischer, J.R., Lewis, L.A., Augspurger, T., and Rocke, T.E., 2002, Avian vacuolar myelinopathy: a newly recognized fatal neurological disease of eagles, waterfowl and other birds: Transactions North American Wildlife and Natural Resources Conference, v. 67, p. 51–61.
190. Weissenbock, H., Kolodziejek, J., Url, A., Lussy, H., Rebel-Bauder, B., and Nowotny, N., 2002, Emergence of *Usutu* virus, an African mosquito-borne *Flavivirus* of the Japanese encephalitis virus group, Central Europe: Emerging Infectious Diseases, v. 8, p. 652–656.
191. Fischer, J.R., Stallknecht, D.E., Luttrell, M.P., Dhondt, A.A., and Converse, K.A., 1997, Mycoplasmal conjunctivitis in wild songbirds: the spread of a new contagious disease in a mobile host population: Emerging Infectious Diseases, v. 3, p. 69–72.
192. Hartup, B.K., Bickal, J.M., Dhondt, A.A., Ley, D.H., and Kollias, G.V., 2001, Dynamics of conjunctivitis and *Mycoplasma gallisepticum*: The Auk, v. 118, p. 327–333.
193. Friend, M., 1999, Salmonellosis, *in* Friend, M., and Franson, J.C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 99–109.
194. Kirkwood, J.K., and MacGregor, S.K., 1998, Salmonellosis in provisioned free-living greenfinches (*Carduelis chloris*)

and other garden birds, *in* Zwart, P., ed., European Association of Zoo and Wildlife Veterinarians and BVZS 2nd Scientific meeting: Bunnik, The Netherlands.
195. Alley, M.R., Connolly, J.H., Fenwick, S.G., Mackereth, G.F., Leyland, M.J., Rogers, L.E., Haycock, M., Nicol, C., and Reed, C.E.M., 2002, An epidemic of salmonellosis caused by *Salmonella typhimurium* DT 160 in wild birds and humans in New Zealand: New Zealand Veterinary Journal, v. 50, p. 170–176.
196. Thornley, C.N., Simmons, G.C., Callaghan, M.L., Nicol, C.M., Baker, M.G., Gilmore, K.S., and Garrett, N.K.G., 2003, First incursion of *Salmonella enterica* serotype typhimurium DT 160 into New Zealand: Emerging Infectious Diseases, v. 9, p. 493–495.
197. Holden, C., 2000, India's vultures declining: Science, v. 289, p. 1679.
198. Rahmani, A.R., and Prakash, V., eds., 2000, A brief report on the international seminar on vulture situation in India: Bombay Natural History Society.
199. Wilson, M.L., Bretsky, P.M., Cooper, G.H. Jr., Egbertson, S.H., Van Kruiningen, H.J., and Cartter, M.L., 1997, Emergence of raccoon rabies in Connecticut, 1991–1994: spatial and temporal characteristics of animal infection and human contact: American Journal of Tropical Medicine and Hygiene, v. 57, p. 457–463.
200. Appel, M.J.G., Yates, R.A., Foley, G.L., Bernstein, J.J., Santinelli, S., Spelman, L.H., Miller, L.D., Arp, L.H., Anderson, M., Barr, M., Pearce-Kelling, S., and Summers, B.A., 1994, Canine distemper epizootic in lions, tigers, and leopards in North American: Journal of Veterinary Diagnostic Investigation, v. 6, p. 277–288.
201. Adler, T., 1996, A common dog virus diminishes lion pride: Science News, v. 149, p. 70.
202. Roelke-Parker, M.E., Munson, L., Packer, C., Kock, R., Cleaveland, S., Carpenter, M., O'Brien, S.J., Pospischll, A., Hofmann-Lehmann, R., Lutz, H., Mwamengele, G.L.M., Mgasa, M.N., Machange, G.A., Summers, B.A., and Appel, M.J.G., 1996, A canine distemper virus epidemic in Serengeti lions (*Panthera leo*): Nature, v. 379, p. 441–445.
203. Brown, M.B., Schumacher, I.M., Klein, P.A., Harris, K., Correll, T., and Jacobson, E.R., 1994, *Mycoplasma agassizii* causes upper respiratory tract disease in the desert tortoise: Infect. Immun., v. 62, p. 4580–4586.
204. Brown, M.B., McLaughlin, G.S., Klein, P.A., Crenshaw, B.C., Shumacher, I.M., Brown, D.R., and Jacobson, E.R., 1999, Upper respiratory tract disease in the gopher tortoise is caused by *Mycoplasma agassizii*: Journal of Clinical Microbiology, v. 37, p. 2262–2269.
205. Jacobson, E.R., Gaskin, J.M., Brown, M.B., Harris, R.K., Gardiner, C.H., LaPointe, J.L., Adams, H.P., and Reggiardo, C., 1991, Chronic upper respiratory tract disease of free-ranging desert tortoises (*Xerobates agassizii*): Journal of Wildlife Diseases, v. 27, p. 296–316.
206. McLaughlin, G.S., 1997, Upper respiratory tract disease in gopher tortoises, *Gopherus polyphemus*: pathology, immune responses, transmission, and implications for conservation and management: Ph.D dissertation, University of Florida, 110 p.
207. Rose, F.L., Koke, J., Koehn, R. and Smith, D., 2001, Identification of the etiological agent for necrotizing scute disease in the Texas tortoise: Journal of Wildlife Disease, v. 37, p. 223–228.
208. Charles Darwin Foundation, 2000, Mysterious disease in Santa Cruz tortoises studied: accessed April 25, 2003, at URL http://www.darwinfoundation.org/articles/ar00040041.htm.
209. Arno, S.F., 1986, Whitebark pine cone crops: a diminishing source of wildlife food?: Western Journal of Applied Forestry, v. 1, p. 92–94.
210. Kendall, K.C., and Keane, R.E., 2000, Whitebark pine decline: Infection, mortality, and population trends, *in* Tomback, D.F., Arno, S.F., and Keane, R.E., eds., Whitebark Pine Communities Ecology and Restoration: Covelo, Calif., Island Press, p. 221–224.
211. Moffat, A.S., 2001, Finding new ways to fight plant diseases: Science, v. 292, p. 2270–2273.
212. Rapport, D.J., and Whitford, W.G., 1999, How ecosystems respond to stress—common properties of arid and aquatic systems: Bioscience, v. 49, p. 193–203.
213. Keet, D.F., Kriek, N.P.J., Penrith, M.-L., Michel, A., and Huchzermeyer, H., 1996, Tuberculosis in buffaloes (*Syncerus caffer*) in the Kruger National Park: Spread of the disease to other species: Ondestepoort Journal of Veterinary Research, v. 63, p. 239–244.
214. 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. 598–601.
215. Randerson, J., 2002, Watch out for the tourists: New Scientist, v. 174, no. 2346, p. 10.
216. Ferber, D., 2000, Human diseases threaten great apes: Science, v. 289, p. 1277–1278.
217. Tauxe, R.V., 2002, Emerging foodborne pathogens: International Journal of Food Microbiology, v. 78, p. 31–41.
218. Mead, P.S., Slutske, R., L., Dietz, V., McCaIG, L.F., Bresee, J.S., Shapiro, C., Griffin, P.M., and Tauxe, R.V., 1999, Food related illness and death in the United States: Emerging Infectious Diseases, v. 5, p. 607–625.
219. Murphy, F.A., 1999, The threat posed by the global emergence of livestock, food-borne, and zoonotic pathogens: Annals New York Academy of Sciences, v. 894, p. 20–27.
220. Alterkruse, S.F., Cohen, M.L., and Swerdlow, D.L., 1997, Emerging foodborne diseases: Emerging Infectious Diseases, v. 3, p. 285–293.
221. Jaykus, L., 2000, Enteric viruses as 'emerging agents' of foodborne disease: Irish Jornal of Agriculture and Food Research, v. 39, p. 245–255.
222. Phillips, C.A., 2001, Arcobacters as emerging human foodborne pathogens (a review): Food Control, v. 12, p. 1–6.
223. Slifko, T.R., Smith, H.V., and Rose, J.B., 2000, Emerging parasite zoonoses associated with water and food: International Journal for Parasitology, v. 30, p.1379–1393.
224. Chomel, B.B., 1998, New emerging zoonoses: a challenge and an opportunity for the veterinary profession: Comparative Immunology Microbiology and Infectious Diseases, v. 21, p. 1–14.
225. Meslin, F.X., 1992, Surveillance and control of emerging zoonoses: World Health Statistics Quarterly, v. 45, p. 200–207.
226. Ward, L.R., Threlfall, J., Smith, H.R., and O'Brien, S.J.,

2000, *Salmonella enteritidis* epidemic: Science, v. 287, p. 1753–1754.
227. Baumler, A., Hargis, B.M., and Tsolis, R.M., 2000, *Salmonella enteritidis* epidemic response: Science, v. 287, p. 1755–1756.
228. Riemann, H., Kass, P., and Cliver, D., 2000, *Salmonella enteritidis* epidemic: Science, v. 287, p.1754–1755.
229. Hennessy, T.W., Hedberg, C.W., Slutsker, L., White, K.E., Besser-Weik, J.M., Moen, M.E., Feldman, J., Coleman, W.W., Edmonson, L.M., MacDonald, K.L., Osterholm, M.T., and The Investigation Team, 1996, A national outbreak of *Salmonella enteritidis* infections from ice cream: New England Journal of Medicine, v. 334, p. 1281–1286.
230. Ferber, D., 2000, Superbugs on the hoof?: Science, v. 288, p. 792–794.
231. Pilot, K.E., Dalley, E., and Brown, J.W., 1996, Threat from the food we eat: Medical Laboratory Observer, April, p. 42–53.
232. Park, S., Worobo, R.W., and Durst, R.A., 1999, *Escherichia coli* 0157:H7 as an emerging foodborne pathogen (a review): Critical Reviews in Food Science and Nutrition, v. 39, p. 481–502.
233. Feder, I., Wallace, F.M., Gray, J.T., Fratamico, P., Fedorka-Cray, P.J., Pearce, R.A., Call, J.E., Perrine, R., and Luchansky, J.B., 2003, Isolation of *Escherichia coli* 0157:H7 from intact colon fecal samples of swine: Emerging Infectious Diseases, v. 9, p. 380–383.
234. U.S. Fish and Wildlife Service, 1999, 1980–1995 participation in fishing, hunting, and wildlife watching, national and regional demographic trends: U.S., Fish and Wildlife Service, report no. 96–5, 83 p.
235. Keene, W., Sazie, E., Kok, J., Rice, D., Hancock, D., Balan, V., Zhao, T., and Doyle, M.P., 1997, An outbreak of *Escherichia coli* 0157:H7 infections traced to jerky made from deer meat: Journal of the American Medical Association, v. 277, p. 1229–1231.
236. Rabatsky-Ehr, T., Dingman, D., Marcus, R., Howard, R., Kinney, A., and Mshar, P., 2002, Deer meat as the source for a sporadic case of *Escherichia coli* 0157:H7 infection, Connecticut: Emerging Infectious Diseases, v. 8, p. 525–527.
