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OVERVIEW

DISEASE EMERGENCE IN BIRDS: CHALLENGES FOR THE TWENTY-FIRST CENTURY

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THE PAPER BY Hartup et al. (2001) on House Finch (*Carpodacus mexicanus*) conjunctivitis is an example of the rapid geographic spread that can result from disease emergence in naïve populations. That event was neither novel nor transient relative to its occurrence or effects. Disease emergence and reemergence are hallmarks of the latter part of the twentieth century (Center for Disease Control 1994, Levins et al. 1994, DaSilva and Laccarino 1999, Gratz 1999). Current examples involving domestic animals include the problems in Europe with bovine spongiform encephalopathy (BSE, or ''mad cow disease'') (Brown 2001) and foot-and-mouth disease (FMD) (Kitching 1999). Human health has been affected by diseases caused by an array of viruses (Morse 1993, Nichol et al. 1993, Murphy and Nathanson 1994), bacteria (Dennis 1998, DaSilva and Laccarino 1999), rickettsia (Walker and Dumier 1996, Azad et al. 1997), protozoans (Tuerrant 1997, Saini et al. 2000), and metazoan parasites (Hildreth et al. 1991, Gubler 1998), as well as other causes. Acquired immune deficiency syndrome (AIDS) has received the most notoriety of those diseases (Hahn et al. 2000, Schwartlander et al. 2000). A similar pattern exists on a global scale for free-ranging wildlife populations (Table 1) (Friend 1994, 1995; Epstein et al. 1998, Daszak et al. 2000). However, in comparison to disease emergence affecting humans and domestic animals, response to emerging diseases of wildlife is generally superficial. We present concepts and data to support our contention that failure to adequately address disease emergence in free-ranging wildlife is resulting in a diminished capability to achieve and sustain desired geographic distributions and population abundance for species of wild birds, including some threatened and endangered avifauna.

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For clarity, we define *disease* and *disease emergence* in the context of our use of those terms because they are the focus of our comments. *Disease* is any departure from health (Guralnik 1982); that is, dysfunction contributing to physiological, physical, reproductive, behavioral, or other impairment that reduces the probability of survival of individuals. If enough individuals are affected, the collective effects can reduce the sustainability of the population. Although disease can result from exposure to a wide variety of physical, chemical, and biological agents and other conditions, we focus this paper on microbes and parasites and to overt mortality caused by them. Thus, disease effects presented only represent the proverbial ''tip of the iceberg'' relative to the challenges wild avifauna face from disease. Our perspective of *disease emergence* expands the earlier definitions of emerging diseases by others (Centers for Disease Control and Prevention 1994, Morse 1995) to include all species. Our comments are defined by the context of disease occurrences that have increased within the past three decades, or threaten to increase in the near future relative to populations affected, geographic distribution, or magnitude of effects.

DISEASE EFFECTS

Haldane (1949) cited by May (1988) stated ''. . . infectious diseases have undoubtedly been the main agents of morbidity and mortality (and thus the dominant selective factors) in human populations at least for the past 10,000 years.'' Support for the continued dominance of microbes and protozoan and metazoan parasites over humankind can be found in the writings of such notable scholars as McNeill (1976) and Lederberg (1988, 1993, 1997).

It is folly to think that wild birds and other wildlife are less susceptible to the influences of disease than humans and domestic animals. Nevertheless, ''. . . ecologists and evolutionary

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biologists virtually ignored parasites [including microbes] until recently, even as a source of mortality for host species [wildlife] of primary interest'' (Toft 1991). This is inconsistent with the viewpoints of Price (1980) that ''. . . parasites affect the life and death of practically every other living organism;'' and that

> . . . parasites are likely to play a role in practically every aspect of the evolutionary biology of birds, and probably vertebrates in general. Such a view has been a long time in gestation, probably because in a fetal condition it was roundly thwarted by two eminent ecologists (Price 1991).

The writings of Elton (1927) and Lack (1954, 1966) and support for their conclusions by other notable ecologists suppressed for decades considerations of disease as a significant factor for avian population dynamics (Price 1991).

