WILDLIFE DISEASE AS AN INDICATOR FOR ECOSYSTEM HEALTH

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ABSTRACT

During recent years, a popular concept has emerged that ecosystem health is measurable in the context of human values and can be defined by certain indices. Wildlife mortality from disease agents, whether infectious or chemical, may be considered as one such indicator of ecosystem health. Some disease agents, such as microbes and parasites, are normal and even essential components of ecosystems. These agents may act to maintain normal ecosystem function by altering or balancing populations of animals. However, increasing losses from certain diseases, such as avian botulism and algal toxicoses, and catastrophic mortality events in wildlife may be an indication of major perturbations in environmental conditions or human-induced acceleration of natural ecosystem changes. We view some wildlife mortality events as a reflection of declining ecosystem health that may negatively impact wildlife conservation and biodiversity. The motivation to manage wildlife disease is a value judgment focused on reversing the affects of disease on some valued component of the ecosystem or to restore conditions to a desired level of ecosystem status. We contend that standards regarding specific diseases can be established as an important measurement for ecosystem health and management, such as their frequency of occurrence, the magnitude of losses, their spatial and temporal patterns of occurrence and, when possible, environmental conditions that promote outbreaks. Recent mortality events involving fish and birds at the Salton Sea of California and avian botulism in general are used to illustrate these concepts. The environmental conditions that facilitate avian botulism outbreaks are described as the type of standards that could be applied for guiding ecosystem management.

INTRODUCTION

Ecosystem health and ecosystem management are relatively new paradigms for environmental management (Costanza et al. 1992) and will continually be redefined and reevaluated relative to their purpose and utility in serving human society (Costanza et al. 1992; Grumbine 1994; Lackey 1998, 1999; Sutter 1993). The stimulus for ecosystem management is often associated with sustaining or restoring ecosystem health to provide values sought by humans (Costanza et al. 1992). We accept the concepts of ecosystem health and ecosystem management as useful precepts. However, we argue that the orientation in serving human values often has resulted in inadequate attention being given to ecosystem health for the direct benefit of nonhuman species. The need to address ecosystem health for the benefit of all species that share planet earth is noted by Merchant (1997) who stated that "…ecologically sound management is consistent with the continued health of both the human and the nonhuman communities." Also, it has been stated that:

> "The nation's biological resources are the basis of much of our current prosperity and an essential part of the wealth that we will pass on to future generations" (Pulliam 1995).

Despite the viewpoints just noted, disease occurrence in wildlife receives inadequate consideration with regard to perspectives of ecosystem health and as a focus for ecosystem management.

We use the Salton Sea in Southern California as a case study of an ecosystem where increasing disease occurrence in wild birds and fish is an obvious indicator of declining ecosystem health. In addition, we contend that for migratory birds, the concept of ecosystem health should be expanded from individual units to that of nested but disparate units that provide for species well-being during the entire annual migration cycle. We also describe recent findings on wetland characteristics associated with avian botulism in water birds to illustrate the point that environmental indices can be used to assess and perhaps reduce the risk of disease in specific ecosystems. Our viewpoints have their foundation in our personal opinions of what constitutes ecosystem health and are influenced by our experiences as "practitioners" in dealing with disease in free-ranging wildlife populations.

DISEASE AS AN OUTCOME

Traditionally, population and evolutionary biologists have generally viewed infectious disease to be agents of natural selection in higher organisms (Levin et al. 1999) and, in some cases, disease agents may act in this capacity. However, this concept of disease as solely a natural event fails to recognize that disease is an outcome rather than a cause (Friend 1995)—a product of interactions between a host population, one or more agents, and the environment (Fig. 1). Because environmental conditions play a major role in the outcome of host-agent interactions and can influence the outcome for better or worse (from the perspective of the host population), it is reasonable to consider disease occurrence and ecology as parameters for evaluating ecosystem health, regardless of the etiologic agent involved.

Human health has benefited greatly from application of this concept. Environmental standards for sanitation are testimony to the recognized relation between environmental quality and disease outcomes. "The ancient belief that war, famine, and pestilence ride together was a manifestation of awareness that the general standard of living, and the state of nutrition in particular, profoundly affect the resistance of man to his potential pathogens. ...history provides many examples illustrating the relation of wars and social upheavals to the spread and severity of epidemics...." (Dubos 1958). It has also been noted for many years that "…low standards of personal and public hygiene furnish the conditions under which most of the tropical diseases thrive" (Van Cleave 1945). These lessons are continually relearned as a result of infrastructure destabalization and breakdown due to war, political turmoil, and catastrophic natural disasters (Kohn 1995; Maurice 1995).

Human-induced changes in environmental conditions are the major reason that environmental standards are needed to protect human health. Nevertheless, the relation between disease occurrence and environmental conditions has largely been ignored in the application of ecosystem health concepts to nonhuman species. An exception is the common focus on chemical contaminants. Thus it is noteworthy that the first annual meeting of the Society for Conservation Biology included a symposium titled, "Conservation and Disease". During the meeting, it was stated that:

> "Given the conspicuous role that diseases have played, and in many parts of the world continue to play, in human demography, it is surprising that ecologists have given so little attention to the way diseases may affect the distribution and abundance of other animals and plants. Until recently, for example, ecology textbooks had chapters discussing how vertebrate and invertebrate predators may influence prey abundance, but in most cases you will search the index in vain for mention of infectious diseases" (May 1988).