237. Besser, RE., Lett, S.M., Weber, J.T., Doyle, M.P., Barrett, T.J., Wells, J.G., and Griffin, P.M., 1993, An outbreak of diarrhea and hemolytic uremic syndrome from *Escherichia coli* 0157: H7 in fresh-pressed apple cider: Journal of the American Medical Association, v. 269, p. 2217–2220.
238. Shane, S.M., 2000, *Campylobacter* infection of commercial poultry: Revue Scientifique et Technique Du Office International des Epizooties, v. 19, p. 376–395.
239. Skirrow, M.B., 1977, *Campylobacter* enteritis: a "new" disease: British Medical Journal, v. 2, p. 9–11.
240. Gillespie, I.A., O'Brien, S.J., Frost, J.A., Adak, G.K., Horby, P., Swan, A.V., Painter, M.J., Neal, K.R., and the Campylobacter Sentinel Surveillance Scheme Collaborators, 2002, A case-case comparison of *Campylobacter coli* and *Campylobacter jejuni* infection: a tool for generating hypotheses: Emerging Infectious Diseases, v. 8, p. 937–942.
241. Dingle, K.E., Colles, F.M., Ure, R., Wagenaar, J.A., Duim, B., Bolton, F.J., Fox, A.J., Wareing, D.R.A., and Maiden, M.C.J., 2002, Molecular characterization of *Campylobacter jejuni* clones: a basis for epidemiologic investigation: Emerging Infectious Diseases, v. 8, p. 949–955.
242. Luechtefeld, N.A.W., Blaser, M.S., Reller, L.B., and Wang, W.-L.L., 1980, Isolation of *Campylobacter fetus* subsp. *jejuni* from migratory waterfowl: Journal of Clinical Microbiology, v. 12, p. 406–408.
243. Pacha, R.E., Clark, G.W., Williams, E.A., and Carter, A.M., 1988, Migratory birds of central Washington as reservoirs of *Campylobacter jejuni*: Canadian Journal of Microbiology, v. 34, p. 80–82.
244. Yogasundram, K., Shane, S.M., and Harrington, K.S., 1989, Prevalence of *Campylobacter jejuni* in selected domestic and wild birds in Louisiana: Avian Diseases, v. 33, p. 664–667.
245. Hill, G.A., and Grimes, D.J., 1984, Seasonal study of freshwater lake and migratory waterfowl for *Campylobacter jejuni*: Canadian Journal of Microbiology, v. 30, p. 845–849.
246. Millar, B.C., Finn, M., Xiao, L., Lowery, J.C., Dooley, J.S.G., and Moore, J.E., 2002, *Cryptosporidium* in foodstuffs—an emerging aetiological route of human foodborne illness: Trends in Food Science & Technology, v. 13, p. 168–187.
247. Mackenzie, W.R., Hoxie, N.J., Proctor, M.E., Gradus, M.S., Blair, K.A., Peterson, D.E., Kazmierczak, J.J., Addiss, D.G., Fox, K.R., Rose, J.B., and Davis, J.P., 1994, A massive outreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply: New England Journal of Medicine, v. 331, p. 161–167.
248. Naumova, E.N., Egorov, A.I., Morris, R.D., and Griffiths, J.K., 2003, The elderly and waterborne *Cryptosporidium* infection: gastroenteritis hospitalizations before and during the 1993 Milwaukee outbreak: Emerging Infectious Diseases, v. 9, no. 4, p. 418–425.
249. Corso, P.S., Kramer, M.H., Blair, K.A., Addiss, D.G., Davis, J.P., and Haddix, A.C., 2003, Cost of illness in the 1993 waterborne *Cryptosporidium* outbreak, Milwaukee, Wisconsin: Emerging Infectious Diseases, v. 9, p. 426–431.
250. McCarthy, J., and Moore, T.A., 2000, Emerging helminth zoonoses: International Journal for Parasitology, v. 30, p. 1351–1360.
251. Garrett, S.E., Jahncke, M.L., and Tennyson, J.M., 1997, Microbiological hazards and emerging food-safety issues associated with seafoods: Journal of Food Protection, v. 60, p. 1409–1415.
252. Eastaugh, J., and Shepherd, S., 1989, Infectious and toxic syndromes from fish and shellfish consumption: a review: Archives of Internal Medicine, v. 149, p. 1735–1740.
253. Lopman, B.A., Adak, G.K., Reacher, M.H., and Brown, D.W.G., 2003, Two epidemiologic patterns of *Norovirus* outbreaks: surveillance in England and Wales, 1992–2000: Emerging Infectious Diseases, v. 9, p. 71–77.
254. Lopman, B.A., Reacher, M.H., van Duijnhoven, Y., Hanon, F.-X., Brown, D., and Koopmans, M., 2003, Viral gastroenteritis outbreaks in Europe, 1995–2000: Emerging Infectious Diseases, v. 9, p. 90–96.
255. Centers for Disease Control and Prevention, 2002, Outbreak of gastroenteririts associated with Noroviruses on cruise ships—United States, 2002: Morbidity and Mortality

Weekly Report, v. 51, p. 1112–1115.
256. Jaykus, L., 1997, Epidemiology and detection as options for control of viral and parasitic foodboorne disease: Emerging Infectious Diseases, v. 3, p. 529–539.
257. Lipp, E.K., Rodriguez-Palacios, C., and Rose, J.B., 2001, Occurrence and distribution of the human pathogen *Vibrio vulnificus* in a subtropical Gulf of Mexico estuary: Hydrobiologia, v. 460, p. 165–173.
258. Wittman, R.J., and Flick, G.J., 1995, Microbial contamination of shellfish: prevalence, risk to human health, and control strategies: Annual Review of Public Health, v. 16, p. 123–140.
259. Todd, E.C.D., 1993, Seafood-associated diseases in Canada: Journal of Association of Food and Drug Officials, v. 56, p. 45–52.
260. Döller, P.C., Dietrich, K., Filipp, N., Brockmann, S., Dreweck, C., Vonthein, R., Wagner-Wiening, C., and Wiedenmann, A., 2002, Cyclosporiasis outbreak in Germany associated with the consumption of salad: Emerging Infectious Diseases, v. 8, p. 992–994.
261. Tauxe, R.V., Kruse, H., Hedberg, C., Potter, M., Madden, J., and Wachsmuth, K., 1997, Microbial hazards and emerging issues associated with produce—a preliminary report to the National Advisory Committee on microbiologic criteria for foods: Journal of Food Protection, v. 60, p. 1400–1408.
262. Souness, R., and Desmarchelier, T., 1997, Emerging foodborne pathogens, 24–26 March 1997, Alexandria, Virginia, USA: Food Australia, v. 49, p. 258–260.
263. Bahia-Oliveira, L.M.G., Jones, J.L., Azevedo-Silva, J., Alves, C.C.F., Orefice, F., and Addiss, D.G., 2003, Highly endemic, waterborne toxoplasmosis in North Rio de Janeiro State, Brazil: Emerging Infectious Diseases, v. 9, p. 55–62.
264. Murrell, K.D., 2001, Trichinellosis: now and forevermore?: Parasite, v. 8, p. S11–S13.
265. Honey, M., and Rome, A., 2000, Ecotourism and sustainable tourism certification: Part 1: Where are we today?; Part 2: Case Studies, Draft report prepared for the Ecotourism and Sustainable Tourism Certification Workshop, New Paltz, N.Y., Institute for Policy Studies and Ford Foundation.
266. The International Ecotourism Society, 2000, Ecotourism statistical fact sheet, accessed April 28, 2003, at URL http://www.ecotourism.org/textfiles/statsfaq.pdf.
267. Centers for Disease Control and Prevention, 1996, Outbreak of trichinellosis associated with eating cougar jerky—Idaho, 1995: Morbidity and Mortality Weekly Report, v. 45, p. 205–206.
268. Dupouy-Camet, J., 1999, Is human trichinellosis an emerging zoonosis in the European community?: Helminthologia, v. 36, p. 201–204.
269. Murrell, K.D., and Pozio, E., 2000, Trichinellosis: the zoonosis that won't go quietly: International Journal for Parasitology, v. 30, p. 1339–1349.
270. Riordon, A., and Tarlow, M., 1996, Pets and diseases: British Journal of Hospital Medicine, v. 56, p. 321–324.
271. American Pet Products Manufacturers Association, 2002, 2001/2002 National Pet Owner's Survey, press release, May 1, 2001, accessed Feb. 5, 2003, at URL http://www.appma.org/press/news_releases/2001/nr_05-05-01-01.asp.
272. Gehrke, B.C., 1997, Results of the 1997 AMVA survey of U.S. pet-owning households regarding use of veterinary services and expenditures: Journal of Veterinary Medical Association, v. 211, p. 417–418.
273. Chomel, B.B., 1992, Zoonoses of house pets other than dogs, cats, and birds: Pediatrics Infectious Diseases, v. 11, p. 479–487.
274. Robertson, I.D., Irwin, P.J., Lymbery, A.J., and Thompson, R.C.A., 2000, The role of companion animals in the emergence of parasitic zoonoses: International Journal of Parasitology, v. 30, p. 1369–1377.
275. Weinstein, J.W., Seltzer, E.G., Nelson, R.S., Hadler, J.L., Paul, S.M., Sorhage, F.E., Pilot, K., Matluck, S., Spitalny, K., Gupta, M., Misage, J., Balzano, G., Root, T., Birkhead, G., Morse, D.L., Kopelman, A., Engelke, S., Jones, L., Latour, L., Perry, P., Jenkins, B., Maillard, J.M., MacCormack, J.N., Richards, C., Fruth, P., Hufford, S., Dick, B., Bundesen, M., Salehi, E.P., Halpin, T.J., Lurie, P., Deasy, M., Mihelcic, K., and Rankin, J.T., 1995, Reptile-associated salmonellosis-selected states: Morbidity and Mortality Weekly Report, v. 44, p. 347–350.
276. Schantz, P.M., 1994, Of worms, dogs, and human hosts: continuing challenges for veterinarians in prevention of human disease: Journal of American Veterinary Medical Association, v. 204, p. 1023–1028.
277. Plaut, M., Zimmerman, E.M., and Goldstein, R.A., 1996, Health hazards to humans associated with domestic pets: Annual Review of Public Health, v. 17, p. 221–245.
278. Tan, J.S., 1997, Human zoonotic infections transmitted by dogs and cats: Archives of Internal Medicine, v. 157, p. 1933–1943.
279. Hill, S.L., Cheney, J.M., Taton-Allen, G.F., Reif, J.S., Bruns, C., and Lappin, M.R., 2002, Prevalence of enteric zoonotic organisms in cats: Journal of the American Veterinary Medical Association, v. 216, p. 687–692.
280. Macpherson, C.N.L., Meslin, F.X., and Wandeler, A.I., eds., 2000, Dogs, Zoonoses and Public Health: Oxon, UK, CBI publishing, p. xi–xii.
281. Clapp, D.W., Kleiman, M.B., Reynolds, J.K., and Allen, S.D., 1986, Pasteurella multocida meningitis in infancy, an avoidable infection: American Journal of Diseases of Children, v. 140, p. 444–446.