More recent evaluations have clearly demonstrated that the emergence of avian malaria (*Plasmodium relictum*) and avian pox in Hawaii has regulated the geographic distribution and abundance of native Hawaiian forest birds on the Island of Hawaii (Warner 1968, van Riper et al. 1986, Atkinson et al. 1995). Others (Hudson 1986, Hudson and Dobson 1991) have shown that the parasite *Trichostrongylus tenuis* regulates population numbers of Red Grouse (*Lagopus lagopus scoticus*).

The biological significance of *Mycoplasma gallisepticum* infections in House Finches is equivocal. The Hartup et al. (2001) study did not detect gross differences in survival for diseased and normal finches. However, others have concluded that mycoplasmal conjunctivitis is a significant mortality factor for eastern populations of House Finches and is capable of causing population declines (Luttrell et al. 1998, Nolan et al. 1998, Hochachka and Dhondt 2000). Our purpose is not to debate the merits of different evaluations involving House Finch conjunctivitis. Instead, we note that this new disease for wild birds is a recent addition to the continuum of emerging diseases that are challenging the integrity of avian communities (Table 2).

It is noteworthy that disease challenges for avian communities are occurring in some of the more pristine ecosystems on Earth. For example, it has recently been stated that

> the birds of the Galapagos now confront serious problems caused by introduced diseases. . . . For Galapagos to survive, increased technical and financial means must be found to defeat the worst of the plagues and to monitor and respond to the threats of these introduced species. (Vargas 2000).

Avian disease has also become an issue in Antarctic penguins. An infectious agent is suspected as the cause for mass mortality in Adelie Penguin (*Pygoscelis adelaiae*) chicks during the 1990s (Gardner et al. 1997a, b). More recently, antibodies to infectious bursal disease virus (IBDV), a pathogen of domestic chickens (*Gallus domesticus*), have been found in sera collected from wild Emperor (*Aptenodytes forsteri*) and Adelie penguins (Gardner et al. 1997a, b). Antibodies to IBDV have also been found in sera of Spectacled Eiders (*Somateria fischeri*) nesting in a remote area of western Alaska and in nesting Common Eiders (*S. mollissima*) and Herring Gulls (*Larus argentatus*) in the Baltic Sea (Hollmén et al. 2000).

Disease emergence.—Disease emergence in avifauna has taken different forms (Table 3), and the frequency of recurrence, geographic spread, species affected, and magnitude of losses following initial events have generally been unpredictable and highly variable. For example, it took only three years from the first reported case of House Finch conjunctivitis in 1994 in the Washington, D.C. area to steadily spread westward to the Mississippi River and essentially occupy the entire eastern range of House Finches (Fischer et al. 1997, Friend 1999a). Other diseases, such as the reovirus responsible for the deaths of moderate numbers of American Woodcock (*Scolopax minor*), have remained highly localized (Docherty et al. 1994). Only a single recurrence of that reovirus has been documented (Docherty 1999). In contrast, annual losses of substantial numbers of deaths annually of American Coot (*Fulica americana*) have been caused by the trematode *Leyogonimus polyoon* (Cole and Friend 1999). Duck plague has spread from the initial 1967 outbreak in wild waterfowl on Long Island, New York (Leibovitz and Hwang 1968), in a haphazard manner across much of the United States and into Canada (Friend 1999b, Converse and Kidd 2001).

Variability also has been great relative to species affected. House Finch conjunctivitis in the wild has essentially been limited to House Finches. Duck plague is a disease limited to Anseriformes (Sandhu and Leibovitz 1997),

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TABLE 1. Examples of disease emergence in unconfined biota.

