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Avian disease at the Salton Sea

Milton Friend

Salton Sea Science Office, La Quinta, CA 92253, U.S.A.

Key words: Avian disease, Salton Sea, avian botulism, avian cholera, salmonellosis, Newcastle disease, waterfowl, shorebirds, pelicans, cormorants, grebes, *Clostridium botulinum, Pasteurella multocidia, Salmonella typhimurium*, Sonny Bono National Wildlife Refuge, pesticides

Abstract

A review of existing records and the scientific literature was conducted for occurrences of avian diseases affecting free-ranging avifauna within the Salton Sea ecosystem. The period for evaluation was 1907 through 1999. Records of the U.S. Department of Agriculture, Bureau of Biological Survey and the scientific literature were the data sources for the period of 1907–1939. The narrative reports of the U.S. Fish and Wildlife Service's Sonny Bono National Wildlife Refuge Complex and the epizootic database of the U.S. Geological Survey's National Wildlife Health Center were the primary data sources for the remainder of the evaluation. The pattern of avian disease at the Salton Sea has changed greatly over time. Relative to past decades, there was a greater frequency of major outbreaks of avian disease at the Salton Sea during the 1990s than in previous decades, a greater variety of disease agents causing epizootics, and apparent chronic increases in the attrition of birds from disease. Avian mortality was high for about a decade beginning during the mid-1920s, diminished substantially by the 1940s and was at low to moderate levels until the 1990s when it reached the highest levels reported. Avian botulism (Clostridium botulinum type C) was the only major cause of avian disease until 1979 when the first major epizootic of avian cholera (Pasteurella multocidia) was documented. Waterfowl and shorebirds were the primary species affected by avian botulism. A broader spectrum of species have been killed by avian cholera but waterfowl have suffered the greatest losses. Avian cholera reappeared in 1983 and has joined avian botulism as a recurring cause of avian mortality. In 1989, avian salmonellosis (Salmonella typhimurium) was first diagnosed as a major cause of avian disease within the Salton Sea ecosystem and has since reappeared several times, primarily among cattle egrets (Bubulcus ibis). The largest loss from a single epizootic occurred in 1992, when an estimated 155 000 birds, primarily eared grebes (Podiceps nigricollis), died from an undiagnosed cause. Reoccurrences of that unknown malady have continued to kill substantial numbers of eared grebes throughout the 1990s. The first major epizootic of type C avian botulism in fish-eating birds occurred in 1996 and killed large numbers of pelicans (Pelecanus occidentalis & P. erythrorhynchos). Avian botulism has remained as a major annual cause of disease in pelicans. In contrast, the chronic on-Sea occurrence of avian botulism in waterfowl and shorebirds of previous decades was seldom seen during the 1990s. Newcastle disease became the first viral disease to cause major bird losses at the Salton Sea when it appeared in the Mullet Island cormorant (*Phalacrocorax auritus*) breeding colony during 1997 and again during 1998.

Introduction

Disease is rarely a random event and does not just happen. Instead, the occurrence of disease is an outcome involving susceptible hosts, agents capable of causing disease and suitable environmental factors that facilitate host-agent interactions in a manner that results in disease (Friend, 1995a). Disease processes are effected by a variety of factors. In addition to genetics (Read et al., 1995), factors that stress the host in various ways can predispose the host to disease agents that may be encountered (Friend & Trainer, 1970, 1974a, b; Wobeser 1981; Zapata & Cooper, 1990; Ficken, 1991; Lloyd, 1995; Haschek & Rousseaux, 1998). Also, the environmental persistence of disease agents at levels capable of initiating disease may be greatly affected by environmental conditions (Price & Brand, 1984; Metcalf et al., 1994). There also must be adequate pathways for disease agents to reach the host and cause disease (Atkinson & Van Riper, 1991; Ewald, 1995; Lockhart et al., 1996; Gratz, 1999; Rocke & Friend, 1999; Hillis, 2000). The factors just noted are important forces driving the changing pattern of avian disease at the Salton Sea.

A wide variety of avian species have been affected by disease at the Salton Sea. The array and frequency of disease outbreaks (epizootics) involving migratory birds within that ecosystem has increased dramatically within the 1990s. Disease outbreaks now occur annually, often killing thousands of birds. Mortality has exceeded 150 000 during a single event (Salton Sea National Wildlife Refuge Narrative Reports; National Wildlife Health Center Epizootic Database). The increased frequency of occurrence, magnitude of losses and variety of diseases occurring at the Salton Sea (Table 1; Fig. 1) are indicative of an ecosystem under severe stress (Rapport & Whitford, 1999).

Environmental change as a factor for disease emergence

The Salton Sea is a young ecosystem initially formed during the period of 1905–7 by floodwaters of the Colorado River (Kennan, 1917). The resulting freshwater lake became nearly as salty as ocean water by 1929 (Arnal, 1961). Salinity now exceeds ocean water by about 25% (González et al., 1998). The fauna of the Salton Sea has changed from freshwater to marine species in response to increasing salinity and introductions of salt tolerant species (Arnal, 1961; Walker, 1961; Black, 1988). Thus, considering the time-frame for the major transition that has occurred, the physical environment and biota of the Salton Sea have been subject to dynamic change.