282. Bugg, R.J., Robertson, I.D., Elliot, A.D., and Thompson, R.C.A., 1999, Gastrointestinal parasites of urban dogs in Perth, Western Australia: The Veterinary Journal, v. 157, p. 295–301.
283. Morris, J.G., and Potter, M., 1997, Emergence of new pathogens as a function of changes in host susceptibility: Emerging Infectious Diseases, v. 3, p. 435–441.
284. U.S. Department of Commerce, 1990, 1990 Census of population: Washington, D.C., U.S. Government Printing Office, CP–1–1 20240.
285. Plant, J.A., Baldock, J.W., and Smith, B., 1996, The role of geochemistry in environmental and epidemiological studies in developing countries: a review, *in* Appleton, J.D., Fuge, R., and McCall, G.J.H., eds., Environmental Geochemistry and Health: Geological Society Special Publication, v. 113, p. 7–22.
286. Siegel, J.M., Angulo, F.J., Detels, R., Wesch, J., and Mullen, A., 1999, AIDS diagnosis and depression in the multicenter AIDS cohort study: the ameliorating impact of

pet ownership: AIDS Care, v. 11, p. 157–170.
287. Juckett, G., 1997, Pets and parasites: American Family Physician, v. 56, p. 1763–1774, 1777–1778.
288. Morse, S.S., 1991, Emerging viruses—defining the rules for viral traffic: Perspectives in Biology and Medicine, v. 34, p. 387–409.
289. Kohn, G.C., ed., 1995, Encyclopedia of plague and pestilence: New York, Facts On File, Inc., 408 p.
290. Verano, J.W., and Ubelaker, D.H., 1992, Disease and demography in the Americas: Washington, D.C., Smithsonian Institution Press, 294 p.
291. Zinsser, H., 1934, Rats, lice, and history: New York, Blue Ribbon Books, 301 p.
292. Friend, M., 1962, Tumors, streptothricosis and deer: New York State Conservationist, Oct.–Nov., p. 32.
293. Friend, M., Knoll, E.T., and Gruff, H., 1963, Tuberculosis in a wild white-tailed deer: New York Fish and Game Journal, v. 10, p. 118–123.
294. Muraschi, T.F., Friend, M., and Bolles, D., 1965, Erwinia-like microorganisms isolated from animal and human hosts: Applied Microbiology, v. 13, p. 128–131.
295. Brown, C., 1997, Emerging diseases—what veterinarians need to know: Journal of Veterinary Diagnostic Investigation, v. 9, p. 113–117.
296. Dobson, A., and Foufopoulos, J., 2001, Emerging infectious pathogens of wildlife: Philosophical Transactions of the Royal Society of London, Series B, v. 356, p. 1001–1012.
297. Fayer, R., 2000, Global change and emerging infectious diseases: Journal of Parasitology, v. 86, p. 1174–1181.
298. Levin, B.R., 1996, The evolution and maintenance of virulence in microparasites: Emerging Infectious Diseases, v. 2, p. 93–102.
299. National Institutes of Health, 1992, Report of the task force on microbiology and infectious diseases: U.S. Department of Health and Human Services, NIH Publication no. 92–3320, 57 p.
300. Osburn, B.I., 1996, Emerging diseases with a worldwide impact and the consequences for veterinary curricula: Veterinary Quarterly, v. 18, Suppl. 3, p. S124–S126.
301. Wilson, M.E., 1995, Travel and the emergence of infectious diseases: Emerging Infectious Disease, v. 1, p. 39–46.
302. Warner, R.E., 1968, The role of introduced diseases in the extinction of the endemic Hawaiian avifauna: Condor, v. 70, p. 101–120.
303. Van Riper, C. III, Van Riper, S.G., Golf, M.L., and Laird, M., 1986, The epizootiology and ecological significance of malaria in Hawaiian land birds: Ecological Monographs, v. 56, p. 327–344.
304. Atkinson, Carter, U.S. Geological Survey, personal communication.
305. Atkinson, C.T., Woods, K.L., Dusek, R.J., Sileo, L.S., and Iko, W.M., 1995, Wildlife disease and conservation in Hawaii: pathogenicity of avian malaria (*Plasmodium relictum*) in experimentally infected iiwi (*Vestiaria coccinea*): Parasitology, v. 111, p. S59–S69.
306. Ambroise-Thomas, P., 2000, Emerging parasite zoonoses: the role of host-parasite relationship: International Journal for Parasitology, v. 30, p. 1361–1367.
307. Osterhaus, A., 2001, Catastrophes after crossing species barriers: Philosophical Transactions of the Royal Society of London, Series B, v. 356, p. 791–793.
308. Dahl, T.E., Johnson, C.E., and Frayer, W.E., 1991, Wetlands status and trends in the conterminous United States mid-1970's to mid-1980's: U.S. Fish and Wildlife Service, 28 p.
309. U.S. Fish and Wildlife Service, File Data, 2000, Migratory bird management office, Portland, Ore.
310. Hussong, D., Damare, J.M., Limpert, R.J., Sladen, W.J., Weiner, R.M., and Colwell, R.R., 1979, Microbial impact of Canada geese (*Branta canadensis*) and whistling swans (*Cygnus columbianus*) on aquatic ecosystems: Applied Environmental Microbiology, v. 37, p. 14–20.
311. U.S. Fish and Wildlife Service, June 1986, Final Supplement Environmental Impact Statement: Use of lead shot for hunting migratory birds in the United States: U.S. Fish and Wildlife Service, p. S1–O52.
312. Friend, M., and Trainer, D.O., 1970, Some effects of sublethal levels of insecticides on vertebrates: Journal of Wildlife Diseases, v. 6, p. 335–342.
313. Friend, M., and Trainer, D.O., 1972, Duck hepatitis virus interactions with DDT and dieldrin in adult mallards: Bulletin of Environmental Contamination and Toxicology, v. 7, p. 202–206.
314. Friend, M., and Trainer, D.O., 1974, Experimental DDT-duck hepatitis virus interaction studies: Journal of Wildlife Management, v. 38, p. 887–895.
315. Friend, M., and Trainer, D.O., 1974, Experimental dieldrin-duck hepatitis virus interaction studies: Journal of Wildlife Management, v. 38, p. 896–902.
316. Whiteley, P.L., and Yuill, T.M., 1991, Interactions of environmental contaminants and infectious diseases: Acta Congressus Internationalis Ornithologici 20, v. 4, p. 2338–2342.
317. Friend, M., 1999, Mycoplasmosis, *in* Friend, M., and Franson, J.C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 115–119.
318. Naylor, R.L, Williams, S. and Strong, S.R., 2001, Aquaculture—a gateway for exotic species: Science, v. 294, p. 1655–1656.
319. Galvin, G.B., Bose, R., and Pinsky, C., 1990, Infections and toxic syndromes from fish and shellfish consumption: Archives of Internal Medicine, v. 150, p. 2425.
320. Todd, E.C.D., 1993, Domoic acid and amnesic shellfish poisoning—a review: Journal Food Protection, v. 56, p. 69–83.
321. Chen, Z.-X., Zheng, J.C., and Jiang, Y.L., 1999, A new iridovirus isolated from soft-shelled turtle: Virus Research, v. 63, p. 147–151.
322. Robinson, R.M., Ray, A.C., Reagor, J.C., and Holland, L.A., 1982, Waterfowl mortality caused by aflatoxicosis in Texas: Journal of Wildlife Diseases, v. 18, p. 311–313.
323. Jensen, W.I., and Allen, J.P., l981, Naturally occurring and experimentally induced castor bean (*Ricinus communis*) poisoning in ducks: Avian Diseases, v. 25, p. 184–194.
324. Wobeser, G.A., 1997, Diseases of wild waterfowl (2nd ed.): New York, Plenum Press, 324 p.
325. Windingstad, R.M., Cole, R.J., Nelson, P.E., Roffe, T.J.,

George, R.R., and Dorner, J.W., 1989, *Fusarium* mycotoxins from peanuts suspected as a cause of sandhill crane mortality: Journal of Wildlife Diseases, v. 25, p. 39–46.
326. Forrester, D.J., Gaskin, J.M., White, F.H., Thompson, N.P., Quick, J.A., Henderson, G.E., Woodard, J.C., and Robertson, W.D., 1977, An epizootic of waterfowl associated with a red tide episode in Florida: Journal of Wildlife Diseases, v. 13, p. 160–167.
327. Schreiber, R.W., Dunstan, F.M., and Dinsmore, J.J., 1975, Lesser scaup mortality in Tampa Bay, Florida, 1974: Florida Field Naturalist, v. 3, p. 13–15.
328. Steidinger, K.A., and Haddad, K., 1981, Biologic and hydrologic aspects of red tides: Bioscience, v. 31, p. 814–819.
329. Rocke, T.E., Euliss, N.H., and Samuel, M.D., 1999, Environmental characteristics associated with the occurrence of avian botulism in wetlands of a northern California refuge: Journal of Wildlife Management, v. 63, p. 358–368.
330. Rocke, T.E., and Samuel, M.D., 1999, Water and sediment characteristics associated with avian botulism outbreaks in wetlands: Journal of Wildlife Management, v. 63, p. 1249–1260.
331. Centers for Disease Control and Prevention, 1995, Diphtheria epidemic—new independent states of the former Soviet Union, 1990–1994: Morbidity and Mortality Weekly Report, v. 44, p. 177–180.
332. Maurice, J., 1995, Russian chaos breeds diphtheria outbreak: Science, v. 267, p. 1416–1417.
333. Reintjes, R., Dedushaj, I., Gjini, A., Jorgensen, T.R., Cotter, B., Lieftucht, A., D´Ancona, F., Dennis, D.T., Kosoy, M.A., Mulligi-Osmani, G., Grunow, R., Kalaveshi, A., Gashi, L., and Humolii, I., 2002, Tularemia outbreak investigation in Kosovo: case control and environmental studies: Emerging Infectious Diseases, v. 8, p. 69–73.
334. Wobeser, G., Leighton, F.A., Norman, R., Meyers, D.J., Onderka, D., Pybus, M.J., Neufeld, J.L., Fox, G.A., and Alexander, D.J., 1993, Newcastle disease in wild water birds in western Canada, 1990: Canada Veterinary Journal, v. 34, p. 353–359.
335. Lipp, E.K., Huq, A., and Colwell, R.R., 2002, Effects of global climate on infectious disease: the cholera model: Clinical Microbiology Reviews, v. 15, p. 757–770.
336. Guerrant, R.L., 1997, Cryptosporidiosis: an emerging, highly infectious threat: Emerging Infectious Diseases, v. 3, p. 51–57.
337. Fleming, G., 1871, Animal plagues: their history, nature, and prevention: London, Chapman and Hall, 539 p.
338. Enserink, M., 2003, Scientists chase fast-moving and deadly global disease: Science, v. 299, p. 1822.
339. Eisner, T., and Ehrlich, P.R., 2001, New world pathogen strategy disclosed: Science, v. 292, p. 2397.
340. The White House, May 1, 1996, Memorandum to members of the National Science and Technology Council, clearance request of the Presidential Decision Directive on emerging infectious diseases: Washington, D.C., 3 p.
341. Centers for Disease Control and Prevention, 1998, Emerging infectious diseases—a strategy for the 21st century, accessed May 1, 2003, at URL http://www.cdc.gov/ncidod/emerplan.