Agent type	Disease	Initial event	Primary taxa affected	Geographic location	Citation
Virus	Canine Distemper	1994	African Lion (*Panthera leo*)	Serengeti National Park, Tanzania	Roelke-Parker et al. 1996
	Canine Distemper	2000	Caspian Seal (*Phoca caspida*)	Caspian Sea, Russia; Kasakhstan	Kennedy et al. 2000; Stone 2000
	Rabies	1980s	Raccoon (*Procyon lotor*)	Eastern USA	Ruppecht and Smith 1994; Rupprect et al. 1995
	Iridovirus infection	Late 1970s	Amphibia (Multiple species of stream dwelling frogs)	Eastern Australia	Laurance et al. 1996
	Adenovirus infection	1993	Mule Deer (*Odocoileus hemonius*)	Central California, USA	Woods et al. 1996
Bacteria	Tuberculosis	1990s	African Buffalo (*Syncerus caffer*) Chacma Baboon (*Papio ursinus*) Felidae	Kruger National Park, South Africa	Bengis 2000
	Tuberculosis	1994	White-tailed Deer (*Odocoileus virginianus*)	Michigan, USA	Schmitt et al. 1997
	Tortoise chronic upper respiratory disease	1980s	Desert Tortoise (*Gopherus agassizii*)	California; Nevada; Utah; Arizona, USA	Jacobson et al. 1991
Fungi	Chytridiomycosis	1990s	Hylidae Multiple families Bufonidae	Australia Central America USA	Berger et al. 1998
	Coccidiomycosis	1992	Southern Sea Otter (*Enhydra lutris nereis*)	California, USA	Thomas et al. 1996
Protozoan parasite	Whirling disease	1990s	Rainbow Trout (*Onchorhynchus mykiss*)	Rocky Mountain States, USA	Potera 1997
Metazoan parasite	Bothriocephaliasis	1980s	Humpback Chub (*Gila cypha*)	Grand Canyon, Arizona, USA	Brouder and Hoffnagle 1977
Prion	Chronic wasting disease	1980s	Mule Deer (*Odocoileus hemionus*)		Williams and Young 1982; Spraker et al. 1997; Williams et al. 2000
Rickettsia	Rickettsiosis	1992	Atlantic Salmon (*Salmo salar*)	Canada; Norway; Ireland	Freyer et al. 1992; Rodger and Drinan 1993

TABLE 1. Continued.

Agent type	Disease	Initial event	Primary taxa affected	Geographic location	Citation
Multiple	Multiple diseases	1980s	Tropical coral reef ecosystem	Atlantic, Pacific, Indian Oceans; Caribbean Sea	Hayes and Gorcau 1990
	Multiple diseases	1990s	Southern Sea Otter (*Enhydra lutes nereis*)	California, USA	Thomas et al. 1996
Unknown	Fibropapillomatosis[a]	1980s	Cheloniidae Dermochelyidae (marine sea turtles)	Gulf of Mexico; Pacific, Western Atlantic and Indian Oceans; Carribean Sea	Balazas and Peolay 1991

[a] First recognized in the 1930s as an infrequent finding (Smith and Coates 1938); high prevalence became apparent during 1980s.

and there is a great deal of variability in the susceptibility for different species of waterfowl (Spieker et al. 1996). In contrast, West Nile virus has been isolated from more than 60 species of dead free-ranging birds and from an additional 20 species of wild birds that have died in zoological and other collections (Steele et al. 2000) since the initial North American appearance of West Nile fever in 1999 (Lanciotti et al. 1999). West Nile virus is also responsible for the deaths of several species of mammals, including humans (Center for Disease Control 1999). The magnitude of bird deaths associated with specific diseases is also highly variable, ranging along a spectrum from individual bird events from chronic diseases of attrition such as avian tuberculosis (*Mycobacterium avium*), to small numbers of deaths per event for diseases such as renal coccidiosis (*Eimeria truncata*), to large-scale epizootics such as those caused by West Nile virus and avian cholera (*Pasteurella multocida*) (Friend and Franson 1999). Avian botulism (*Clostridium botulinum* type C) and avian cholera currently stand out as major problems because of the magnitude of losses they cause, broad spectrum of species affected, annual frequency of epizootics, and their continually increasing geographic area of occurrence (Table 4) (Friend 1999c, Rocke and Friend 1999). However, it is the cumulative effects of the wide variety of emerging diseases that is of concern, rather than individual disease events or diseases.