Environmental change is commonly associated with disease emergence, maintenance and spread (Friend, 1992; May, 1993; Schrag & Wiener, 1995; Epstein et al., 1998; DaSilva & Iaccarino 1999). Important factors include environmental quality as a function of organism health, and species associations and interactions as opportunities for transfer of disease agents. The environmental quality of the Salton Sea is degrading due to increasing salinity, excess nutrients that result in the Sea being hypereutrophic, and other factors. In combination with the harsh physical conditions present, those factors place stress on much of the Sea's fauna. In addition, biotic relations within this ecosystem are still evolving due to the young age of the Salton Sea and the unique species associations within this waterbody.

The Salton Sea has become an attractant for birds because of habitat losses within California, an abundant food base, and a minimum of disturbance factors for the birds using the Sea. California leads the United States in wetland losses (more than 90% of the inland wetland acreage present at the time of settlement [Dahl, 1990]). The abundant fishery of the Salton Sea provides a reliable food base for fish-eating birds, while other wetland-dependant avian species feed on the invertebrates present and other species heavily utilize the surrounding agricultural fields, another part of the changing landscape of the California Desert. Isolation from disturbance is provided by the large size of the Salton Sea (surface area of 980 km²) and low human density surrounding the Sea. The physical location of this waterbody along the Pacific Flyway is another factor contributing to the high bird use during migration and wintering periods.

The pattern of bird-use at the Salton Sea is more than just a transient stop-over for migratory birds. In addition to resident species, a high percentage of the populations for some other species are present for prolonged periods of time. Increased vulnerability to epizootic disease has been an outcome. The causes for disease emergence are multiple, complex, different for different diseases and are currently under investigation. Disease carriers among the birds using the Sea may be contributing to the infectious diseases present (Brand, 1984; Samuel et al., 1997; Friend & Franson, 1999). Novel species interactions within the evolving environment of the Salton Sea are another likely factor. This article reports the changing pattern of avian disease at the Salton Sea as an initial compendium against which other evaluations of avian disease can be made.

Methods

Narrative reports of the U.S. Fish and Wildlife Service National Wildlife Refuge System and the U.S. Geological Survey's National Wildlife Health Center Epizootic Database are the primary data sources utilized for the evaluations made. Those data were supplemented by correspondence obtained from a search of records in the National Archives and by published literature and government agency reports.

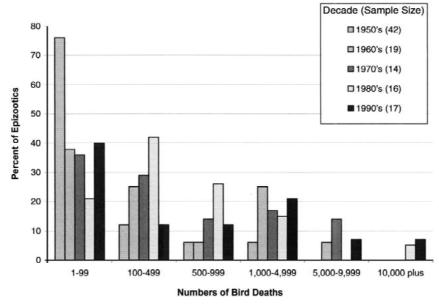


Figure 1. Reported avian mortality events within the Salton Sea ecosystem, 1950-2000.

Disease ^c	Causative agent	Agent type	Year of first report	Frequency of subsequent reports ^d
Avian botulism	Clostridium botulinum, type C	Bacterial toxin	1917	Common
Avian cholera	Pasteurella multocida	Bacteria	1978	Common
Salmonellosis	Salmonella typhimurium	Bacteria	1989	Frequent
Staphylococcosis	Staphylococcus aureus	Bacteria	1997	Rare
Pseudomoniasis	Pseudomonas sp	Bacteria	1992	Rare
Avian tuberculosis	Mycobacterium avium	Bacteria	1999	Rare
Aspergillosis	Aspergillus fumigatus	Fungus	1992	Occasional
Newcastle disease	Newcastle disease virus (PMV-3)	Virus	1997	Common
Parmyxovirus infection	Avian paramyxovirus type 3 (PMV-3)	Virus	1997	Rare
Hepatitis	Not identified	Unknown	1991	Rare
Neoplasia	Not identified	Unknown	1999	Rare
Lead poisoning	Spent lead shot	Heavy metal	1920s ^e	Occasional
Pesticides	Diamethoate, phorate, parathion	Organophosphates	1978	Occasional
	Carbofuran	Carbamate	1974	Occasional
	Strychnine	Poison	1991	Rare

Table 1. Reported causes of avian mortality within the Salton Sea ecosystem, 1907 through 1999^{a, b}

^aNational Wildlife Health Center diagnostic cases database primary data source (1975-2000).

^bDoes not include hunting or predation.

^cDoes not include diagnoses lacking supporting information, trauma, emaciation, drowning or secondary findings associated with primary cause of death (such as parasites). ^dCommon = numerous diagnosis with occurrence in most years; Frequent = numerous diagnoses with occurrence more

years than not; Occasional = periodic appearance; Rare = seldom observed. ^eEstimated occurrence based on descriptions of sick birds and history of disease in waterfowl.

Table 2.	Data sources	used fo	r Salton	Sea avian	disease	synopsis

Time period	Bureau of Biological Survey records	Salton Sea ^a National Wildlife Refuge Narrative Reports		l Wildlife ^b iter Databases Diagnostic	Scientific literature	Miscellaneous reports
				8		
1907–29	Х				Х	
1930–39	Х				Х	
1940–49		X ^c			Х	
1950–59		X ^d			Х	
1960-69		X ^e			Х	
1970–79		X ^f	Х	Х	Х	Х
1980-89		X ^g	Х	Х	Х	Х
1990–99		X ^h	Х	Х	Х	Х

^aRefuge initiated in 1930, first report available was 1939.

^bCenter came into existence in 1975.

^cReports found only for 1940, 1941, 1947–49.

^dReports found for all years.

eReports found for all years.

^fNo report found for 1977.

^gNo reports found for 1981 or 1982.

^hReports found for all years.