342. Centers for Disease Control and Prevention, 2002, Protecting the Nation's health in an era of globalization: CDC's global infectious disease strategy, accessed May 1, 2003, at URL http://www.cdc.gov/globalidplan.
343. Garrett, L., 1994, The coming plague: newly emerging diseases in a world out of balance: New York, Farrar, Straus and Giroux, 750 p.
344. Burnet, F.M., 1962, Natural history of infectious disease (3rd ed.): London, Cambridge University Press, 377 p.
345. Stewart, W.H., 1967, "A mandate for state action" presented at the Association of State and Territorial Health Officers, Washington, D.C., December 4, 1967.
346. Cairns, J., 1978, Cancer: Science and Society: San Francisco, Calif., W.H. Freeman and Co., 199 p.
347. Nelson, A.M., Sledzik, P.S., and Mullick, F.G., 1996, The Army Medical Museum/Armed Forces Institute of Pathology and Emerging Infections: from camp fevers and diarrhea during the American Civil War in the 1860s to global molecular epdemiology and pathology in the 1990s: Archives of Pathology and Laboratory Medicine, v. 120, p. 129–133.
348. National Wildlife Health Center, 2003, Plan for assisting states, federal agencies, and tribes in managing chronic wasting disease in wild and captive cervids, June 26, 2002, accessed May 1, 2003, at URL http://www.nwhc.usgs.gov/research/chronic_wasting/cwd62602.pdf
349. Friel, J.P., ed., 1985, Dorland's illustrated medical dictionary (26th ed.): Philadelphia, Pa., W.B. Saunders Co., 1485 p.
350. Mish, F.G., ed., 1985, Webster's ninth collegiate dictionary: Springfield, Mass., Merriam-Webster Inc., 563 p.
351. Wisconsin Department of Natural Resources, 2002, Wisconsin regulations related to chronic wasting disease: PUB–WM–401–2002, accessed December 23, 2002, at URL http://www.dnr.state.wi.us/org/land/wildlife/regs/02cwdregs.pdf.
352. Bishop, R.C., 2002, The economic effects in 2002 of chronic wasting disease (CWD) in Wisconsin: Department of Agriculture and Applied Economics, University of Wisconsin-Madison, staff paper no. 450, 6 p., accessed December 23, 2002, at URL http://www.aae.wisc.edu/www/pub/sps/stpap450.pdf.
353. Berquist, L., and Riepenhoff, B., 2002, 88 percent plan to hunt this fall, survey says: Journal Sentinel Inc., JSOnline, accessed December 23, 2002, at URL http://www.jsonline.com/news/state/nov02/96328.asp.
354. Colorado Division of Wildlife, 2002, Chronic wasting disease: accessed December 23, 2002, at URL http://wildlife.state.co.us/cwd.
355. Colorado Division of Wildlife, 2002, Colorado Division of Wildlife Strategic Plan, January 11, 2002, accessed June 10, 2003, at URL http://wildlife.state.co.us/about/strategicplan/Final_Adoption.pdf.
356. U.S. Department of Agriculture, 2002, Wisconsin chronic wasting disease program environmental assessment: Animal and Plant Health Inspection Service, accessed December 23, 2002, at URL http://www.aphis.usda.gov/ppd/es/vs/wicwdea.pdf.
357. Bartelt, G., Pardee, J., and Thiede, K., 2003, Environmental impact statement on rules to eradicate chronic wasting disease in Wisconsin's free-ranging white-tailed deer herd: Wisconsin Department of Natural Resources, 175 p., (available at URL http://www.dnr.state.wi.us/us/org/land/wild-

life/whealth/issues/CWD/InsideFrnt.pdf)
358. Centers for Disease Control and Prevention, 1999, Epidemic/epizootic West Nile Virus in the United States: guidelines for surveillance, prevention, and control: accessed December 23, 2002, at URL http://www.cdc.gov/ncidod/dvbid/arbor/WN_surv_guide_Mar_2000.pdf.
359. New York State Department of Health, 2001, State health commissioner announces West Nile Virus prevention education campaign for 2001, accessed December 23, 2002, at URL http://www.health.state.ny.us/nysdoh/commish/2001/wnvrel.htm.
360. Eisner, R., 2000, Armed and ready, researchers around nation working to stop West Nile spread: ABC News Internet Ventures, accessed December 23, 2002, at URL http://abcnews.go.com/sections/living/DailyNews/westnile000503.html.
361. Centers for Disease Control and Prevention, 2002, Responding to West Nile Virus: public health implications and federal response, testimony of James M. Hughes, Director, CDC's National Center for Infectious Diseases, accessed December 23, 2002, at URL http://www.cdc.gov/washington/testimony/id100302.htm.
362. Bogel, K., and Abdussalam, M., 1976, International movement of wild animals in relation to the dissemination of zoonoses *in* Page., L.A., ed., Wildlife Diseases: New York, Plenum Press, p. 107–113.
363. Hoogstraal, H., 1961, Migrating birds and their ectoparasites in relation to disease: East African Medical Journal, v. 38, p. 221–226.
364. Hoogstraal, H., Makram, N., Kaiser, N., Traylor, M.A., Guindy, E., and Gaber, S., 1963, Ticks (Ixodidae) on birds migrating from Europe and Asia to Africa, 1959–61: Bulletin World Health Organization, v. 28, p. 235–262.
365. Janout, V., Urizl, M., Chmela, J., Tumova, B., Stumpa, A., and Smekal, M., 1979, A study on the role of birds in the spread of infections: Journal of Hygiene, Epidemiology, Microbiology and Immunology, v. 23, p. 457–461.
366. Schaaf, K., 1974, Free-flying birds as spreaders of disease: Poultry Digest, January, p. 15–18.
367. Steiniger, F., 1971, Transport of micro-organisms by migratory birds between Europe and South Africa, in relation to bird-ringing and disinfection: The Ostrich, Suppl. 8, p. 283–297.
368. Colwell, R., and Huq, A., 2001, Marine ecosystems and cholera: Hydrobiologia, v. 460, p. 141–145.
369. Lee, K., 2001, The global dimensions of cholera: Global Change and Human Health, v. 2, p. 6–16.
370. Glass, R.I., Libel, M., and Brandling-Bennett, A.D., 1992, Epidemic cholera in the Americas: Science, v. 256, p. 1524–1525.
371. Centers for Disease Control and Prevention, 1992, Update: cholera—Western Hemisphere, 1992: Morbidity and Mortality Weekly Report, v. 41, p. 667–668.
372. Centers for Disease Control and Prevention, 1995, Update: *Vibrio cholerae* 01—Western Hemisphere, 1991–1994, and *V. cholerae* 0139—Asia, 1994: Morbidity and Mortality Weekly Report, v. 44, p. 215–219.
373. Jiang, S.C., 2001, *Vibrio cholerae* in recreational beach waters and tributaries of southern California: Hydrobiologia, v. 460, p. 157–164.
374. Santavy, D.L., and Peters, E.C., 1997, Microbial pests: coral disease in the western Atlantic: Proceedings of the International Coral Reef Symposium, v. 8, p. 607–612.
375. Bruckner, A.W., and Bruckner, R.J., 1997, The persistence of black-band disease in Jamaica: impact on community structure: Proceedings of the International Coral Reef Symposium, v. 8, p. 601–606.
376. Kuta, K.G., and Richardson, L.L., 1997, Black band disease and the fate of diseased coral colonies in the Florida keys: Proceedings of the International Coral Reef Symposium, v. 8, p. 575–578.
377. Cervino, J., and Smith, G., 1997, Corals in peril: Ocean Realm, summer issue, p. 33–35.
378. Grosholz, E.D., and Ruiz, G.M., 1997, Evidence for regional adaptation of black band disease at Carrie Bow Cay, Belize: Proceedings of the International Coral Reef Symposium, v. 8, p. 579–582.
379. Carlton, R.G., and Richardson, L.L., 1995, Oxygen and sulfide dynamics in a horizontally migrating cyanobacterial mat: black band disease of coral: FEMS Microbiology Ecology, v. 18, p. 155–162.
380. Richardson, L.L., Kuta, K.G., Schnell, S., and Carlton, R.G., 1997, Ecology of the black band disease microbial consortium: Proceedings of the International Coral Reef Symposium, v. 8, p. 597–600.
381. Richardson, L.L., 1996, Horizontal and vertical migration patterns of *Phormidium corallyticum* and Beggiatoa spp. associated with black-band disease of corals: Microbiology Ecology, v. 32, p. 323–335.
382. Richardson, L.L., 1993, Red band disease; a new cyanobacterial infestation of corals: Proceedings of the American Academy of Underwater Sciences Annual Scientific Diving Symposium, v. 10, p. 153–160.
383. Goreau, T.J., Cervino, J.M., Goreau, M., Hayes, R.L., Hayes, M., Richardson, L., Smith, G., DeMeyer, K., Nagelkerken, I., Garzon-Ferrera, J., Gil, D., Garrison, G., Williams, E.H., Bunkley-Williams, L., Quirolo, C., Patterson, K., Porter, J.W., and Porter, K., 1998, Rapid spread of diseases in Caribbean coral reefs: Revista de Biología Tropical, v. 46, p. 157–171.
384. Ritchie, K.B., and Smith, W.G., 1998, Description of type II white band disease in acroporid corals: Revista de Biología Tropical, v. 46, p. 199–203.
385. Cervino, J., Goreau, T.J., Nagelkerken, I., Smith, G.W., and Hayes, R., 2001, Yellow band and dark spot syndromes in Caribbean corals: distribution, rate of spread, cytology, and effects on abundance and division rate of zooxanthellae: Hydrobiologia, v. 460, p. 53–63.
386. Hayes, R.L., and Bush, P., 1990, Microscopic observations of recovery in the reef building scleractinian coral, *Montastrea annularis*, after bleaching on a Cayman reef: Coral Reefs, v. 5, p. 201–204.
387. Garzón-Ferreira, J., Gil-Agudelo, D.L., Barrios, L.M., and Zea, S., 2001, Stony coral diseases observed in southwestern Caribbean reefs: Hydrobiologia, v. 460, p. 65–69.
388. McCarty, H.B., and Peters, E.C., 2003, The coral disease page, accessed May 1, 2003, at URL http://ourworld.compuserve.com/hompages/mccarty_and_peters/coraldis.htm.
389. Cervino, J., Goreau, T.J., Hayes, R., Smith, G.W., Santavy, D., Peters, E.C., de Meyer, K., Nagelkerken, I., and

Boekhoudt, B., 1997, A new Caribbean coral disease: Proceedings of the Association of Marine Laboratories of the Caribbean, v. 28, p. 43.
390. Richardson, L.L., Goldberg, W.M., Carlton, R.G., and Halas, J.C., 1998, Coral disease outbreak in the Florida Keys: plaque type II: Revista de Biología Tropical, v. 46 (suppl. 5), p. 187–198.
391. Littler, M.M., and Littler, D.S., 1997, Disease induced mass mortality of crustase coralline algae on coral reefs provides rationale for the conservation of herbivorous fish stocks: Proceedings 8th International Coral Reef Symposium, v. 1, p. 719–724.