We contend that the conservation (sustainability) of wildlife is a valued function and product of ecosystems. Therefore, we view some wildlife mortality situations as a reflection of declining ecosystem health that may negatively impact wildlife conservation, biodiversity, and human benefits derived from those ecosystems. However, it is not the mere presence of disease in a population, even an event causing catastrophic losses that necessarily indicates poor ecosystem health. Rather, evaluations should be based on changes in the frequency of disease occurrence, the etiologic agents involved, and the magnitude of losses relative to the capacity of an ecosystem to sustain its wildlife species and populations of value to human society. Further, we do not advocate an absence of disease as necessarily being a desirable aspect of ecosystems, especially for diseases caused by endemic infectious agents. Species naivety to some infectious diseases can result in enhanced vulnerability to the extent that the ecosystem no longer retains a positive habitat value for species sustainability. Also, some host-parasite interactions may truly function to regulate populations (Dobson 1988; Hudson 1986) as hypothesized for other causes of mortality, such as predator-prey interactions (Crawley 1992; Greenwood and Sovada 1996). Depending on the functional relations of the affected species within an ecosystem, the disease impacts (or lack of disease) may result in broader ecosystem perturbations.

We have emphasized two key points: 1) the outcome of disease has a strong linkage to environmental quality; and 2) disease impacts on wildlife populations damage values of human society. The first point supports disease evaluations as an index for ecosystem health. The second point supports the need to consider sustainability of wildlife populations at levels consistent with human values (consumptive and non-consumptive uses) when evaluating ecosystem health and considering management of ecosystems.

AVIAN DISEASE AND THE SALTON SEA

The Salton Sea, located in the Imperial Valley of southern California, is a very unique ecosystem, and a brief review of its history will provide the necessary context for our discussion.

This largest inland water body in California is of recent origin. "As recently as the year 1900, the Imperial Valley had not a single civilized inhabitant, and not one of its hot, arid acres had ever been cultivated" (Kennan 1917). Irrigation was brought to the Imperial Valley in 1901 and was quickly followed by settlers who cultivated the land. Flooding by the Colorado River resulted in a breach of an irrigation structure in March 1905 and flow into the Salton Trough. Unprecedented multiple flood events resulted in the continuing flood flows causing further diversion, eventually resulting in the entire flow of the River discharging into the Salton Trough. Following Herculean efforts to close the breach, the Colorado River was finally returned to its riverbed on February 10, 1907 (Kennan 1917). Left behind was a freshwater lake that reached the level of 195 feet below sea level, 76 feet above the pre-flood level. By 1925, evaporation that exceeded inflows resulted in the Sea subsiding to 250 feet below sea level (Laflin 1995). Augmented primarily by increased irrigation, plus wastewater from industry and sanitation that flows into the drains, the Sea is currently stabilized at 227 feet below sea level. Evaporation and inflows have equilibrated at about 1.363 million acre feet[1], resulting in a current Sea that is 35 miles long, 9 to 15 miles wide, with a surface area of 380 square miles, and a volume of approximately 7.3 million acre-feet. The Sea has a maximum depth of 51 feet and an average depth of approximately 31 feet (Cohen et al. 1999).

Since the Sea was first formed, salinity has risen to approximately 44 parts per thousand due to precipitation of salts left behind following evaporation of inflow water. This level of salinity exceeds ocean water by 26 percent and is increasing at a rate of approximately 0.5 percent per year due to an annual input of approximately 4 million tons of dissolved salts[1] (Fig. 2). In addition, nutrient loading from the inflows has created a highly eutrophic water body.

Approximately 15,000 tons of nutrients such as nitrogen and phosphorus annually flow into the Sea (Cohen et al. 1999). Despite the hypersaline, eutrophic conditions, the Salton Sea supports the most productive sportfishery in California inland water bodies (Black 1985) and is habitat for an extraordinary number of bird species. With more than 400 species of birds reported at the Sea and surrounding environs[2], the avian biodiversity of this ecosystem is greater than nearly all other wetlands in the contiguous United States.

Avian disease is not new to the Salton Sea. Correspondence of the Bureau of Biological Survey in the National Archives indicates major die-offs of waterfowl and other water birds as early as the mid-1920's[3]. The focal points for these events were the deltas of the New and Alamo Rivers at the southern end of the Sea. Initial descriptions of sick birds and field signs strongly suggest type C avian botulism was the causative agent, a disease that was diagnosed as the cause of major mortality events at the Sea during 1933 and 1934 (Kalmbach 1938). A review of the Salton Sea National Wildlife Refuge (now the Sonny Bono National Wildlife Refuge) narrative reports from 1939 to the present (no reports could be located for the period from 1930 (year of establishment) to 1939, nor during the war years from 1942 through 1946) disclosed periodic occurrences of avian botulism and little else until the 1980's.

The U.S. Fish and Wildlife Service's National Wildlife Health Center (NWHC) diagnosed avian cholera as the cause of a small die-off during February 1983. A total of 170 carcasses were collected. The following year, 312 carcasses were collected during an October die-off that the California Department of Fish and Game, Wildlife Disease Investigations Laboratory diagnosed as avian botulism. Salton Sea National Wildlife Refuge records indicated an estimated loss of 500 birds from avian cholera in 1987. The following year, an estimated

2,000 birds died from avian botulism and an additional 600 from avian cholera. Three different diseases were reported during 1989; 215 carcasses were collected as a result of avian botulism, approximately 50 from avian cholera, and more than 4,500 birds were found dead from salmonellosis at a wetland near the Sea.