National Wildlife Refuge System narrative reports

The National Wildlife Refuge System has narrative reports for each of its holdings. Those reports provide a mixture of data, anecdotal comments and other information that serves as a chronicle for that refuge, as well as the Salton Sea ecosystem as a whole. Disease occurrence is one of the items regularly commented on and recorded in those reports. The Salton Sea Migratory Bird Refuge was established in 1930, later became the Salton Sea National Wildlife Refuge and then the Sonny Bono Salton Sea National Wildlife Refuge. Narrative reports for that refuge were individually reviewed for information on avian mortality. Information found often lead to other sources where greater detail or questions about the information could be resolved. The format and frequency for the narrative reports varied over time and reports could not be located for all years (Table 2). Tables and comments in some reports filled in some of the data gaps for years when reports were missing. In other instances, there is no information for years for which reports could not be found. Photographs found in reports showing field conditions and clinical signs of affected birds were of value in reaching judgments about diseases involved (Friend & Franson, 1999).

National Wildlife Health Center epizootic database

The National Wildlife Health Center began in January, 1975 as a research center within the U.S. Fish and Wildlife Service (FWS). Along with the other FWS research centers, it was placed within the National Biological Survey in 1993 (Friend, 1995b) and in 1996 became part of the U.S. Geological Survey during the continuing Department of the Interior reorganizations of the 1990s. Major activities of the Center include disease diagnosis and field investigations of wildlife mortality events on U.S. Department of Interior holdings. The great majority of those activities are associated with the National Wildlife Refuge System. The Center maintains an epizootic database that has records of wildlife mortality events from across the United States. The epizootic database contains a broad spectrum of information associated with each epizootic. Computer searches were utilized to obtain basic information and where needed, individual case files were examined to resolve questions. The author founded the National Wildlife Health Center and served as its Director from 1975–98. As a result, data evaluation for this paper was facilitated by the personal knowledge of Center operation, data acquisition processes and the databases.

Data evaluations

Epizootic events were assigned to specific diseases on the basis of laboratory confirmed diagnoses when such data were available. In other instances, narrative descriptions of clinical signs, field observations, photographs that were part of the record and other information provided or present in other reports of the same event were used to exercise professional judgment regarding the validity of the stated cause of the mortality and to infer cause where none was stated and it was reasonable to make such judgments.

Results

1907-1919

In 1917, duck sickness, now known to be type C avian botulism, was reported from the Salton Sea (Kalmbach & Gunderson, 1934). No other reports of avian mortality at the Salton Sea were found for that time period.

1920-1929

Correspondence between a Westmoreland resident and the U.S. Bureau of Biological Survey focused on large-scale avian mortality events the resident had been observing annually since 1926, with the exception of 1932 (Holmes, 1933a,b). Based on descriptive information from a 1933 field investigation of the area of the Salton Sea referred to by the resident (Tonkin, 1933) and disease diagnostic evaluations of other events at the Salton Sea by the leading avian botulism investigators of that time (Hobmaier, 1930a,b, 1932; Kalmbach, 1934a,b) it is likely that the 1926–29 mortality events (Holmes, 1933a,b) were due to type C avian botulism. The deltas of the New and Alamo Rivers were identified as the locations for those events.

1930–1939

Hobmaier (1932) reported avian botulism at the Salton Sea and noted a major epizootic occurred in 1931. Two years later, a field investigation was conducted at the southeastern shore of the Salton Sea in response to the claim, "... that several *hundred thousand* ducks have been lost this season from this cause" (Holmes, 1933a). Although that level of mortality is highly suspect based on Bureau of Biological Survey file correspondence it is clear that a major epizootic did occur. The field report states: "On the mud flats between the mouths of the Alamo and New Rivers there were hundreds of sick ducks and gulls and others out in the soup, where the boat could barely run. Out in the muddy water there were still fewer birds. Probably the number of birds lost by sickness since the hunting season opened will equal the number killed by hunters in that area, or on all the Sea, during the same period" (Tonkin, 1933).

The field situation and clinical signs described strongly suggest avian botulism as the cause of the 1933 epizootic. This evaluation is further supported by reference to an unavailable 1934 report by E.R. Kalmbach titled, "The Salton Sea Refuge With Respect to Possible Future Outbreaks of Duck Sickness and their Control". That missing report was referenced in a response to considerations for enlargement of the Salton Sea Migratory Bird Refuge and as further support for his position that:

"...I am convinced that one of the most valuable features of the improvement works on the Salton Sea Refuge will be those that will curb botulism and thus save many waterfowl. Without adequate provisions for disease control, an enlarged refuge might become simply an aggravated death trap" (Kalmbach, 1935a).

Kalmbach (1938) reported 800 dead birds per linear mile on some sections of the Salton Sea shoreline during the late summer 1933 epizootic. He also reported a recurring outbreak at the same locations the following spring. The only other reported epizootic for the 1930s occurred in 1939. An estimated 3000 birds, primarily waterfowl and shorebirds, died from avian botulism in the vicinity of Mullet Island. It was noted in the 1939 Narrative Report that, "... This is a small number when compared with some of the estimated figures of past outbreaks in the Salton Sea area".