Population effects.—The ability to determine and evaluate the effect of disease on the population dynamics of free-ranging avifauna is fraught with difficulties and confounded by a host of factors that complicate the determination of cause-and-effect relationships. As a result, evaluations are primarily at a gross scale and, in the absence of long-term studies on discrete populations or population cohorts, are generally associated with major changes in population numbers, rather than subtle changes such as long-term population depression, reproductive effects, or indirect mortality.

The current decline of vultures on the Indian subcontinent (Holden 2000) is an extreme example of the effects disease can have on avifauna. Over the past three to five years, populations of the Indian White-Backed (*Gyps bengalensis*) and the Indian Long-Billed (*G. indicus*) vultures over much of India have fallen to <5% of their

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TABLE 2. Examples of disease emergence in wild avifauna.

Agent type	Disease	Initial event	Primary taxa affected	Geographic location/current status	Citation
Virus	Duck plague (Duck virus enteritis)	1967	Anatidae	New York, USA; spreading since 1970s throughout USA and Canada.	Converse and Kidd 2001; Friend 1999b; Leibovitz and Hwang 1968;
	Newcastle disease	1990	Phalacrocoracidae	Saskatchewan, Canada; spreading since 1992 within USA and Canada.	Docherty and Friend 1999; Glaser et al. 1999; Meteyer et al. 1997; Wobeser et al. 1990
	West nile fever	1999	Corvidae	New York, USA; spreading	Steele 2000
	Avian pox[a]	1978	Accipitridae	Alaska, USA; spread throughout USA.	Hansen 1999; Windingstad et al. 1993
	Woodcock reovirus	1989	Scolopacidae	New Jersey, Virginia, USA; quiescent since second event.	Docherty et al. 1994
Bacteria	Avian cholera	1944	Anatidae	California, Texas, USA; spread across USA and into Canada since 1970s.	Friend 1999c
	Mycoplasmosis	1994	Fringillidae	District of Columbia, USA; spread throughout entire eastern range of House Finch.	Fischer et al. 1997
	Avian tuberculosis	1986	Gruidae	New Mexico, USA; sporadic cases in other avian species.	Snyder et al. 1991; Friend 1999c
	Salmonellosis	1980s	Fringillidae	Various, USA; increasing in USA and Canada.	Friend 1999e; National Wildlife Health Center 2001
	Avian botulism (type C in fish-eating birds)	1996	Pelecanidae	California, USA; annual occurrence at Salton Sea.	National Wildlife Health Center 2001
	Avian Botulism (type C, classical)	1890s[b]	Anatidae	California, Utah, USA; Nationwide expansion within USA since 1980s and increasing problem in Canada and Mexico.	Kalmback 1968; Rocke and Friend 1999

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TABLE 2. Continued.

Agent type	Disease	Initial event	Primary taxa affected	Geographic location/current status	Citation
Bacteria	Necrotic enteritis	1982	Anatidae	Wisconsin, Illinois, USA; events followed in Canada and other areas of USA.	Siegfried and Brand 1982; Wobeser and Rainnie 1987
Fungi	Mycotoxicosis	1982	Gruidae	Texas, USA; localized occasional occurrences.	Windingstad et al. 1989
Metazoan parasite	Trematodiasis	1996	Rallidae	Wisconsin, USA; localized annual occurrences.	Cole and Friend 1999

[a] Example is specific for avian pox in Bald Eagles; in general avian pox is occurring with increased frequency in several groups of birds including Anatidae and Fringillidae in addition to occurrences in Hawaii affecting Depanididae.

[b] Avian botulism first appeared as a major killer of waterbirds in the late 1890s to early 1900s; for the next 40 years that disease, with rare exception, was only reported to occur west of the Mississippi River (Kalmback 1968).

former abundance. Pathological findings strongly suggest the cause to be a disease of viral etiology (Rahmani and Prakash 2000). That disease has also reached Pakistan and Nepal, and may be the cause for the decline in vulture populations in other parts of Asia.