The 1990's stand in marked contrast to the losses reported during most of the previous decade. Disease outbreaks have been reported at the Salton Sea during every year of the 1990's, and the frequency of events, magnitude of losses and variety of causes is unprecedented for any previous decade (Tables 1 and 2). The most noteworthy of these mortality events is the estimated loss of 155,000 eared grebes (*Podiceps nigricollis*) during 1992—approximately 7 percent of the North American population of this species. A similar number may have perished during a 1989 die-off that was not investigated (Jehl 1996). The causative agent for most of these deaths remains unknown, although algal toxins are suspected of playing a role in those die-offs and in losses of thousands of grebes since then. During 1996, more than 15,000 water birds died from avian botulism. Approximately 9,000 of these were white pelicans (*Pelecanus erythrorhynchos*), a loss that was estimated at between 10-15 percent of the western population of this species[4], and more than 1,200 were endangered California brown pelicans (*P. occidentalis californicus*), making this the largest recorded loss from disease of an endangered species. Virtually the entire production of nestling double-crested cormorants died from Newcastle disease during 1997. A similar event occurred the following year killing an estimated 6,000 birds and, although virus was not isolated from dead birds, the clinical and field signs were strongly suggestive of Newcastle disease being the cause of mortality.

Frequent large-scale fish kills that range in size from tens of thousands to a million or more fish have also occurred during the 1990's, some of which have also been linked to parasites and other microbial pathogens, such as *Vibrio* spp[5], *Amyloodinium* infestations (Kuperman and Matey 1999) and algal toxicoses[6]. An estimated 7.6 million fish succumbed during the fish kill during the first week of August 1999[7]. Also, the massive fish kills have been linked to disease mortality in birds, at least for the occurrence of botulism outbreaks in fish-eating birds[8]. We believe this drastic increase in mortality in fish and birds at the Salton Sea is reflective of an ecosystem under severe stress (Rapport and Whitford 1999). Certainly, the magnitude of bird losses during this decade can only be viewed as a decline in the functional ability of this ecosystem to provide for the sustainability of some of its species. Studies have been initiated to define the factors contributing to those disease events and provide a foundation for ecosystem management to restore the health of the Sea.

ECOSYSTEM HEALTH AND MIGRATORY BIRDS

We have used the Salton Sea as an example where changes in wildlife disease patterns reflect declining ecosystem health and have noted the challenges imposed by those diseases for the species being affected. Another important consideration for migratory species is that their sustainability is dependent upon more than one geographic location. Therefore, since the scale of an ecosystem is determined by the management problem at hand (Lackey 1999), an area much greater than the Salton Sea must be considered, including all of the geographic areas that provide for critical stages in the species annual migratory cycle. Since these geographic areas are not contiguous, nested ecological units at disparate locations become the functional management units (ecosystems) for the various species involved. The functional processes and products to be provided from each ecosystem are related to the life stage needs by those species. The collective contributions from each of these disparate ecosystems provides for species sustainability.

ENVIRONMENTAL INDICES FOR EVALUATING DISEASE RISK IN ECOSYSTEMS

Because environmental conditions play such a key role in defining the outcome of host-disease agent interactions, in some cases environmental indices other than disease mortality or morbidity can be used to assess the potential risk of disease outbreaks in specific ecosystems. To illustrate this concept, we consider recent findings regarding wetland characteristics associated with type C avian botulism outbreaks in interior wetlands (Rocke and Samuel 1999). Avian botulism, a paralytic disease caused by ingestion of a neurotoxin, often kills thousands of water birds in a single outbreak, with annual continental losses in North America approaching the hundreds of thousands to millions (Rocke and Friend 1999). In recent years (1994-97), outbreaks of avian botulism killed > 4 million waterfowl in Canada and the United States[9], focusing renewed attention on the potential effects of this disease in North American waterfowl populations. This is particularly true for species like the northern pintail (*Anas acuta*) which has accounted for a large portion of the recorded botulism mortality (15-20 percent) and has failed to show increased population levels despite generally favorable breeding conditions[10].

The disease is caused by a neurotoxin produced by *Clostridium botulinum* type C, an anaerobic bacterium that forms dormant spores in aerobic or other environmental conditions adverse to the bacterium (Smith and Sugiyama 1988). These spores are highly resilient (Hofer and Davis 1972) and are widely distributed in wetland sediments (Smith and Sugiyama 1988). In addition, spores are also highly prevalent in the tissues of aquatic invertebrates (Jensen and Allen

1960) and vertebrates (Reed and Rocke 1992). However, despite the ubiquitous distribution of botulinum spores, outbreaks of avian botulism are unpredictable, often occurring annually in certain wetlands but not in adjacent ones. In one study, conducted on a northern California wetland, no relationship was found between botulinum cell and spore density and the occurrence of botulism outbreaks (Sandler et al. 1993), suggesting that density of the agent is probably not a limiting factor in the occurrence of outbreaks. Instead, results of studies conducted in wetlands with ongoing botulism outbreaks and paired control wetlands throughout the United States suggested that the relative risk of a botulism outbreak was predicted by complex relations among redox potential, pH, and temperature measured at the sediment-water interface (Rocke and Samuel 1999).

To illustrate these findings, a series of predictive models were developed that relate environmental conditions to the relative risk of botulism outbreaks in wetlands (Fig 3). A similar risk assessment for avian botulism is currently being conducted for both fresh and brackish wetlands associated with the Salton Sea. These risk models can potentially provide a new method to identify wetlands with high risk of botulism outbreaks, to develop and evaluate alternative strategies for reducing the risk of botulism in high risk areas, and to evaluate the effect of current wetland management practices on the risk of botulism outbreaks. A substantial advantage provided by these models is that evaluation of different management strategies could be based on the predicted relative risk of an outbreak and is not dependent on the occurrence of avian mortality from botulism. With additional research, these risk models also may be useful in evaluating suitability of specific wetland acquisition and management actions in providing for the conservation of wetland-dependent avifauna.