1940–1949

There were no reports of major epizootics at the Salton Sea during the 1940s. A few cases of avian botulism were reported during 1940 and the November–January Narrative Report contains the statement, "The season in which botulism ordinarily breaks out passed without its occurrence in the area, only a few sick gulls were found". An accompanying photograph of a sick gull was consistent with avian botulism. Reports in 1942 of disease in waterfowl resulted in a visit to the Salton Sea National Wildlife Refuge that year by a high level FWS administrator (O'Neill, 1999). The September–December 1947 Narrative Report documents an estimated loss of 250 waterfowl from avian botulism at the Imperial Waterfowl Management Area. An undocumented number of dead eared grebes were also noted along the east shore of the Salton Sea during 1947 (O'Neill, 1999). The 1948 report noted that no avian disease was detected in the general vicinity of the Salton Sea. Less than 10 sick and dead birds were observed during 1949, none of which were submitted for diagnostic evaluations.

1950-1959

Avian botulism was again the only disease reported to be affecting birds at the Salton Sea during the 1950s. The 1950 Narrative Reports indicated no disease occurrence at the Salton Sea but note an outbreak of botulism that killed 4600 pheasants (Phasianus colchicus) at the State's game farm near Calipatria. Approximately 300 waterfowl and an equal number of shorebirds died at the Salton Sea during 1951 and between 4000 and 5000 waterfowl and 1000 shorebirds during 1952. That epizootic appears to be the same event referred to by O'Neil (1999) as occurring during 1953. Very few botulism affected birds were seen during 1953 but the Narrative Report noted continued observations of large numbers of flightless ruddy ducks (Oxyura jamaicensis) along the Sea. No cause was postulated for what was reported to be a recurring situation. No specimens were submitted for diagnostic evaluations. A 1953 serologic survey for Newcastle disease disclosed five pintails (Anas acuta) and two wigeon (A. americana) from among 313 waterfowl tested to have antibody titers between 1:64 and 1:128 or greater. One hundred and seventy-six coots (Fulica americana) tested were negative. Follow-up sampling during 1954 resulted in 1 of 338 waterfowl having a positive antibody titer to Newcastle disease. None of the five additional coots tested positive.

A few shorebirds, egrets and eared grebes were noted sick in the January–April 1954 Narrative Report; the May–August report for that year indicated no disease among waterfowl, two sick snowy egrets (*Egretta thula*) and a few sick sandpipers.

Observations and documentation of avian botulism were absent from the 1955 Narrative Reports, but reports of sick birds included approximately 300 flightless coots seen on the Sea. Those birds were reported to be emaciated and to have ink-black watery feces. At that same time period, a few sick white pelicans unable to fly and in a weakened condition were also seen. Other notations of disease during 1955 were 20 dead egrets due to unknown causes and additional small numbers of sick and dead white pelicans and egrets.

Small numbers of sick and dead birds were also reported for 1956. Avian botulism affected unspecified small numbers of black-necked stilts (*Himantopus mexicanus*). Mortality of egrets and mourning doves (*Zenaida macroura*) from unknown causes was also reported along with several cases of lead poisoning among pintail ducks. Similar reports involving small numbers of birds dying from avian botulism and from unknown causes appear in the 1957–9 reports. Avian botulism was diagnosed as the cause of mortality (number of affected birds not given) on the State's Finney-Ramer Unit of the Imperial Waterfowl Management Area near Calipatria.

1960–1969

In 1962, following two years of reports of no disease occurrence, several thousand birds died at the Salton Sea. Avian botulism killed an estimated 4000 ducks and 2000 shorebirds during one event. An additional 1000 shorebirds died from unknown causes during another event. Other undiagnosed mortality events reported during the year included the loss of 2000 shorebirds and 500 ducks, a considerable die-off of sandpipers (numbers not given), and the deaths of 4 white pelicans. During 1963, avian botulism killed 2000 waterfowl and 4000 shorebirds during one event and 2000 shorebirds and 500 waterfowl during an additional event. An estimated 2000-3000 shorebirds and a few white pelicans also died during another event for which the cause was not determined. Reported losses for 1964 were 300 ducks and 500 shorebirds dying from avian botulism. Parathion was diagnosed as the cause of death for 13 Canada geese (Branta canadensis) found dead in an alfalfa field a few miles from the Salton Sea near Brawley. Losses of approximately 400 waterfowl but only a small number (30) of shorebirds from avian botulism were reported for 1965.

Reported bird losses from disease continued to decline during the remainder of the 1960s. An estimated 30 waterfowl and 50 shorebirds died from avian botulism during 1966. Estimated losses from avian botulism were approximately 100 per year during 1967–9 with waterfowl and shorebirds being the primary species affected. No other diseases were reported during those years.

The low levels of avian mortality for the latter half of the 1960s persisted through 1970 and 1971. Two outbreaks of avian botulism were recorded for 1970, the first killing an estimated 25 waterfowl and 50 shorebirds and the other an estimated 450 waterfowl and 200 shorebirds. Losses from avian botulism during 1971 were estimated at 150 waterfowl and 100 shorebirds. An epizootic of avian botulism causing substantial but unspecified losses occurred during the spring of 1972 on the Wister Unit of the Imperial Waterfowl Management Area (Hosier, 1975). That event was followed by a major avian botulism epizootic during the winter of 1972 at the Alamo River delta that resulted in a total carcass pick up of 4325 waterfowl, 465 other dead birds and 199 sick birds during a 3 week disease response effort. Total losses were not estimated. Small numbers of botulism affected birds were reported during 1973. During 1974, botulism resulted in the collection of over 1000 shorebirds of 10 different species in addition to coots, grebes and ruddy ducks. Less than 200 dead ducks were found. A major mortality event involving wigeon due to the pesticide carbofuran also occurred during 1974, but not at the Salton Sea. That event occurred in Riverside County agricultural fields and killed 2400 birds.