392. Nagelkerken, I., Buchan, K., Smith, G.W., Bonair, K., Bush, P., Garzon Ferreira, J., Botero, L., Gayle, P., Petrovic, C., Heberer, C., Pors, L., and Yoshioka, P., 1997, Widespread disease in Caribbean sea fans: I. spreading and general characteristics: Proceedings of the International Coral Reef Symposium, v. 8, p. 679–682.
393. Alker, A.P., Smith, G.W., and Kim, K., 2001, Characterization of *Aspergillus sydowii*, a fungal pathogen of Caribbean sea fan corals: Hydrobiologia, v. 460, p. 113–130.
394. Smith, G.W., Ives, L.D., Nagelkerken, I.A., and Ritchie, K.B., 1996, Caribbean sea-fan mortalities: Nature, v. 383, p. 487.
395. Smith, G.W., Harvel, C.D., and Kim, K., 1998, Response of sea fans to infection with *Aspergillus* sp. (fungi): Revista de Biología Tropical, v. 46, p. 205–208.
396. Palm Beach Post, May 22, 1996, State biologists baffled by holes in sponges on reef off Palm Beach: The Palm Beach Post, press release, accessed January 29, 2003, at URL http://www.newslibrary.com/nlsite/region_pgs/fl_search.htm.
397. Carpenter, R.C., 1990, Mass mortality of *Diadema antillarum* I. Long-term effects on sea urchin population-dynamics and coral reef algal communities: Marine Biology, v. 104, p. 67–77.
398. Lessios, H.A., Robinson, D.R., and Cubit, J.D., 1984, Spread of *Diadema* mass mortality through the Caribbean: Science, v. 226, p. 335–337.
399. Acosta, A., 2001, Disease in zoanthids—dynamics in space and time: Hydrobiologia, v. 460, p. 113–130.
400. Armstrong, I.H., Coulson, J.C., Hawkey, P., and Hudson, M.J., 1978, Further mass seabird deaths from paralytic shellfish poisoning: British Birds, v. 71, p. 58–68.
401. Burkholder, J.M., Noga, E.J., Hobbs, C.H., Glasgow, H.B., Jr., and Smith, S.A., 1992, New 'phantom' dinoflagellate is the causative agent of major estuarine fish kills: Nature, v. 358, p. 407–410.
402. Steidinger, K.A., and Haddad, K., 1981, Biologic and hydrographic aspects of red tides: BioScience, v. 31, p. 814–819.
403. Samet, J., Bignami, G.S., Feldman, R., Hawkins, W., Neff, J., and Smayda, T., 2001, *Pfiesteria*: review of the science and identification of research gaps, report for the National Center for Environmental Health, Centers for Disease Control and Prevention: Environmental Health Perspectives, v. 109 (suppl. 5), p. 639–659.
404. Steidinger, K.A., Burkholder, J.M., Glasgow, H.B., Hobbs, C.W., Garrett, J.K., Truby, E.W., Noga, E.J., and Smith, S.A., 1996, *Pfiesteria piscicida* gen. et sp. nov. (Pfiesteriaceae fam. nov.), a new toxic dinoflagellate with a complex life cycle and behavior: Journal of Phycology, v. 32, p. 157–164.
405. Glasgow, H.B., Burkholder, J.M., Mallin, M.A., Deamer-Melia, N.J., and Reed, R.E., 2001, Field ecology of toxic *Pfiesteria* complex species and a conservative analysis of their role in estuarine fish kills: Environmental Health Perspectives, v. 109 (suppl. 5), p. 715–730.
406. Noga, E.J., Khoo, L., Stevens, J.B., Fan, Z., and Burkholder, J.M., 1996, Novel toxic dinoflagellate causes epidemic disease in estuarine fish: Marine Pollution Bulletin, v. 32, p. 219–224.
407. Law, M., 2001, Differential diagnosis of ulcerative lesions in fish: Environmental Health Perspectives, v. 109 (suppl. 5), p. 681–686.
408. Vogelbein, W.K., Shields, J.D., Haas, L.W., Reece, K.S., and Zwerner, D.E., 2001, Skin ulcers in estuarine fishes: a comparative pathological evaluation of wild and laboratory-exposed fish: Environmental Health Perspectives, v. 109 (suppl. 5), p. 687–693.
409. Glasgow, H.B., Jr., Burkholder, J.M., Schmechel, D.E., Tester, P.A., and Rublee, P.A., 1995, Insidious effects of a toxic estuarine dinoflagellate on fish survival and human health: Journal of Toxicology and Environmental Health, v. 46, p. 501–522.
410. Grattan, L.M., Oldach, D., Perl, T.M., Lowitt, M.H., Matuszak, D.L., Dickson, C., Parrott, C., Shoemaker, R.C., Kauffman, C.L., Wasserman, M.P., Hebel, J.R., Charache, P., and Morris, J.G., Jr., 1998, Learning and memory difficulties after environmental exposure to waterways containing toxin-producing *Pfiesteria* or *Pfiesteria*-like dinoflagellates: Lancet, v. 352, p. 532–539.
411. Morris, J.G., 2001, Human health effects and *Pfiesteria* exposure: a synthesis of available clinical data: Environmental Health Perspectives, v. 109 (suppl. 5), p. 787–790.
412. McGeehin, M.A., and Rubin, C.H., eds., 2001, *Pfiesteria* from biology to public health: U.S. Department of Health and Human Services, National Institute of Health, National Institute of Environmental Sciences, Environmental Health Perspectives, v. 109 (suppl. 5), p. 633–808.
413. Rubin, C., McGeehin, A., Holmes, A.K., Backer, L., Burreson, G., Earley, M.C., Griffith, D., Levine, R., Litaker, W., Mei, J., Naeher, L., Needham, L., Noga, E., Poli, M., and Rogers, H.S., 2001, Emerging areas of research reported during the CDC National Conference on *Pfiesteria*: from biology to public health: Environmental Health Perspectives, v. 109 (suppl. 5), p. 633–637.
414. Bossart, G.D., Baden, D.G., Ewing, R.Y., Roberts, B., and Wright, S.D., 1998, Brevetoxicosis in manatees (*Trichechus manatus latirostris*) from the 1996 epizootic: gross, histologic, and immunohistochemical features: Toxicologic Pathology, v. 26, p. 276–282.
415. O'Shea, T.J., Rathbun, G.B., Bonde, R.K., Buergelt, C.D., and Odell, D.K., 1991, An epizootic of Florida manatees associated with dinoflagellate bloom: Marine Mammal Science, v. 7, p. 165–179.
416. Work, T.M., Barr, B., Beale, A.M., Fritz, L., Quilliam, M.A., and Wright, J.L.C.,1993, Epidemiology of domoic acid poisoning in brown pelicans (*Pelecanus occidentalis*) and Brandt's cormorants (*Phalacrocorax penicillatus*) in

California: Journal of Zoo and Wildlife Medicine, v. 24, p. 54–62.
417. Hofmann, E., Ford, S., Powell, E., and Klinck, J., 2001, Modeling studies of the effect of climate variability on MSX disease in eastern oyster (*Crassostrea virginica*) populations: Hydrobiologia, v. 460, p. 195–212.
418. Farley, C.A., 1975, Epizootic and enzootic aspects of *Minchinua nelsoni* (Haplosporida) disease in Maryland oysters: Journal of Protozoology, v. 22, p. 418–427.
419. Haaker, P.L., Harris, S.L., and Taniguchi, I. K., 1995, Withering syndrome in California: does it occur in subtidal abalones?: Aquaculture '95, Abstracts, Feb. 1-4, p. 267.
420. Friedman, C.S., Thomson, M., Chun, C., Haaker, P.L., and Hedrick, R.P., 1997, Withering syndrome of the black abalone, *Haliotis cracherodii* (Leach): water, temperature, food availability and parasites as possible causes: Journal of Shellfish Research, v. 16, p. 403–411.
421. Haaker, P.L., Harris, S.L., Taniguchi, I.K., and Friedman, C.S., 1995, Withering syndrome in California: does it occur in subtidal abalones: Journal of Shellfish Research, v. 14, p. 267.
422. Kleinschuster, S.J., Smolowitz, R., and Parent, J., 1998, *In vitro* life-cycle and propagation of quahog parasite unknown: Journal of Shellfish Research, v. 17, p. 75–78.
423. Whyte, S.K., Cawthorn, R.J., and McGladdery, S.E., 1994, QPX (quahuag parasite X) a pathogen of northern quahog *Mercenaria mercenaria* from the Gulf of St. Lawrence, Canada: Diseases of Aquatic Organisms, v. 19, p. 129–136.
424. Lee, M., Taylor, G.T., Bricelj, V.M., Ford, S.E., and Zahn, S., 1996, Evaluation of *Vibrio* spp. and microplankton blooms as causative agents of juvenile oyster disease in *Crassostrea virginica* (Gmelin): Journal of Shellfish Research, v. 15, p. 319–329.
425. Lewis, E.J., Farley, C.A., Small, E.B., and Baya, A.M., 1996, A synopsis of juvenile oyster disease (JOD) experimental studies in *Crassostrea virginica*: Aquatic Living Research, v. 9, p. 169–178.
426. Farley, C.A., Plutschak, D.L., and Scott, R.F., 1991, Epizootiology and distribution of transmissible sarcoma in Maryland softshell clams, *Mya arenaria*, 1984–1988: Environmental Health Perspectives, v. 90, p. 35–41.
427. Dungan, C.F., Hamilton, R.M., Hudson, K.L., McCollough, C.B., and Reece, K.S., 2002, Two epizootic diseases in Chesapeake Bay commercial clams, *Mya arenaria* and *Tagelus plebeius*: Diseases of Aquatic Organisms, v. 50, p. 67–78.
428. Office International des Epizooties, 2002, Mikrocytosis (*Mikrocytos mackini*) in the United States of America: Disease Information, v. 15, 19 July 2002.
429. Quayle, D.B., 1961, Denman Island oyster disease and mortality, 1960: Journal of Fisheries Research Board of Canada, v. 713, p. 1–9.
430. La Peyre, M.K., Nickens, A.D., Volety, A.K., Tolley, G.S., and La Peyre, J.F., 2003, Environmental significance of freshets in reducing *Perkinsus marinus* infection in eastern oysters *Crassostrea virginica*: potential management applications: Marine Ecology Progress Series, v. 248, p. 165–176.
431. Ford, S.E., 1996, Range extension by the oyster parasite *Perkinsus marinus* into the Northeastern U.S.: response to climate change?: Journal of Shellfish Research, v. 15, p. 45–56.
432. Lightner, D.V., 1999, The penaeid shrimp viruses TSV, IHHNV, WSSV and YHV: current status in the Americas, available diagnostic methods and management strategies: Journal of Applied Aquaculture, v. 9, p. 27–52.
433. Hasson, K.W., Lightner, D.V., Poulos, B.T., Redman, R.M., White, B.L., Brock, J.A., and Bonami, J.R., 1995, Taura syndrome in *Penaeus vannamei*: demonstration of viral etiology: Diseases of Aquatic Organisms, v. 23, p. 115–126.