DISCUSSION

Ecosystem health represents a desired endpoint of environmental management, and the use of this concept requires an adequate operational definition of the desired endpoint (Costanza 1992). The key point in this statement is the connectivity between ecosystem health as a concept and the application of the concept for a specific purpose. Without this connectivity, considerations of ecosystem health are largely intellectual exercises. A recent definition of ecosystem management is: "the application of ecological and social information, options, and constraints to achieve desired social benefits within a defined geographic area and over a specified period" (Lackey 1998). The goals for the Salton Sea ecosystem identified in the Salton Sea Restoration Project[11] satisfy both this definition of ecosystem management and the desired ecosystem health endpoints called for by Costanza (1992).

Despite this, restoration of the Salton Sea is a subject of considerable controversy (Kaiser 1999; Cohen et al. 1999). The fundamental reason for this controversy is ideology rather than science. It has been noted that the most divisive issues of ecosystem management "…are not scientific; they are most often clashes over moral and philosophical positions or different individual preferences". As a result, "…ecosystem management has become a lightning rod for controversy in public policy" (Lackey 1999).

Our focus in this presentation is not the issue of restoration of the Salton Sea, but the application of ecosystem health and ecosystem management for the direct benefit of wildlife resources rather than humans. We contend that our limited perspective endpoint has index values that extend to human interests in ecosystem health and that greater attention to the health of free-ranging wildlife will provide substantial benefits for wildlife and humans alike. This philosophy goes beyond the common perspective of wildlife serving as the canary-in-the coal mine" as an index to impending doom. We argue that wildlife disease occurrence, in addition to being a component of an environmental "distress syndrome" for human values (Rapport and Whitford 1999) has important considerations for wildlife conservation (sustainability) and species biodiversity.

The "canary in the coal mine" concept is valid for protecting human health and has received various levels of attention over time, particularly in association with monitoring zoonoses (Korch et al. 1989; Reisen et al. 1997) and chemical contamination of the environment (Beyer et al. 1996; University of Connecticut 1979). However, the complexity of interacting factors that results in environmental impacts from contaminants requires more than measuring the presence of chemical residues in the environment and in animals (Anderson 1998). Also, detecting antibody for infectious diseases in animals, or virus in insect vectors, does not directly translate to cases of human disease. Clinical cases of disease in animals are generally of greater importance as an index for human health risks.

The recent resurgence of infectious disease within human populations and concerns related to changing patterns of disease associated with predicted global climate change (Colwell 1996; McMichael et al. 1996; Patz et al. 1996) have also created renewed interest in wildlife disease as indices for human health (Epstein et al. 1998). However, disease emergence during recent years among free-ranging wildlife populations (Friend 1995, 1999; Murphy 1994) has not been seriously considered as an index of ecosystem health for wildlife. In general, response to wildlife disease events has been typical of the response to other catastrophic events, focusing on clean-up and documentation of the event rather than stimulating a collective focus on the environmental conditions that are influencing disease outcomes.

A final point for discussion is the assertion that "natural and undisturbed is inherently preferable to altered and disturbed" (Lackey 1999). A corollary of altered and disturbed ecosystems is that exotic species are often prominent components of such systems. In his courageous comments, Lackey (1999) noted that preferences for natural and undisturbed ecosystems to altered and disturbed ecosystems are purely societal judgments and concluded that, "There is nothing inherent in science that makes either pristine or altered ecosystems inherently preferable from a policy standpoint". This issue has been a component of the controversy associated with the Salton Sea. The sustainability and enhancement of values provided by this "altered ecosystem" are the basis for local support for enhanced ecosystem health for introduced and indigenous species within the Sea. Others have argued that society would be better served by a Sea with an environment more like the Great Salt Lake and Mono Lake (Cohen et al. 1999).

Acceptance that native species are more important than exotic species is a matter of human values that tend to shift if the species is a valued intentional introduction or has other origin and status (Lackey 1999). An undeniable fact is that natural resource agencies expend considerable resources each year in the conservation of nonnative wildlife species such as the ring-necked pheasant (*Phasianus colchicus*) and brown trout (*Salmo trutta*) and in attempting to establish species in geographic areas where they are not native. These efforts are motivated and supported by segments of human society that place value in the benefits they derive from those species. Those segments of society do not necessarily view species replacements or reductions in biodiversity to be indicators of changes in ecosystem health, a standard for evaluation by some other segments of society (Coleman 1993; Ehrlich and Daily 1993).

An additional potential outcome from exotics is interspecies association that may create new disease problems or enhance existing ones. The entry of humans into new habitats and early global movements of humans is a form of exotic invasion that has had major ramifications regarding disease in humans (Levins et al. 1994; McNeill 1976, 1993; Verano and Ubelaker 1992). The unusual occurrence of type C avian botulism in fish-eating birds at the Salton Sea may also have a strong association with exotics, specifically tilapia. The physiology of this fish species provides unique environmental conditions within the gut that may play an important role in the epizootiology of avian botulism at the Salton Sea.