Several mortality events were reported during 1975. About 400 ducks died from avian botulism during one event, and approximately 200 ducks and a few shorebirds during another event. In addition, about 200 eared grebes were found dead of unknown causes. The death of more than 100 soft-shelled turtles (*Trionyx spiniferus*) was also reported from a drainage canal that runs through one of the refuge units. The California Department of Fish and Game examined specimens and issued a diagnosis of pesticide poisoning from applications farther south in the Imperial Valley. The pesticide involved was not identified in the Narrative Report.

Disease is not reported to have occurred during 1976 and the Narrative Report for 1977 could not be found. There are no records of any specimens from the Salton Sea being submitted to the National Wild-life Health Center during 1976–77. Disease outbreaks are absent from the 1978 Narrative Report. However, several events were recorded for 1978. A total of 10 (waterfowl and shorebirds), for which no diagnosis was made, were found on the Salton Sea. Phorate was diagnosed as the cause of poisoning of 20 ring-billed gulls (*Larus delawarensis*) from agricultural fields in

Calipatria. The most significant event was the first appearance of avian cholera in waterfowl in Imperial County. A total of 261 carcasses were retrieved from the Imperial Wildlife Area due to avian cholera, gunshot (hunting season birds not retrieved) and unknown causes. At the start of 1979, the first major epizootic of avian cholera appeared at the Salton Sea. Mortality was reported by Brand & Duncan (1983) to be 3800 waterfowl, shorebirds and wading birds. However, other records indicate that event killed more than 9000 birds, primarily waterfowl. Avian botulism also occurred during 1979. A total of 795 carcasses were collected in association with that event.

1980-1989

Disease occurrence is not prominent in the Narrative Reports for most of the 1980s. The National Wildlife Health Center established nine wildlife mortality transects at the Salton Sea during 1980, but those transects were only run during that year. None of the 10 bird carcasses found were suitable for necropsy. Avian cholera was diagnosed for 12 carcasses (waterfowl, coots, an eared grebe and a gull) found incidental to conducting the transects; type C avian botulism was diagnosed for two white pelicans; and gunshot for a snow goose (Chen caerulescens) and a yellow-headed blackbird (Xanthocephalus xanthocephalus) (Brand, 1981). The National Wildlife Health Center epizootic database contains an entry of avian botulism killing 200 birds at the Salton Sea during 1980 and an avian cholera outbreak killing 250 birds during 1981. There are no reports of disease for 1982. Another small avian cholera event that occurred during 1983 resulted in a tally of 235 carcasses. Avian botulism resulted in 312 carcasses during 1984. There are no reports of disease at the Salton Sea during 1985 or 1986. Carbofuran killed 32 Canada geese in agricultural fields at El Centro during 1986.

As estimated 500 waterfowl and other species died from avian cholera at the Salton Sea during 1987. Avian cholera reappeared during 1988, killing an estimated 600 birds, primarily ruddy ducks and eared grebes. Several outbreaks of avian botulism also occurred during that year. Estimated total losses were 2000 birds. An additional botulism epizootic resulted in 595 waterfowl and shorebird carcasses being disposed of from a private hunting club in the immediate vicinity of the Salton Sea.

Several different mortality events were recorded during 1989. A total of 215 waterfowl and shorebird caresses were collected during an outbreak of avian botulism. Avian cholera resulted in an estimated 750 bird loss during one event and 56 carcasses being recovered from another event. Lead poisoning was diagnosed as the cause of death for 4 pintail ducks found dead on the Sea and as the cause of an estimated 200 ducks found dead at the Brawley Gun Club. In addition, 4515 cattle egrets died from salmonellosis during an epizootic at Brawley. Dimethoate was diagnosed as the cause of mortality for 5 snow geese and 1 Ross' goose (*C. rossii*). Also, Jehl (1996) reported an estimated 40 000 bird die-off of eared grebes to unknown causes.

1990-1999

Disease outbreaks at the Salton Sea were reported for each year of the 1990s. The magnitude of bird losses recorded during the 1990s exceeds the totals reported for all other years combined since 1930. Judgments cannot be made relative to losses prior to 1930. The frequency of reported large-scale epizootics was greater during the 1990s than for any other decade (Fig. 1) as was the frequency of reported disease events (Fig. 2) and the variety of disease conditions diagnosed (Table 1). The most notable epizootics of the decade are those killing an estimated 155 000 birds during 1992; 20 000 birds during 1994 and 1996; 8000 birds during 1997; 6000 birds during 1998; and 7000 birds during 1999 (Table 4).

The great majority of birds lost during the 1992 epizootic were eared grebes. Other than ruddy ducks, no other species suffered major losses. The primary cause for the event was not determined despite extensive diagnostic efforts by the National Wildlife Health Center. Jehl (1996) reported similar field signs for the 1989 grebe mortality event and postulated that the loss during that year may have equaled that for 1992. Algal toxins were suspected but that diagnosis could not be confirmed. Avian cholera was diagnosed as the cause of some of the bird deaths but was considered to be a secondary mortality factor. The primary cause of the 20000 bird loss in 1994 also was not determined. Once again, the eared grebe was the primary species affected, and avian cholera was determined to be a secondary cause of mortality associated with the die-off.