Common Eider populations in the Gulf of Finland are declining at a rate of 6 to 10% yearly (Hario 1998). Disease emergence is postulated to be the cause for the recent decline. In the late 1980s, duckling survival dropped to 1 to 5% in some areas and mortality events have killed large numbers of young and some adult eiders (Hollmén et al. 1999, 2000). A high prevalence of antibodies to IBDV has been found in the blood of eiders nesting within the Gulf of Finland. Those findings are of concern because IBDV causes substantial mortality in chickens (Hollmén et al. 2000). In addition, two viruses other than IBDV have been isolated from Gulf of Finland eiders. Investigations are ongoing relative to the role of those viruses in the eider decline (T. Hollmén pers. comm.).

Emerging diseases can have added significance when they appear in avian populations already in decline or when threatened and endangered species are affected because of low population numbers associated with those status categories. During 1996, a single event of type C avian botulism was responsible for the loss of an estimated 15 to 20% of the western population of White Pelicans (*Pelacanus erythrorhynchos*). This subpopulation has been in decline for several decades (D. Anderson pers. comm.). Substantial numbers of endangered California Brown Pelican (*Pelacanus occidentalis*) also died during that event. Despite the near global occurrence for type C avian botulism (Eklund and Dowell 1987), large-scale mortality of fish-eating birds from type C toxin is without precedent. The situation occurring at the Salton Sea in California appears to involve disease emergence in the form of an aberrant disease cycle for this environmental disease. Since 1996, type C avian botulism has been an annual cause of pelican mortality at the Salton Sea.

The Northern Pintail (*Anas acuta*) is another species whose population levels within North America are being affected by disease. In 1957, the estimated breeding population for that species exceeded 10 million, but then steadily declined to ~ 3.5 million by 1964, and after eventually increasing back to ~7 million in 1972, declined again to a low of slightly above 2 million in 1991 (Wilkins and Cooch 1999). Despite years of major efforts focused on restoring that species, the breeding population in 1999 was 30% below the long-term average and well below the long-term goal (Wilkins and Cooch 1999). The continued suppression of Northern Pintail populations is not surprising given the fact that the Northern Pintail is often the dominant species in major mortality events from

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TABLE 3. Forms of disease emergence in wild birds.

Event characterization	Description	Example
New disease	First appearance in wild birds within geographic area (region, nation).	House Finch conjunctivitis (Fischer et al. 1997; Hartup et al. 2001)
Geographic expansion	Movement of an enzootic disease of birds beyond the geographic boundaries of historic and common occurrence.	Avian cholera (Friend 1999c)
Reemergence[a]	Recurrence of a dormant disease of wild birds in similar or the same species and within the historic geographic boundaries for previous disease activity.	Velogenic Newcastle disease in Double-crested Cormorants (*Phalacrocorax auritus*) (Glaser et al. 1991)
Novel appearance	Occurrence of a disease of birds in species that it does not normally affect.	Type C avian botulism in pelicans (T. Rocke unpubl. data; National Wildlife Health Center 2001)

[a] Reemergence can lead to geographic expansion as has occurred for Newcastle disease.

avian botulism and avian cholera (Ball et al. 1998, Miller and Duncan 1999, National Wildlife Health Center 2001). Those events occur from their Canadian breeding grounds to wintering areas in the southern United States and Mexico. Heavy fall and spring losses also occur within the United States during some years. During 1997, an estimated 1.5 million water birds, primarily waterfowl (the majority of those birds being Northern Pintails) died from avian botulism during two sequential events. The first occurred in Canada and the other on the marshes in the vicinity of the Great Salt Lake in Utah (Ball et al. 1998).

The Whooping Crane (*Grus americana*) serves as our final example of disease effects on avian populations. A foster parenting project was undertaken to establish an additional migratory flock of Whooping Cranes in the western United States (Drewien and Bizean 1978). The flock peaked at 32 birds and then declined rapidly. Approximately 39% of the Whooping Cranes found dead from that flock died from, or were infected and would have died from, avian tuberculosis (Snyder et al. 1991). Typically, <1% of wild birds received for necropsy are diagnosed with avian tuberculosis (Smit et al. 1987, Converse and Dein 1991, Friend 1999d). Avian tuberculosis was clearly a major factor in the failure of the Whooping Crane foster parenting project.