434. Lightner, D.V., 1995, Taura syndrome: an economically important viral disease impacting the shrimp farming industries of the Americas including the United States: Proceedings of the United States Animal Health Association, 99th Annual Meeting.
435. Lightner, D.V., Redman, R.M., Poulos, B.T., Nunan, L.M., Mari, J.L., and Hasson, K.W., 1997, Risk of spread of penaeid shrimp viruses in the Americas by the international movement of live and frozen shrimp: Revue Scientifique et Technique, Office International des Epizooties, v. 16, p. 146–160.
436. Vogan, C.L., Costa-Ramos, C., and Rowley, A.F., 2001, A histological study of shell disease syndrome in the edible crab *Cancer pagurus*: Diseases of Aquatic Organisms, v. 47, p. 209–217.
437. Cowan, D.F., House, C., and House, J.A., 2001, Public Health, *in* Dierauf, L.A., and Gulland, F.M.D., eds., CRC handbook of marine mammal medicine (2nd ed.): Boca Raton, Fla., CRC Press, p. 767–778.
438. Hicks, B.D., and Worthy, G.A.J., 1987, Sealpox in captive grey seals (*Halichoerus grypus*) and their handlers: Journal of Wildlife Diseases, v. 23, p. 1–6.
439. Barlough, J.E., Berry, E.S., Skilling, D.E., and Smith, A.W., 1986, The marine calicivirus story-Part II: Compendium on Continuing Education for the Practicing Veterinarian, v. 8, p. F75–F82.
440. Smith, A.W., and Boyt, P.M., 1990, Calciviruses of ocean origin: a review: Journal of Zoo and Wildlife Medicine, v. 21, p. 3–23.
441. Webster, R.G., Geraci, J., Petursson, G., and Skirnisson, K., 1981, Conjunctivitis in human beings caused by influenza A virus of seals: New England Journal of Medicine, v. 304, p. 911.
442. Osterhaus, A.D.M.E., Rimmelzwaan, G.F., Martina, B.E.E., Bestebroer, T.M., and Fouchier, R., 2000, Influenza B virus in seals: Science, v. 288, p. 1051–1053.
443. Brew, S.D., Perrett, L.L., Stack, J.A., and MacMillan, A.P., 1999, Human exposure to *Brucella* recovered from a sea mammal: Veterinary Record, v. 144, p. 483.
444. Sohn, A.H., Probert, W.S., Glaser, C.A., Gupta, N., Bollen, A.W., Wong, J.D., Grace, E.M., and McDonald, W.C., 2003, Human neurobrucellosis with interacerebral granuloma caused by a marine mammal *Brucella* spp.: Emerging Infectious Diseases, v. 9, p. 485–488.
445. Suer, L.D., and Vedros, N.A., 1988, *Erysipelothrix rhusiopathiae* I. Isolation and characterization from pinnipeds and bite abrasion wounds in humans: Diseases of Aquatic Organisms, v. 5, p. 1–5.
446. Thompson, P.J., Cousins, D.V., Gow, B.L., Collins, D.M., Williamson, B.H., and Dagnia, H.T., 1993, Seals, seal

trainers, and mycobacterial infection: American Review of Respiratory Disease, v. 147, p. 164–167.
447. Flowers, D.J., 1970, Human infection due to *Mycobacterium marinum* after a dolphin bite: Journal of Clinical Pathology, v. 23, p. 475–477.
448. Hillenbrand, F.K.M., 1953, Whale finger and seal finger their relation to erysipeloid: The Lancet, April 4*th*, p. 680–682.
449. Beck, B., and Smith, T.G., 1976, Seal finger: an unsolved medical problem in Canada: Canadian Medical Association Journal, v. 115, p. 105–107.
450. Baker, A.S., Ruoff, K.L., and Madoff, S., 1998, Isolation of *Mycoplasma* species from a patient with seal finger: Clinical Infectious Diseases, v. 27, p. 1168–1170.
451. Stadtlander, C.T.K.-H., and Madoff, S., 1994, Characterization of cytopathogenicity of aquarium seal mycoplasmas and seal finger mycoplasmas by light and scanning electron microscopy: Zentralbatt fur Bakteriologie, v. 280, p. 458–467.
452. Bender, T.R., Jones, T.S., DeWitt, W.E., Kaplan, G.J., Saslow, A.R., Nevius, S.E., Clark, P.S., and Gangarosa, E.J., 1972, Salmonellosis associated with whale meat in an Eskimo community: American Journal of Epidemiology, v. 96, p. 153–160.
453. Haubold, E.M., Aronson, J.F., Cowan, D.F., McGinnis, M.R., and Cooper, C.R. Jr., 1998, Isolation of fungal rDNA from bottlenose dolphin skin infected with *Loboa loboi*: Medical Mycology, v. 36, p. 263–267.
454. Forbes, L.B., 2000, The occurrence and ecology of *Trichinella* in marine mammals: Veterinary Parasitology, v. 93, p. 321–334.
455. Thomas, N.J., 2001, Sea otter mortality: U.S. Geological Survey, National Wildlife Health Center Information Sheet, Madison, Wis., June 2001.
456. Cole, R.A., Lindsay, D.S., Howe, D.K., Roderick, C.L., Dubey, J.P., Thomas, N.J., and Baeten, L.A., 2000, Biological and molecular characterizations of *Toxoplasma gondii* obtained from southern sea otters (*Enhydra lutris nereis*): Journal of Parasitology, v. 86, p. 526–530.
457. Lindsay, D.S., Thomas, N.J., and Dubey, J.P., 2000, Biological characterization of *Sarcocystis neurona* isolated from a southern sea otter (*Enhydra lutris nereis*): International Journal of Parasitology, v. 30, p. 617–624.
458. Miller, M.A., Gardner, I.A., Kreuder, C., Paradies, D.M., Worcester, K.R., Jessup, D.A., Dodd, E., Harris, M.D., Ames, J.A., Packham, A.E., and Conrad, P.A., 2002, Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*): International Journal for Parasitology, v. 32, p. 997–1006.
459. Lindsay, D.S., Thomas, N.J., Rosypal, A.C., and Dubey, J.P., 2001, Dual *Sarcocystis neurona* and *Toxoplasma gondii* infection in a northern sea otter from Washington state, USA: Veterinary Parasitology, v. 97, p. 319–327.
460. Thomas, N.J., Pappagianis, D., Creekmore, L.H., and Duncan, R.M., 1996, Coccidioidomycosis in southern sea otters, *in* Einstein, H.E., and Catanzaro, A., eds., Coccidioidomycosis, Proceedings Fifth International Conference on Cocidiomycosis, Washington, D.C., National Foundation for Infectious Diseases, p. 163–173.
461. Thomas, Nancy J., 2003, U.S. Geological Survey, National Wildlife Health Center, personal communication.
462. Schmale, M.C., Gibbs, P.D.L., and Campbell, C.E., 2002, A virus-like agent associated with neurofibromatosis in damselfish: Diseases of Aquatic Organisms, v. 49, p. 107–115.
463. Schmale, M.C., and Kemerer, T.W., 1996, Melanophores in damselfish neurofibromatosis: alterations in morphology and melanosome aggregation responses: Journal of Aquatic Animal Health, v. 8, p. 1–12.
464. Bouchard, D.A., Keleher, W., Opitz, H.M., Blake, S., Edwards, K.C., and Nicholson, B.L., 1999, Isolation of infectious salmon anemia virus (ISAV) from Atlantic salmon in New Brunswick, Canada: Diseases of Aquatic Organisms, v. 35, p. 131–137.
465. Ferguson, H.W., St. John, V.S., Roach, C.J., Willoughby, S., Parker, C., and Ryan, R., 2000, Caribbean reef fish mortality associated with *Streptococcus iniae*: Veterinary Record, v. 147, p. 662–664.
466. Rhodes, M.W., Kator, H., Kotob, S., van Berkum, P., Kaattari, I., Vogelbein, W., Floyd, M.M., Butler, W.R., Quinn, F.D., Ottinger, C., and Shotts, E., 2001, A unique Mycobacterium species isolated from an epizootic of striped bass (*Morone saxatilis*): Emerging Infectious Diseases, v. 7, p. 896–899.
467. Blazer, V.S., Lilley, J.H., Schill, W.B., Kiryu, Y., Panyawachira, V, Densmore, C.L., and Chinabut, S., 2002, *Aphanomyces invadans* in Atlantic menhaden along the East Coast of the United States: Journal of Aquatic Animal Health, v. 14, p. 1–10.
468. Reed, A., and Cousineau, J.-G., 1967, Epidemics involving the common eider (*Somateria mollissima*) at Ile Blanche, Quebec: Naturaliste Canadien, v. 94, p. 327–334.
469. Gershman, M., Witter, J.F., Spencer, H.E., Jr., and Kalvaitis, A., 1964, Case report: epizootic of fowl cholera in the common eider duck: Journal of Wildlife Management, v. 28, p. 587–589.
470. Korschgen, C.E., Gibbs, H.C., and Mendall, H.L., 1978, Avian cholera in eider ducks in Maine: Journal of Wildlife Diseases, v. 14, p. 254–258.
471. Suarez, J.G., and Ilazabal, L.L., 1941, Epidemia de colera en los patos mainos: Revista de Medicina Veterinaria, v. 23, p. 145–149.
472. Swennen, C., and Smit, T., 1991, Pasteurellosis among breeding eiders *Somateria mollissima* in The Netherlands: Wildfowl, v. 42, p. 94–97.
473. Christensen, T.K., 1996, An outbreak of pasteurellosis in Denmark 1996: Wetlands International Seaduck Specialist Group Bulletin, no. 6 (December), p. 44–48.
474. Christensen, T.K., Bregnballe, T., Andersen, T.H., and Dietz, H.H., 1997, Outbreak of Pasteurellosis among wintering and breeding common eiders *Somateria mollissima* in Denmark: Wildlife Biology, v. 3, p. 125–128.
475. Kaschula, V.R., and Truter, D.E., 1951, Fowl cholera in sea gulls on Dassen Island: Journal of South African Veterinary Medical Association, v. 22, p. 191–192.
476. Crawford, R.J.M., Allwright, D.M., and Heyl, C.W., 1992, High mortality of Cape Cormorants (*Phalacrocorax capensis*) off Western South Africa in 1991 caused by *Pasteurella multocida*: Colonial Waterbirds, v. 15, p. 236–238.
477. Parmelee, D.F., Maxson, S.J., and Bernstein, N.P., 1979, Fowl cholera outbreak among brown skuas at Palmer Sta-

tion: Antarctic Journal of the United States., v. 14, p. 168–169.
478. de Lisle, G.W., Stanislawek, W.L., and Moors, P.J., 1990, *Pasteurella multocida* infections in rockhopper penguins (*Eudyptes chrysocome*) from Campbell Island, New Zealand: Journal of Wildlife Diseases, v. 26, p. 283–285.
479. Rafferty, K.A. Jr., 1965, The cultivation of inclusion-associated viruses from Lucke tumor frogs: Annals of the New York Academy of Sciences, v. 126, p. 3–21.
480. Wolf, K., Bullock, G.L., Dunbar, C.E., and Quimby, M.C., 1968, Tadpole edema virus—a viscerotropic pathogen for anuran amphibians: Journal of Infectious Diseases, v. 118, p. 253–262.