The 20 000 bird loss in 1996 was a unique event. That was the first reported occurrence of type C botulism involving major losses of fish-eating birds. More than 8500 white pelicans and more than 1100 endangered California brown pelicans died during that event. Another unique event took place during 1997 when Newcastle disease killed approximately 2500 double-crested cormorants nesting on Mullet Island. That event was the first diagnosis within the United States in wild birds of a lethal strain of Newcastle disease west of the Rocky Mountains. Avian cholera was responsible for the deaths of nearly 8000 birds collected during a late 1997 epizootic that extended well into 1998. The great majority of birds killed were ruddy ducks. A repeat Newcastle disease epizootic at Mullet Island during 1998 killed an estimated 6000 cormorants.

In comparison with the other Salton Sea major epizootics of the 1990s, the 1999 avian cholera epizootic is almost mundane. A total of 6921 carcasses was collected for disposal, the majority of those being ruddy ducks and northern shovelers (*A. clypeata*). Seventyfour white pelican carcasses were also among the wide variety of species affected.

Discussion

The primary variables effecting the quality of the information obtained are the probability for disease detection, the availability of records for disease events, the quality of disease diagnosis, and the accuracy of numbers of birds affected. Major events that have bearing on those variables are the 1930 development of the Salton Sea Migratory Bird Refuge, the 1975 development of the National Wildlife Health Center and the 1998 initiation of the Salton Sea Restoration Project.

Disease surveillance prior to the development of the Salton Sea Migratory Bird Refuge was limited to random observations by local residents and visitors to the Sea. Records of observations those individuals may have made generally do not appear as public documents. However, notification and requests for government actions do result in a record of the event (Holmes, 1939a,b). Major disease events commonly result in such requests and subsequent response by government agencies (Tokin, 1933; National Wildlife Health Center Epizootic Database). The early focus on the Salton Sea for recreational activities and for shoreline development (Laflin, 1995; du Boyes & Myers, 1999; Horvitz, 1999) resulted in considerable on-Sea presence by humans prior to the presence of government natural resources agencies having on-Sea programs. Also, the focus for developing the Salton

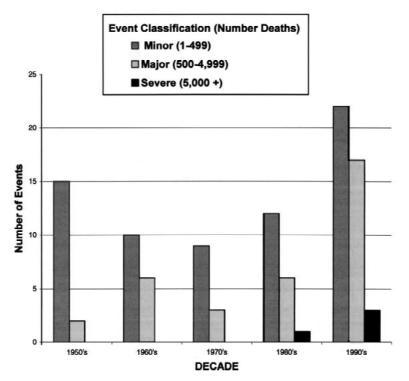


Figure 2. Number and severity over time of avian mortality events within the Salton Sea ecosystem, 1950–2000.

Sea as California's Riviera made it likely that major avian mortality events would have been detected and government agencies notified.

The development of on-Sea natural resources facilities provided for enhanced probability for detection and recording of avian mortality events of all magnitudes. The Salton Sea Migratory Bird Refuge (1930), the Wister Unit of the Imperial Waterfowl Management Area (1954) and the Salton Sea State Park (1955) are the primary programs with staff that are frequently on the Sea and interact with the public that use the Sea. The most intensive disease surveillance activities at the Salton Sea have been those carried out by personnel of the Sonny Bono Salton Sea National Wildlife Refuge. Those efforts were further enhanced as a result of the 1996 avian botulism epizootic and by the initiation of the Salton Sea restoration project.

The greatest Narrative Report data gaps are for the 1930s and during the World War II years of the 1940s. Some coverage for those years is provided by the records of the Bureau of Biological Survey and by the scientific literature. Also, it is noteworthy that O'Neill (1999) does not highlight disease in his coverage of the early history and development of the Salton Sea Migratory Bird Refuge despite considerable focus on disease for some of the other refuges included in his presentation of personal experiences on nine western state and federal wildlife refuges. However, he does provide support for disease being a major problem during the 1930s by citing comments from the 75th Congress (1937) about the Salton Sea Wildlife Refuge "... and the control of the diseases that take such a great toll of the bird life of the region".

The quality of avian disease evaluations at the Salton Sea is also influenced by such factors as the quality of field observations, the selection and quality of specimens provided to diagnostic laboratories (Friend & Franson, 1999), the use of diagnostic laboratories, completeness of diagnostic work, how familiar the laboratory is with the types of diseases likely to affect wild bird populations, technology available and other factors.

A continuum of highly competent wildlife disease investigators and programs have been associated with avian disease evaluations at the Salton Sea. Included are the wildlife disease investigation laboratories of the Bureau of Biological Survey, California Department of Fish and Game and the U.S. Fish and

Table 3. Primary recurring diseases causing major losses of Salton Sea avifauna

Disease/causative agent	First documented occurrence in wild birds	Identification of the causative agent	First documented at the Salton Sea
Avian botulism	1890s	1930	1917
Clostridium botulinum, type C	(Hobmaier, 1932;	(Giltner & Couch, 1930;	(Kalmbach &
ciosintatam bolannam, type c	Kalmbach, 1968; Kalmbach & Gunderson, 1934)	Hobmaier 1930a,b; Kalmbach, 1930)	Gunderson, 1934)
Avian cholera	1940 ^a	1879–1880	1978 ^b
Pasteurella multocida	(Rosen, 1971)	(Rosen, 1971)	
Avian salmonellosis	1930s	1895	1989 ^c
Salmonella typhimurium	(Steele & Galton, 1971)	(Williams, 1965)	
Newcastle disease Newcastle disease virus	1950 (Blaxland, 1951; Brandly & Hanson, 1965; Wilson, 1950)	1926 (Hanson, 1964)	1997°

^aFirst epizootic in free-ranging waterfowl; similar events first occurred in the United States in 1944. Small numbers of wild birds closely associated with poultry rearing operations have previously died from avian cholera.