Zoonoses.—It should also be recognized that wild birds are involved in the dissemination and transmission of a variety of diseases of humans (zoonoses) and domestic animals. Harris (1991) and Cooper (1990) list 24 and 22 zoonoses, respectively, involving wild birds. West Nile virus is the latest zoonosis involving birds to appear in the United States. Other zoonoses involving wild birds are also gaining prominence as disease issues. During recent years, salmonellosis (*Salmonella typhymerium*) has gained international importance as a major killer of birds at feeding stations (Kirkwood and MacGregor 1998, Friend 1999e). It is likely that this situation is an emerging example of problems that will continue to develop due to an increased dependency upon urban and suburban habitats and feeding stations as a result of the diminished base of natural habitat for many species of birds. The enhanced interface between humans and wild birds in the urban and suburban landscape presents increased opportunities for disease transfer to humans. This issue is a focus for growing concerns among some segments of society relative to urban populations of wild waterfowl, primarily Canada Geese (*Branta canadensis*) (Graczyk et al. 1998, Saltoun et al. 2000).

DISCUSSION

The importance of disease emergence for human society is of international scope and has stimulated the development of a wide variety of projects, collaborative efforts, scientific publi-

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TABLE 4. Examples of single event losses of wild birds due to avian botulism (*Clostridium botulinum* type C) and avian cholera (*Pasteurella multocida*).

Year	Geographic location	Primary taxa affected[a]	Losses[b]
Avian botulism			
1980	Utah, USA (Bear River Marshes, Box Elder Co.)	Anatidae	105,000[c]
1982	Caspian Sea, Russia (Guryev Region, Kazachstan)	Anatidae	1,000,000[d]
1995	Alberta, Canada (Pakowki Lake)	Anatidae	(>100,000)[e]
1996	Saskatchewan, Canada (Old Wives Lake)	Anatidae	134,000[e]
1997	Saskatechwan, Canada (Old Wives Lake)	Anatidae	1,000,000[e]
1997	Utah, USA (Bear River Marshes, Box Elder Co.)	Anatidae	500,000[e]
Avian cholera			
1970	Maryland, USA (Chesapeake Bay)	Anatidae	88,000[f,g]
1978	Maryland, USA (Chesapeake Bay)	Anatidae	(31,295)[h]
1980	Nebraska, USA (Rainwater Basins)	Anatidae	80,000[c,i]
1982	Nebraska, USA (Rainwater Basins)	Anatidae	32,800–36,300[j]
1995	Northwest Territories, Canada (Egg River Colony, Banks Island)	Anatidae	30,000[k]
1998	Utah, USA (Great Salt Lake, Salt Lake Co.)	Podicipedidae, Anatidae	50,000[c]

[a] Although Anatidae is the primary taxa affected by avian botulism, large numbers of shorebirds (*Scolopacidae*), American Coot (*Rallidae*) and Recorvirostridae (American Avocets, *Recurvirostra americana*, and Black-Necked Stilts, *Himantopus mexicanus*) also commonly die during avian botulism epizootics.

[b] Estimated losses and (carcasses retrieved = minimum losses).

[c] Data from U.S. Geological Survey, National Wildlife Health Center Epizootiology Database.

[d] Kuznetsov 1992.

[e] Ball et al. 1998.

[f] National Wildlife Federation 1970.

[g] Locke et al. 1970.

[h] Montgomery et al. 1980.

[i] Brand 1984.

[j] Hurt et al. 1983.

[k] Samuel et al. 1999.

cations, and reports and other actions focused on combating that problem on behalf of human health. The economic effects of emerging diseases such as BSE and FMD are enormous and demand aggressive efforts to combat the emergence of not only those diseases, but also other emerging diseases that threaten the products of agriculture needed to provide food and fiber for a continually expanding human population and global economy.