481. Mao, J., Green, D.E., Fellers, G., and Chinchar, V.G., 1999, Molecular characterization of iridoviruses isolated from sympatric amphibians and fish: Virus Research, v. 63, p. 45–52.
482. Jancovich, J.K., Davidson, E.W., Morado, J.F., Jacobs, B.L., and Collins, J.P., 1997, Isolation of a lethal virus from the endangered tiger salamander *Ambystoma tigrinum stebbinsi*: Diseases of Aquatic Organisms, v. 31, p. 161–167.
483. Bollinger, T.K., Mao, J., Schock, D., Brigham, R.M., and Chinchar, V.G., 1999, Pathology, isolation, and preliminary molecular characterization of a novel iridovirus from tiger salamanders in Saskatchewan: Journal of Wildlife Diseases, v. 35, p. 413–429.
484. Speare, R., and Smith, J.R., 1992, An iridovirus-like agent isolated from the ornate burrowing frog *Limnodynastes ornatus* in northern Australia: Diseases of Aquatic Organisms, v. 14, p. 51–57.
485. Cunningham, A.A., Langton, T.E.S., Bennett, P.M., Lewin, J.F., Drury, S.E.N., Gough, R.E., and Macgregor, S.K., 1996, Pathological and microbiological findings from incidents of unusual mortality of the common frog (*Rana temporaria*): Philosophical Transactions of the Royal Society of London, Series B, v. 351, p. 1539–1557.
486. Zhang, Q., Xiao, F., Li, Z., Gui, J., Mao, J., and Chinchar, V.G., 2001, Characterization of an iridovirus from the cultured pig frog *Rana grylio* with lethal syndrome: Diseases of Aquatic Organisms, v. 48, p. 27–36.
487. Docherty, D.E., Meteyer, C.U., Wang, J., Mao, J., Case, S.T., and Chinchar, V.G., 2003, Diagnostic and molecular evaluation of three iridovirus-associated salamander mortality events: Journal Wildlife Diseases, v. 39. p 556–566.
488. Morehouse, E.A., James, T.Y., Ganley, A.R.D., Vilgalys, R., Berger, L., Murphy, P.J., and Longcore, J.E., 2003, Mutilocus sequence typing suggests the chytrid pathogen of amphibians is a recently emerged clone: Molecular Ecology, v. 12, p. 395–403.
489. Blaustein, A.R., Hokit, D.G., O'Hara, R.K., and Holt, R.A., 1994, Pathogenic fungus contributes to amphibian losses in the Pacific Northwest: Biological Conservation, v. 67, p. 251–254.
490. Berger, L., Speare, R., and Hyatt, A., 1999, Chytrid fungi and amphibian declines: overview, implications and future directions, *in* Campbell, A., ed., Declines and disappearances of Australian frogs: Canberra, Environment Australia, p. 23–33.
491. Johnson, P.T., Lunde, K.B., Thurman, E.M., Ritchie, E.G., Wray, S.N., Sutherland, D.R., Kapfer, J.M., Frest, T.J., Bowerman, J., and Blaustein, A.R., 2002, Parasite (*Ribeiroia ondatrae*) infection linked to amphibian malformations in the western United States: Ecological Monographs, v. 72, p. 151–168.
492. Schotthoefer, A.M., Koehler, A.V., Meteyer, C.U., and Cole, R.A., 2003, Influence of *Ribeiroia ondatrae* (Trematoda: Digenea) infection on limb development and survival of northern leopard frogs (*Rana pipiens*)—effects of host-stage and parasite exposure level: Canadian Journal of Zoology, v. 81, p. 1144–1153.
493. Ankley, G.T., Tietge, J.E., DeFoe, D.L., Jensen, K.M., Holcombe, G.W., Durhan, E.J., and Diamond, S.A., 1998, Effects of ultraviolet light and methoprene on survival and development of *Rana pipiens*: Environmental Toxicology and Chemistry, v. 17, p. 2530–2542.
494. Choudhury, A., Hoffnagle, T.L., and Cole, R.A., 2003, Parasites of native and non-native fishes of the Little Colorado River, Grand Canyon, Arizona: Journal of Parasitology (in press).
495. Office International des Epizooties, 2002, Spring viraemia of carp in the United States of America: confirmation of disease in wild fish in the State of Wisconsin: Disease Information, v. 15 (October), p. 18.
496. Gray, W.L., Mullis, L., LaPatra, S.E., Groff, J.M., and Goodwin, A., 2002, Detection of koi herpesvirus DNA in tissues of infected fish: Journal of Fish Disease, v. 25, p. 171–178.
497. Grizzle, J.M., Altinok, I., Fraser, W.A., and Francis-Floyd, R., 2002, First isolation of largemouth bass virus: Diseases of Aquatic Organisms, v. 50, p. 233–235.
498. Plumb, J.A., Grizzle, J.M., Young, H.E., Noyes, A.D., and Lamprecht, S., 1996, An iridovirus isolated from wild largemouth bass: Journal of Aquatic Animal Health, v. 8, p. 265–270.
499. Woodland, J.E., Brunner, C.J., Noyes, A.D., and Grizzle, J.M., 2002, Experimental oral transmission of largemouth bass virus: Journal of Fish Disease, v. 25 p. 669–672.
500. Hedrick, R.P., Groff, J.M., McDowell, T., and Wingfield, W.H., 1990, An iridovirus infection of the integument of the white sturgeon *Acipenser transmontanus*: Diseases of Aquatic Organisms, v. 8, p. 39–44.
501. Watson, L.R., Yun, S.C., Groff, J.M., and Hedrick, R.P., 1995, Characteristics and pathogenicity of a novel herpesvirus isolated from adult and subadult white sturgeon *Acipenser transmontanus*: Diseases of Aquatic Organsims, v. 22, p. 199–210.
502. Office International des Epizooties, 2001, International Aquatic Animal Health Code, fish, mollusks, and crustaceans (4th ed.): Paris, Office International des Epizooties, 155 p.
503. Petrides, G.A., and Byrant, C.R., 1951, An analysis of the 1949–1950 fowl cholera epizootic in Texas panhandle waterfowl: Transactions of the North American Wildlife Conference, v. 16, p. 193–216
504. Jensen, W.I., and Williams, C.S., 1964, Botulism and fowl cholera, *in* Linduska, J.P., and Nelson, A.L., eds., Waterfowl tomorrow: Washington, D.C., U.S. Department of the Interior, Fish and Wildlife Service, p. 333–341
505. Rosen, M.N., and Bischoff, A.I., 1949, The 1948–1949 outbreak of fowl cholera in birds in the San Fransico Bay

area and surrounding countries: California Fish and Game, v. 35, p. 185–192.
506. Rosen, M.N., and Bischoff, A.I., 1950, The epidemiology of fowl cholera as it occurs in the wild: Transactions of the North American Wildlife Conference, v. 15, p. 147–154.
507. Vaught, R.W., McDougle, H.C., and Burgess, H.H., 1967, Fowl cholera in waterfowl of Squaw Creek National Wildlife Refuge, Missouri: Journal of Wildlife Management, v. 31, p. 248–253.
508. Klukas, R.W., and Locke, L.N., 1970, An outbreak of fowl cholera in Everglades National Park: Journal of Wildlife Diseases, v. 6, p. 77–79.
509. Zinkl, J.G., Dey, N., Hyland, J.M., Hurt, J.J., and Heddleston, K.L., 1977, An epornitic of avian cholera in waterfowl and common crows in Phelps County, Nebraska, in the spring, 1975: Journal of Wildlife Diseases, v. 13, p. 194–198.
510. Windingstad, R.M., Hurt, J.J., Trout, A.K., and Cary, J., 1984, Avian cholera in Nebraska's rainwater basin: Transactions of the North American Wildlife Conference, v. 49, p. 576–583.
511. Friend, M., 2002, Avian disease at the Salton Sea: Hydrobiologia, v. 473, p. 293–306.
512. Pursglove, S.R. Jr., Holland, D.F., Settle, F.H., and Gnegy, D.C., 1976, Control of a fowl cholera outbreak among coots in Virginia: Proceedings of the Southeastern Association of Game and Fish Commission, v. 30, p. 602–609.
513. Wobeser, G., and Leighton, F.A., 1988, Avian cholera epizootic in wild ducks: Canadian Veterinary Journal, v. 29, p. 1015–1016.
514. Samuel, M.D., Takekawa, J.Y., Samelius, G., and Goldberg, D.R., 1999, Avian cholera mortality in lesser snow geese nesting on Banks Island, Northwest Territories: Wildlife Society Bulletin, v. 27, p. 780–787.
515. Morell, V., 1996, New virus variant killed Serengeti cats: Science, v. 271, p. 596.
516. Lehmkuhl, H.D., Hobbs, L.A., and Woods, L.W., 2001, Characterization of new adenovirus isolated from black-tailed deer in California: Archives of Virology, v. 146, p. 1187–1196.
517. Woods, L.W., Swift, P.K., Barr, B.C., Horzinek, M.C., Nordhausen, R.W., Stillian, M.H., Patton, J.F., Oliver, M.N., Jones, K.R., and MacLachlan, N.J., 1996, Systemic adenovirus infection associated with high mortality in mule deer (*Odocoileus hemionus*) in California: Veterinary Pathology, v. 33, p. 125–132.
518. Woods, L.W., Hanley, R.S., Chiu, P.H.W., Burd, M., Nordhausen, R.W., Stillian, M.H., and Swift, P.K., 1997, Experimental adenovirus hemorrhagic disease in yearling black-tailed deer: Journal of Wildlife Diseases, v. 33, p. 801–811.
519. Cully, J.F. Jr., Barnes, A.M., Quan, T.J., and Maupin, G., 1997, Dynamics of plague in a Gunnison's prairie dog colony complex from New Mexico: Journal of Wildlife Diseases, v. 33, p. 706–719.
520. Hugh-Jones, M.E., and de Vos, V., 2002, Anthrax and wildlife: Revue Scientifique et Technique, Office International des Epizooties, v. 21, p. 359–383.
521. Kock, R.A., Wambua, J.M., Mwanzia, J., Wamwayi, H., Ndungu, E.K., Barrett, T., Kock, N.D., and Rossiter, P.B., 1999, Rinderpest epidemic in wild ruminants in Kenya 1993–1997: Veterinary Record, v. 145, p. 275–283.
522. Cooke, B.D., 2002, Rabbit haemorrhagic disease: field epidemiology and the management of wild rabbit populations: Revue Scientifique et Technique, Office International des Epizooties, v. 21, p. 347–358.
523. Bornstein, S., Mörner, T. and Samuel, W.M., 2001, *Sarcoptes scabiei* and sarcoptic mange, *in* Samuel, W.M., Pybus, M.J., and Kocan, A.A., eds., Parasitic diseases of wild mammals (2nd ed.): Ames, Iowa, Iowa State University Press, p. 107–119.
524. Pence, D.B., and Ueckermann, E., 2002, Sarcoptic mange in wildlife: Revue Scientifique et Technique, Office International des Epizooties, v. 21, p. 385–398.