CONCLUSIONS

The contemporary paradigms of ecosystem health and management are conceptually debated and perused on the basis of perspectives that address values for human society. Both are useful but arbitrary concepts in the manner in which they are considered and applied. We offer perspectives that expand the considerations and application of these concepts to evaluations of wildlife health as an endpoint in itself; expand the concepts from evaluation and management of a single ecosystem to the need for considering nested, but disparate ecosystems when addressing the sustainability of migratory wildlife populations; and suggest that the occurrence of some diseases and/or environmental indices for the risk of disease can be used for evaluating ecosystem health. We challenge the notion that all disease events are simply indicators of ecosystem distress or natural regulators within ecosystems. We consider that disease is an outcome rather than a cause and note two important associations. First and foremost, environmental conditions are a primary factor underlying disease occurrence. Second, ecosystems in which disease occurrence is of sufficient prevalence to be of concern are rarely "natural systems". These systems are modified /altered by human actions or are human-created systems. Human interventions that created the alterations also often alter the relations within the disease triad of host, agent, and environment. Disease occurrence within these altered environments tend to have more lasting negative biological impacts and can seriously challenge the ability of the ecosystem to provide an environment that supports the wildlife values sought by human society.

Unfortunately, much of the world's wildlife habitat has undergone significant human-induced alterations and a growing amount is human-created habitat. This trend will continue for the foreseeable future due to landscape changes and other actions associated with the continual growth and changing demography of human populations. Greater attention needs to be given to defining ecosystem health and the application of ecosystem management to these altered and created systems as habitat for the sustainability of wildlife populations and biodiversity. In many ways these "ecosystems" are different than "natural ecosystems" and need to be evaluated and managed in their own image and purpose, not against standards established for natural systems. In both instances, human values are the motivation for outcomes to be derived.

If contemporary society is to serve humankind and the other species of our world we must view natural, altered, and human created ecosystems all to be important components of the mosaic that is our environment and not pit one against another as superior and inferior components. To do less would express arrogance regarding our level of understanding of ecosystem health and our ability to manage it. To place this statement in context for wildlife and ecosystems, we quote the following passages by Aldo Leopold.

> For wildlife, "....doctoring is of recessive importance in health control...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" (Leopold 1933).
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> For ecosystems, "The effort to control the health of the land has not been very successful. ...In general, the trend of the evidence indicates that in the land, just as in the human body, the symptom may be in one organ and the cause in another. The practices we now call conservation are, to a large extent, local alleviations to biotic pain. They are necessary, but they must not be confused with cures. The art of land-doctoring is being practiced with vigor, but the science of land-health is a job for the future." (Leopold 1941).

The underlying thesis for Leopold's land-health evaluation is society's failure to understand and respond to cause rather than symptom. We close by asking the question—how far have we come relative to Leopold's statements of more than a half-century ago regarding our understanding and ability to address wildlife and ecosystem health? Our answer is—not nearly far enough, the science of land-health still remains a challenge for the future.

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FOOTNOTES

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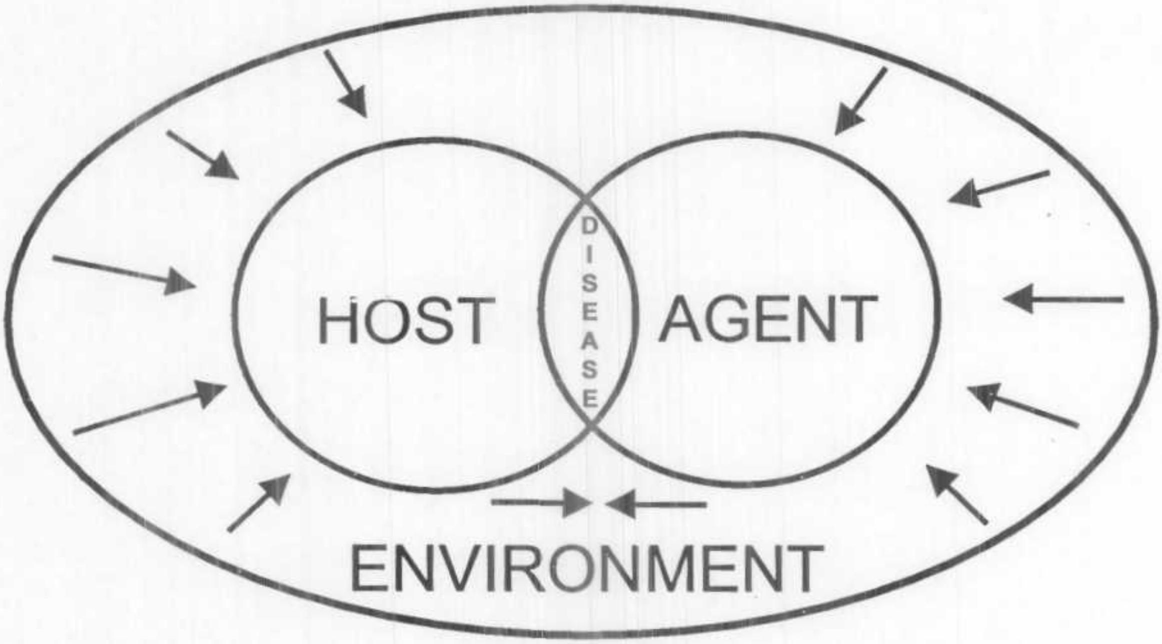
FIGURE LEGENDS