^bSalton Sea National Wildlife Refuge Narrative Report.

^cNational Wildlife Health Center Epizootic Database.

Wildlife Service (subsequently, the National Biological Survey and then the U.S. Geological Survey). Also, the primary diseases diagnosed at the Salton Sea have been recognized elsewhere as distinct diseases for many years prior to their diagnosis at the Salton Sea. They are all diseases routinely diagnosed by commonly applied technology and methods (Table 3).

The ease and speed for getting specimens to diagnostic laboratories has improved over time, as has the amount of on-site mortality event involvement by wildlife disease specialists. Nevertheless, continued weaknesses result from the limited numbers of specimens submitted to diagnostic laboratories and the small percentage of disease events for which on-site investigations are carried out by wildlife disease specialists. Despite those inherent weaknesses, the quality of disease diagnostic evaluations at the Salton Sea has been consistently good over time based on the composite of information available for evaluation.

The amount of laboratory evaluations in support of field observations is as good as that for most other geographic areas and better than for many areas. Also, the clinical and field signs associated with the diseases diagnosed at the Salton Sea, while not pathognomonic, have distinctive differences from one another, but not all other avian diseases (Friend & Franson, 1999). Those differences help to make informed field evaluations. Some diseases may not have been diagnosed. However, with the exception of the recurring unknown cause(s) of eared grebe mortality, it is likely that any major disease affecting the avifauna of the Salton Sea has been identified.

Data relative to the numbers of birds lost during disease events must be interpreted with caution. Carcass counts provide the best information but search effort can be highly variable and carcasses found are minimum numbers of the actual loss. Estimates of total losses can either underestimate or overestimate the actual losses. Light conditions, bird size and color, habitat, methods of search and numerous other factors can effect field estimates for numbers of birds lost and the number of carcasses retrieved (Wobeser, 1994). Therefore, comparisons between numbers of birds lost to disease during different events and times at the Salton Sea can only be based on subjective evaluations because no corrective factor can be applied to enhance interpretations for the data reported.

Pesticide-related deaths of birds documented within the Salton Sea ecosystem have been associated with agricultural fields of the surrounding area. With the exception of the 1974 loss from carbofuran of approximately 2400 American wigeon, pesticide

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Year	Location	Disease	Mortality	Primary species affected
1930	Alamo & New River deltas	Avian botulism	Large-scale ^b	Waterfowl
1931	Alamo & New River deltas	Avian botulism	Large-scale ^c	Waterfowl
1933	Alamo & New River deltas	Avian botulism	'Thousands' ^d	Waterfowl
1952	Salton Sea shoreline & impoundments	Avian botulism	4–5000	Waterfowl
1963	Shallow muddy areas of	Avian botulism	4000	Ducks
	Salton Sea shoreline		2000	Shorebirds
1972	Alamo & New River deltas	Avian botulism	6000	Waterfowl & shorebirds
1979	East side of Salton Sea	Avian cholera	9037	Waterfowl
1989	Imperial County breeding colonies, Brawley ^f	Salmonellosis	4515 ^g	Cattle egrets
1989	Salton Sea ^h	Not determined	40000^{i}	Eared grebes
1992	Salton Sea-Sea wide	Not determined; ^j avian	150 000	Eared grebes
		cholera secondary problem	45 000	Ruddy ducks
1994	Salton Sea – Sea wide	Not determined; ^j avian cholera secondary problem	20 000	Mostly eared grebes, also ruddy ducks & gulls as lesser numbers of primary species
1996	Alamo & New River deltas primary area	Avian botulism ^k	20 000 ¹	White pelican, brown pelican, great egret, gulls
1997	Wister Unit, southern areas of the Sea, impoundments	Avian cholera	7952 ^e	Ruddy duck, northern shoveler, coot
1998	Mullet Island	Newcastle disease	6000 ^m	Double-crested cormorant
1999	Alamo & New River deltas, Unit 1	Avian cholera	6921 ^e	Ruddy duck northern shoveler, gulls

Table 4. Mortality events within the Salton Sea ecosystem causing severe losses of avifauna, 1930 through 1999^a

^aSevere losses are arbitrarily considered to be approximately 5000 birds or greater based on estimated losses and 4000 birds or greater based on carcass counts.

^bThe actual severity cannot be determined; Holmes (1933a, b) reported annual large-scale mortalities at those locations starting in 1926. ^cThe actual severity cannot be determined; Hobmair (1932) reported a major epizootic.

^dThe actual severity cannot be determined; Holmes (1933a) reported several hundered thousand but field investigations by Tonkin (1933), while confirming a major epizootic, do not support the magnitude of loss reported by Holmes (1933a).

^eCarcass count, actual mortality greater.

^fTwo breeding colonies at separate locations.

^gBased on field investigation of affected colonies.

^hCarcasses found along west and north shores but mortality extended across the Sea.

ⁱJehl (1996) speculated that mortality may have equaled the 1992 event.

^jAvian cholera was responsible for much of the ruddy duck mortality.

^kFirst epizootic of type C avian botulism in fish-eating birds.

¹Largest documented die-off of an endangered species (over 1100 brown pelicans); an estimated 15–20% loss of the western population of white pelicans (over 8500 carcasses).