525. Skerratt, L.F., Campbell, N.J.H., Murrell, A., Walton, S., Kemp, D., and Barker, S.C., 2002, The mitochondrial 12S gene is a suitable marker of populations of *Sarcoptes scabiei* from wombats, dogs and humans in Australia: Parasitology Research, v. 88, p. 376–379.
526. McLean, R.G., 2002, West Nile Virus—a threat to North American avian species: Transactions of the North American Wildlife and Natural Resources Conference, v. 67, p. 62–74.
527. ProMED-mail, 2003, Rabies and vulture die-off (India), 20030207.0329: accessed Feb. 7, 2003 at URL http://www.promedmail.org.
528. Doster, G.L., 1999, Aflatoxicosis in Louisiana geese: Southeastern Cooperative Wildlife Disease Study Briefs, University of Georgia, v. 15, p. 1–2.
529. Artois, M., Delahay, R., Guberti, V., and Cheeseman, C., 2001, Control of infectious diseases of wildlife in Europe: The Veterinary Journal, v. 162, p. 141–152.
530 ProMED-mail, 2002, Chronic wasting disease, cervids—USA (IL), December 16, 2002, 20021216.6078: accessed December 17, 2002, at URL http://www.promedmail.org.
531. Williams, E.S., Kirkwood, J.K., and Miller, M.W., 2001, Transmissable spongiform encephalopathies, *in* Williams, E.S., and Barker, I.K., eds., Infectious diseases of wild mammals (3*rd* ed.): Ames, Iowa, Iowa State University Press, p. 292–301.
532. Schmitt, S.M., Fitzgerald, S.D., Cooley, T.M., Bruning-Fann, C.S., Sullivan, L., Berry, D., Carlson, T., Minnis, R.B., Payeur, J.B., and Sikarskie, J., 1997, Bovine tuberculosis in free-ranging white-tailed deer from Michigan: Journal of Wildlife Disease, v. 33, p. 749–758.
533. de Lisle, G.W., Bengis, R.G., Schmitt, S.M., and O'Brien, D.J., 2002, Tuberculosis in free-ranging wildlife: detection, diagnosis and management: Revue Scientifique et Technique, Office International des Épizooties, v. 21, p. 317–334.
534. Giacometti, M., Janovsky, M., Belloy, L., and Frey, J., 2002, Infectious keratoconjunctivitis of ibex, chamois and other Caprinae: Revue Scientifique et Technique, Office International des Epizooties, v. 21, p. 335–345.
535. Centers for Disease Control, 1994, Raccoon rabies epizootic—United States, 1993: Morbidity and Mortality Weekly Report, v. 43, p. 269–272.
536. Chang, H.G.H., Eidson, M., Noonan-Toly, C., Trimarchi, C.V., Rudd, R., Wallace, B.J., Smith, P., and Morse, D.L., 2002, Public health impact of reemergence of rabies, New

York: Emerging Infectious Diseases, v. 8, p. 909–913.
537. Fishbein, D.B., and Robinson, L.E., 1993, Rabies: New England Journal of Medicine, v. 329, p. 1632–1638.
538. Rupprecht, C.E., Smith, J.S., Fekadu, M., and Childs, J.E., 1995, The ascension of wildlife rabies: a cause for public health concern or intervention: Emerging Infectious Diseases, v. 1, p. 107–114.
539. Barker, I.K., and Parrish, C.R., 2001, Parvovirus infections, *in* Williams, E.S., and Barker, I.K., eds., Infectious diseases of wild mammals (3rd ed.): Ames, Iowa, Iowa State University Press, p. 131–146.
540. Brand, C.J., 2002, Landscape ecology of plague in the American Southwest, September 19–20, 2000, Fort Collins, Colorado, Proceedings of an American Southwest Workshop: U.S. Geological Survey, Information and Technology Report 2002–0001, 24 p.
541. Swearengen, J.R., and Worsham, P.L., 2000, Plague, *in* Brown, C., and Bolin, C., eds., Emerging diseases of animals: Washington, D.C., ASM Press, p. 259–279.
542. Docherty, D.E., Converse, K.A., Hansen, W.R., and Norman, G.W., 1994, American woodcock (*Scolopax minor*) mortality associated with a reovirus: Avian Diseases, v. 38 p. 899–904.
543. Docherty, D.E., Hansen, W., Franson, J.C., Meteyer, C., and Converse, K., 1997, Second occurrence of woodcock mortality associated with orthoreovirus: U.S. Geological Survey, Biological Resources, Biological Information & Technology Notes, no. 97–002.
544. Kirkwood, J.K., 1998, Population density and infectious disease at bird tables: The Veterinary Record, v. 142, p. 468.
545. Hansen, W., 1999, Avian pox, *in* Friend, M., and Franson, J.C., eds., Field manual of wildlife diseases—general field procedures and diseases of birds: U.S. Geological Survey, Information and Technology Report 1999–001, p. 163–169.
546. Hyatt, A.D., Williamson, M., Coupar, B.E.H., Middleton, D., Hengstberger, S.G., Gould, A.R., Selleck, P., Wise, T.G., Kattenbelt, J., Cunningham, A.A., and Lee, J., 2002, First identification of a ranavirus from green pythons (*Chondropython viridis*): Journal of Wildlife Diseases, v. 38, p. 239–252.
547. Taormina, P.J., Beuchat, L.R., and Slutsker, L., 1999, Infections associated with eating seed sprouts: an international concern: Emerging Infectious Diseases, v. 5, p. 626–634.
548. Soh, C.-T., 1991, Current status of food-borne parasitic zoonoses in Korea: The Southeast Asian Journal of Tropical Medicine and Public Health, v. 22, (suppl.) p. 54–55.
549. Vidaver, A.K., 1996, Emerging and reemerging infectious diseases; perspectives on plants, animals, and humans: ASM News, v. 62, p. 583–585.
550. Brown, C., 2000, Emerging infectious diseases of animals: an overview, *in* Brown, C. and Bolin, C., eds., Emerging diseases of animals: Washington, D.C., ASM Press, p. 1–12.
551. Ruiz, G.M., Rawlings, T.K., Dobbs, F.C., Drake, L.A., Mullady, T., Huq, A., and Colwell, R.R., 2000, Global spread of microorganisms by ships: Nature, v. 408, p. 49–50.
552. Ellis, J.S., Alvarez-Agureo, A., Gregory, V., Lin, Y.P., Hay, A., and Zambon, M.C., 2003, Influenza A H1N2 viruses, United Kingdom, 2001–02 influenza season: Emerging Infectious Diseases, v. 9, p. 304–310.
553. Swayne, D.E., 2000, Understanding the ecology and epidemiology of avian influenza viruses: implications for zoonotic potential, *in* Brown, C., and Bolin, C., eds., Emerging diseases of animals: Washington, D.C., ASM Press, p. 101–130.
554. Webby, R.J., and Webster, R.G., 2001, Emergence of influenza A viruses: Philosophical Transactions of the Royal Society of London, Series B, v. 356, p. 1817–1828.
555. Huff, J.L., and Barry, P.A., 2003, B-virus (*Cercopithecine herpesvirus* 1) infection in humans and macaques: potential for zoonotic disease: Emerging Infectious Diseases, v. 9, p. 246–250.
556. McCormick, J.B, and Fisher-Hoch, S.P., 1994, Zoonoses caused by filoviridae, *in* Beran, G.W., and Steele, J.H., eds., Handbook of zoonoses (2nd ed.): Boca Raton, Fla., CRC Press, p. 375–383.
557. Hooper, P.T., 2000, New fruit bat viruses affecting horses, pigs, and humans, *in* Brown, C., and Bolin, C., eds., Emerging diseases of animals: Washington, D.C., ASM Press, p. 85–99.
558. Breman, J.G., 2000, Monkeypox—an emerging infection for humans?, *in* Scheld, W.M., and others, eds., Emerging infections (4th ed.): Washington, D.C., ASM Press, p. 45–67.
559. Calisher, C.H., Mills, J.N., Root, J.J., and Beaty, B.J., 2003, Hantaviruses: etiologic agents of rare, but potentially life-threatening zoonotic diseases: Journal of the American Veterinary Medical Association, v. 222, p. 163–166.
560. Clement, J., McKenna, P., van der Groen, G., Vaheri, A., and Peters, C.J., 1998, Hantaviruses, *in* Palmer, S.R., Soulsby, L., and Simpson, D.I.H., eds., Zoonoses: biology, clinical practice, and public health control: New York, Oxford University Press, p. 331–351.
561. Thompson, C., Spielman, A., and Krause, P.J., 2001, Coinfecting deer-associated zoonoses: lyme disease, babesiosis, and ehrlichiosis: Clinical Infectious Diseases, v. 33, p. 676–685.
562. Yabsley, M.J., Varela, A.S., Tate, C.M., Dugan, V.G., Stallknecht, D.E., Little, S.E., and Davidson, W.R., 2002, *Ehrlichia ewingii* infection in white-tailed deer (*Odocoileus virginianus*): Emerging Infectious Diseases, v. 8, p. 668–671.
563. Kjemtrup, A.M., and Conrad, P.A., 2000, Human babesiosis: an emerging tick-borne disease: International Journal of Parasitology, v. 30, p. 1323–1337.
564. Needham, D.J., 1992, Tuberculosis/mycobacteriosis in pinnipeds: Proceedings 23rd Annual Conference of the International Association for Aquatic Medicine, p. 92–96
565. Green, D.E., 2003, U.S. Geological Survey, National Wildlife Health Center, personal communication.
566. Herman, R.L., 1984, Ichthyophonus-like infection in newts (*Notophthalmus viridescens* Rafinesque): Journal of Wildlife Diseases, v. 20, p. 55–56.
567. Jay, J.M., and Pohley, W.J., 1981, *Dermosporidium penneri* sp. n. from the skin of the American toad, *Bufo americanus* (Amphibia: Bufonidae): Journal of Parasitology, v. 67, p. 108–110.
568. Guyénot, E., and Naville, A., 1922, Un nouveau Protiste du

genre *Dermocystidium*, parasite de la grenouille: *Dermocystidium ranae* n. sp.: Revue Suisse de Zoologie, v. 29, p. 133–145.
569. Williams, E.H., Jr., 1991, Threat to black sea urchins: Nature, v. 352, p. 385.
570. Enserink, M., and Kaiser, J., 2004, Avian flu finds new mammal hosts: Science, v. 305, p. 1385.
571. Kaiser, J., 2004, U.S. releases draft plan for dealing with pandemic flu: Science, v. 305, p. 1387.
572. Oaks, J.L., Gilbert, M., Virani, M.Z, Watson, R.T., Meteyer, C.U., Rideout, B.A., Shivaprasad, H.L., Ahmed, S., Chaudhry, M.J., Arshad, M., Mahmood, S., Ali, A., and Khan, A.A., 2004, Diclofenac residues as the cause of vulture population decline in Pakistan: Nature, v. 427, p. 568–569.
573. Greenblatt, R.J., Quackenbush, S.L., Casey, R.N., Rovnak, J., Balazs, G.H., Work, T.M., Casey, J.W., and Sutton, C.A., 2005, Genomic variation of the fibropapilloma-associated marine turtle herpesvirus across seven geographic areas and three host species: Journal of Virology, v. 79, p. 1125–1132.