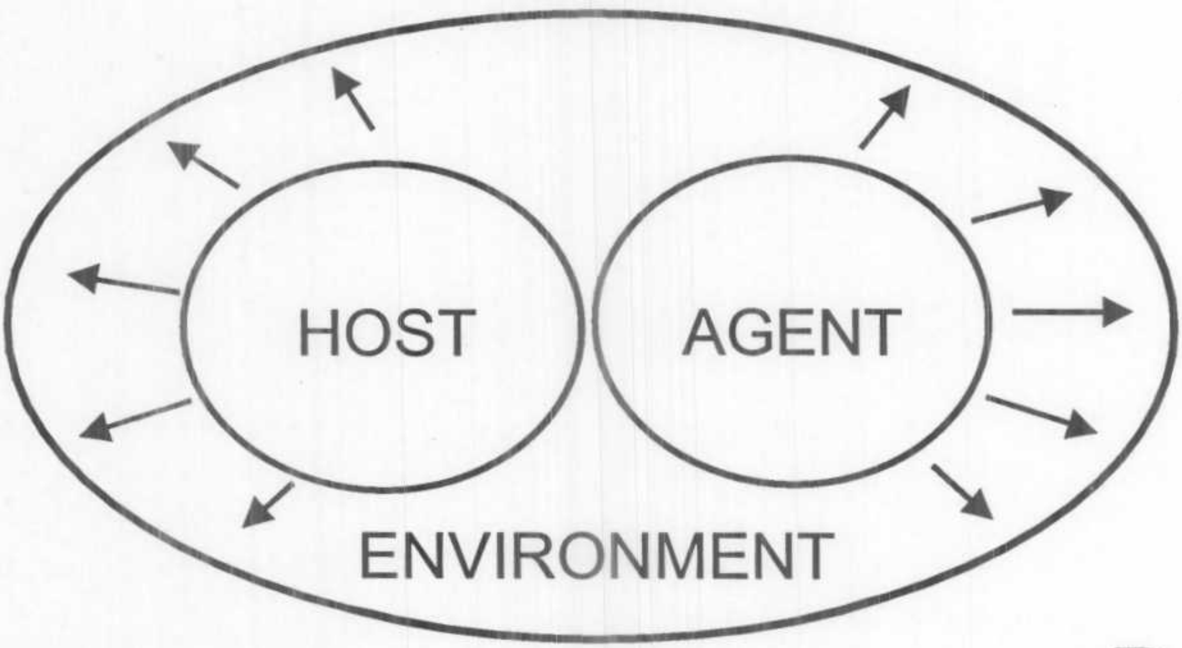
Figure 1. A. Disease may be generically thought of as an outcome of interactions (area of overlap) between susceptible hosts, agents capable of causing disease and environmental factors. In many instances, environmental conditions are the dominant factor.
B. Understanding and managing the environmental component of this triad in a manner that minimizes the potential for major disease outbreaks is a basic ecosystem management need for the conservation of wildlife populations.

Figure 2. Projected increases over time in the level of Salton Sea salinity based on continuation of the current inflow of 1.363 million acre feet of water. The annual rate of increase is approximately 0.5 percent. Salinity graph provided by PA Weghorst, Principal Hydrologic Engineer, Salton Sea Reclamation Project, Bureau of Reclamation.

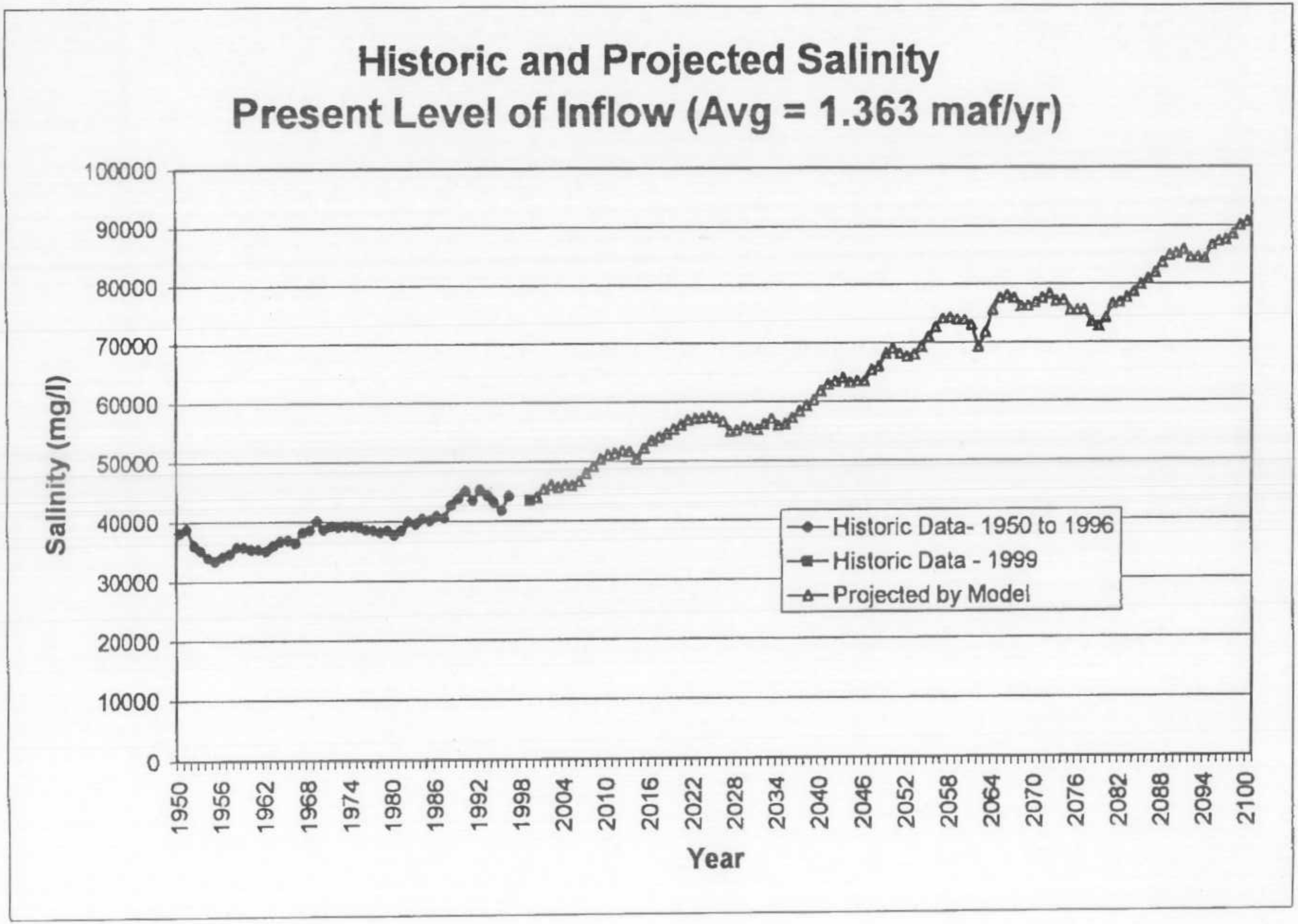
Figure 3. Predictive models showing the relationship between water pH and temperature and the relative risk of botulism outbreaks in United States wetlands with water redox potentials of A) –200 mv, B) + 100 MV, and C) +300 MV. We predicted relative risk of botulism outbreaks using average water values (pH = 8.17, redox potential = -65.38 mv, standardized redox potential = 2.61 mv) for outbreak wetlands as the baseline. Reprinted from the Journal of Wildlife Management (Rocke and Samuel 1999).