We have provided testimony of the biological effects for avifauna of emerging disease, a situation that also extends to other species and groups of free-ranging wildlife. Our testimony deals only with the tip of the iceberg rather than the full effect of disease emergence. The true cost from disease is associated with the chronic attrition that occurs from the broad spectrum of diseases present, rather than from the high-visibility events that generate media attention and transient crisis responses from the conservation community. In addition, the magnitude of losses from some disease events can be of sufficient severity to challenge the ability of already diminished avian populations to overcome those single-event losses.

If disease emergence is not aggressively addressed on behalf of avifauna, the resulting effects will extend beyond the biological to social and economic losses as well. Consider for example the potential effects of significant reductions of scavenger species such as vultures in India and crows in the United States on the removal of carcasses; revenue lost from ecotour-

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ism and sport hunting due to declines in bird populations; and the influence on cultural relations and social needs of native peoples.

The primary causes for the emergence of diseases affecting humans are generally agreed upon and provide a focus for corrective actions (Centers for Disease Control and Prevention 1994, Morse 1995, Wilson 1995, DaSilva and Laccarino 1999). Many of the same factors are involved for disease emergence in free-ranging wildlife. However, neither the avian nor greater conservation communities are responding to disease emergence in a manner consistent with the biological significance of that threat, or with the ecological understanding of how to approach that problem. Successful disease prevention and control for free-ranging wildlife requires that the approaches used are based on the fact that disease is an outcome, rather than a cause. Environmental conditions are often a basic cause associated with disease emergence, persistence, and spread, and must be addressed to successfully combat disease. Thus, two of the barriers inhibiting adequate response to disease emergence in avian and other wildlife species are (1) the persistence of perspectives that disease is not a significant factor relative to the population dynamics of wild species; and (2) a tendency to focus on the affected species or the causative organism rather than on the affected environment.

Aldo Leopold (1933) spoke to the issue of disease control in his classic treatise *Game Management* by stating that treatment of afflicted animals was a recessive approach. He further noted that ''. . . the real determinants of disease mortality are the environment and the population,'' both of which are being ''doctored daily, for better or for worse, by gun and axe, and by fire and plow.'' The wisdom of Leopold (1933) regarding disease has even greater relevance today. Continued landscape changes due to demands for human living space and other basic needs will continue to alter the geographic distribution and aggregation of avifauna in a manner likely to facilitate disease emergence and spread among wild birds. Reductions in the habitat base for avian species due to landscape changes challenges the ability to sustain population levels that can withstand major effects from disease and still provide the collective values human society seeks from wild birds.

Human behavior is additionally an important barrier inhibiting adequate response to disease emergence in wild birds. This factor is complex and is beyond the scope of this paper, other than to note that major differences afforded disease-control efforts for humans and domestic animals are personal ownership and economic influences. Wildlife in the United States and in many other countries is held in trust by government agencies with stewardship responsibilities for various species. Therefore, the public does not have the same incentives that have resulted in the development of major programs and industries to address disease in humans and domestic animals. The common ownership of wildlife by others (government) contributes to a detached perspective and approach to disease that is, in part, a variation on *The Tragedy of the Commons* (Hardin 1968) and, in part, *laissez-faire,* except for transient crisis responses.

We have provided a perspective towards disease that may challenge personal viewpoints of others involved in the conservation of wild birds. Our intent is to stimulate reexamination of the importance of disease on behalf of our avian resources. We strongly believe that due to landscape changes that have already occurred, and those certain to occur in the near future, a proactive, rather than a reactive, approach towards disease is required to discharge our stewardship obligations. Approximately 40 to 50% 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 100 years (Ayensu et al. 2000). Human-induced changes in biotic diversity and alterations in the structure and function of ecosystems are the two most dramatic ecological trends of the past century (Vitousek et al. 1997). The consequences of these changes are that society must now discharge a role of perpetual stewardship to maintain components and functions that once constituted the natural processes at the time Lack (1954, 1966) and others were making their judgments about the role of disease. Combating emerging disease has now become one of the adjustments that must be made to repair ecological integrity in a manner that sustains avian biodiversity and desired levels of avian populations.

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