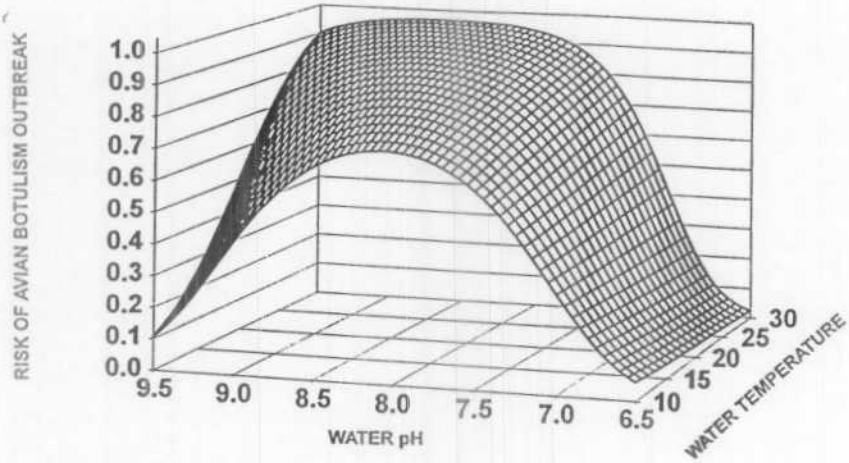
A



B

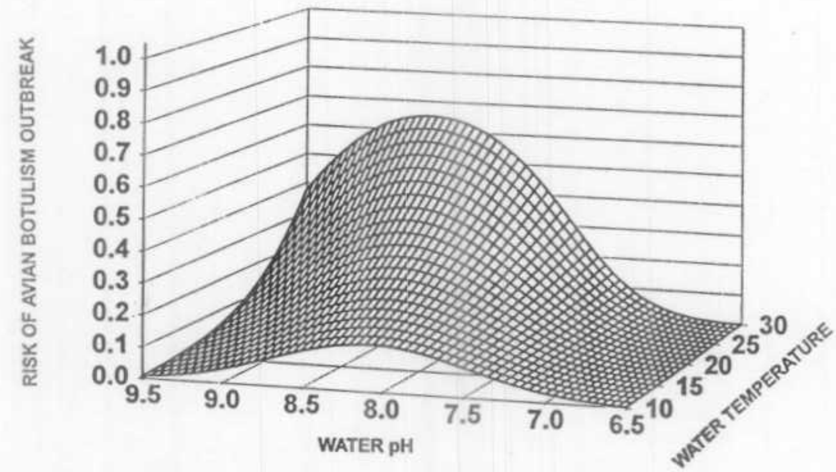

Historic and Projected Salinity
Present Level of Inflow (Avg = 1.363 maf/yr)
Salinity (mg/l)
100000
90000
80000
70000
60000
50000
40000
30000
20000
10000
0
1950
1956
1962
1968
1974
1980
1986
1992
1998
2004
2010
2016
2022
2028
2034
2040
2046
2052
2058
2064
2070
2076
2082
2088
2094
2100
Year
Historic Data- 1950 to 1996
Historic Data - 1999
Projected by Model

A



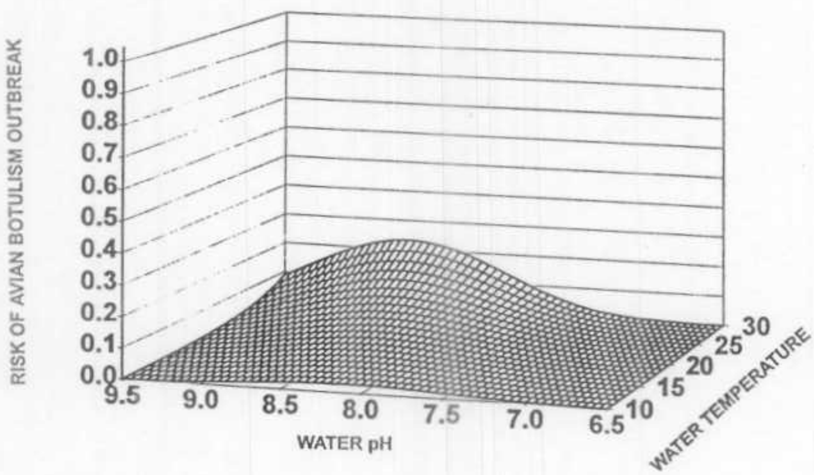
RISK OF AVIAN BOTULISM OUTBREAK
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
9.5
9.0
8.5
8.0
7.5
7.0
6.5
WATER pH
10
15
20
25
30
WATER TEMPERATURE

B



RISK OF AVIAN BOTULISM OUTBREAK
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
9.5
9.0
8.5
8.0
7.5
7.0
6.5
WATER pH
10
15
20
25
30
WATER TEMPERATURE

C



RISK OF AVIAN BOTULISM OUTBREAK
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
9.5
9.0
8.5
8.0
7.5
7.0
6.5
WATER pH
10
15
20
25
30
WATER TEMPERATURE

Table 1: Reported avian disease events at the Salton Sea and nearby wetlands

Time Period	Years with Total Losses Reported to be:					Data Source/Comments
	1-100	101-500	501-1,000	1,001-2,000	2,000+	
1925[2]-1935					Annual for entire period except 1932	Homes (1933)[1]; Kalmbach (1938)
1936-1945[3]	1940				1939	Salton Sea National Wildlife Refuge narrative reports (SSNWR)
1946-1955[4]	1949, 1953, 1954	1947, 1955	1951		1952	SSNWR; 4,000 to 5,000 waterfowl loss in1952.
1956-1965	1956-1959	1965	1964		1962, 1963	SSNWR; bird losses of approximately 10,000 in 1963 and 7,000 in 1962.
1966-1975	1966-1969 1973	1971	1970, 1975	1974	1972	SSNWR; 1972 losses of about 5,500.
1976-1985[5]	1978	1983, 1984			1979	SSNWR; 10,000 bird loss in 1979; National Wildlife Health Center (NWHC)
1986-1995	1990	1987, 1993, 1995			1988-1992, 1994	SSNWR; NWHC; more than 150,000 grebes died during 1992; more than 20,000 birds during 1994.
1996-present[6]					1996-1999	SSNWR; NWHC; more than 20,000 birds, including approximately 10,000 pelicans, died during 1996; more than 10,000 birds during 1997; and approximately 20,000 birds during 1998.

[1] SW Holmes. 1933. Letter to HP Sheldon, Bureau of Biological Survey, Washington, D.C., November 17, 1933: National Archives.

[2] First year information is available (by extrapolation from Holmes (1933)[1].

[3] Information only available for 1939, 1940, 1941.

[4] No data for 1946 or 1950.

[5] No SSNWR data for 1977, 1978, 1981, 1982.

[6] July 31, 1999.

Table 2. Causes of avian disease reported for the Salton Sea and nearby wetlands.[1]

Time Period	Cause of Avian Mortality								Data Source/Comments
	Avian botulism	Avian cholera	Newcastle disease	Salmonellosis	Algal toxins	Lead poisoning	Pesticides	Unknown	
1925[4,5]-1935	Annual except 1932					1933			Holmes (1933)[2]; Kalmbach (1938); It is highly likely that all of the major events were due to avian botulism; lead poisoning was the probable cause of sickness for a small number of ducks commented on (Tonkin 1933)[3].
1936-1945[6]	1940, 1939								Salton Sea National Wildlife Refuge narrative reports (SSNWR).
1946-1955[7]	1947, 1949, 1951-1955				1955[8]			1955	SSNWR.
1956-1965	1956-1959, 1962-1965					1956	1962[8], 1963[8], 1964[10]	1956	SSNWR.
1966-1975	Annual				1975[8]		1974[10]		SSNWR.
1976-1985[9]	1979, 1984, 1980	1979, 1983					1978[10]		SSNWR; National Wildlife Health Center (NWHC); 1979 NWHC confirmed first cases of avian cholera in Imperial Valley.
1986-1995	1988, 1989, 1991, 1994, 1995	1987-1989, 1990-1994	1997, 1998[6]	1989, 1991, 1995	1989[8], 1992[8], 1994[8], 1995[8]	1989, 1991	1986[10], 1989[10], 1995[10]	1992	SSNWR; NWHC; 1997 Newcastle disease was first outbreak in wild birds in the United States west of the Rocky Mountains (NWHC); aspergillosis 1994, 1995.
1996-Present[11]	1996, 1997, 1999	1996, 1998		1996	1996[8], 1997[8]				SSNWR; NWHC.

1 Diagnosis of causes of mortalities prior to 1980 are based on laboratory analysis in some instances and evaluations of field and clinical signs in others. Where there are major questions regarding cause we have considered the event to be of unknown cause.

2 SW Holmes. 1933. Letter to HP Sheldon, Bureau of Biological Survey, Washington, D.C., November 17, 1933: National Archives.

3 G Tonkin. 1933. Letter to Chief, Biological Survey, Washington, D.C., November 26, 1933: National Archives.

4 First year data are available (based on extrapolation from Holmes 1933)[2].

5 No laboratory evaluations prior to 1933.

6 Information only available for 1939, 1940, 1941.

7 No data for 1946 or 1950.

8 Reported as suspected cause of death; no laboratory diagnosis.

9 No SSNWR data for 1977, 1978, 1981, 1982.

10 Die-offs on agricultural fields or in agriculture drains rather than the Sea.

11 July 31, 1999.