^mEstimated on basis of number of nests and the total loss of nestling produced.

mortalities have involved small numbers of birds and have only been sporadically reported. On some occasions when the causes for bird mortalities have not been diagnosed, the reports for those events contain speculation that pesticides may have been the cause. In those instances, no chemical residue analyses or associations with the timing of pesticide applications were provided as supporting data for pesticides as a probable cause. Instead, the context typically appeared to be speculation based on enhanced awareness of pesticide-wildlife problems during the years following the publication of 'Silent Spring' (Carson, 1962) and other scientific findings documenting pesticide impacts (Anderson et al., 1969; Stickel et al., 1969).

Another cause for mortality for which there is even greater speculation is that of algal toxins. This is not a new area for speculation. Statements of algal toxins being the suspected cause of disease in birds appear in Narrative Reports from the 1950s and 1960s. A current hypothesis is that algal toxins are the primary cause for the large-scale losses of eared grebes that have been occurring at the Salton Sea since at least 1989. Algae that are known to produce toxins are present at the Sea (Salton Sea Science Office database; Wood et al., 2001) algal toxins have been isolated from water samples collected at the Sea and algal toxins were recovered from the tissues of dead grebes during the 1992 epizootic, but not at levels that are known to cause avian mortality. Algal toxins isolated at the Salton Sea have been lethal for invertebrates but not for mice when evaluated in the laboratory (National Wildlife Health Center records). Avian cholera and avian botulism have both been diagnosed as causes of eared grebe mortality at the Sea (Salton Sea National Wildlife Refuge Narrative Reports; National Wildlife Health Center Epizootic Database) and both must still be considered as possible causes for the deaths of grebes for which no other etiologic agent has been identified.

An especially noteworthy change in the disease patterns of the Salton Sea during the 1990s is the general absence of the classical type C avian botulism events affecting shorebirds and waterfowl at the deltas of the Alamo and New Rivers. The large scale avian botulism epizootics of the past were not present. Instead, avian botulism is now primarily occurring in off-Sea freshwater impoundments except for the events involving pelicans and other fish-eating birds. The ecology of avian botulism in pelicans has not yet been elucidated but differs from the classical ecology of type C epizootics (Rocke & Friend, 1999). The diminished occurrence of classical outbreaks, typically involving dabbling ducks and shorebirds, on-Sea may be related to increased salinity and other water quality changes (Rocke & Samuel, 1999).

Conclusions

The information presented here is an annotated synopsis of the records for avian disease within the Salton Sea ecosystem. It is likely that additional information exists that was not found during the search for data, that in some instances the reported cause for events may be in error, and that there has been an increasing probability for detecting disease events over the timeframe considered. It is far less likely that major disease events have been missed or that the cause for major events have been misdiagnosed.

It is reasonable to conclude that the frequency for disease occurrence, variety of diseases causing avian mortality and magnitude of bird loses have changed greatly over time. Based on information available, more birds may have died from disease at the Salton Sea since 1989 than during the previous 82 years.

Avian botulism was the only major cause of bird mortality at the Sea until the 1979 epizootic of avian cholera. There are now four major diseases affecting Salton Sea avifauna; avian botulism, avian cholera, salmonellosis (1989) and Newcastle disease (1997); plus recurring mortality of eared grebes (1989) that is recognized on the basis of field signs, but for which the etiologic agent remains elusive. The 1996 epizootic of avian botulism in pelicans was an unprecedented event. The continued recurrence of type C toxin as a cause of mortality in fish-eating birds represents a unique expression of avian botulism that appears to be related to the live fish food base rather than to the typical maggot cycle for that disease.

All diagnosed major causes of avian mortality at the Salton Sea involve microbes as the disease agent. Those findings provide a clear indication of where attention should be focused to minimize avian losses not associated with predation and hunting. Pesticides have not been documented as a cause of on-Sea bird deaths. Several minor and one moderate pesticide mortality event have been documented in nearby agricultural fields. Those events have been sporadic and of low impact relative to numbers of birds lost to infectious disease and avian botulism.

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References

- Anderson, D. W., J. J. Hickey, R. W. Risebrough, D. F. Hughes & R. E. Christensen, 1969. Significance of chlorinated hydrocarbon residues to breeding pelicans and cormorants. Can. Field-Natur. 83: 91–112.
- Atkinson, C. T. & C. Van Riper III, 1991. Pathogenicity and epizootiology of avian haematozoa: *Plasmodium, Leucocytozoon*, and *Haemoproteus*. In Loye, J. E. & M. Zuk (eds), Bird– Parasite Interactions, Ecology, Evolution, and Behaviour. Oxford University Press, New York: 19–48.
- Arnal, R. E., 1961. Limnology, sedimentation, and micro-organisms of the Salton Sea, Cal. Geolog. Soc. am. Bull. 72: 427–478.
- Black, G.F., 1988. Description of the Salton Sea sport fishery 1982– 1983. Cal. Fish Game Admin. Rept: 88–89.
- Blaxland, J. D., 1951. Newcastle disease in shags and cormorants its significance as a factor in the spread of this disease among domestic poultry. Vet. Rec. 63: 731–733.
- Brand, C. J., 1981. Migratory bird mortality transects in California – 1980–81. National Wildlife Health Center, Madison, WI: 20 pp.
- Brand, C. J., 1984. Avian cholera in the Central and Mississippi Flyways during 1979–80. J. Wildl. Manage. 48: 399–406.
- Brand, C. J. & R. M. Duncan, 1983. Avian cholera in the American flamingo, Phoenicopterus ruber: a new host record. Calif. Fish and Game 69: 190–191.
- Brandly, C. A. & R. P. Hanson, 1965. Newcastle disease. In Biester, H. E. & L. H. Schwarte (eds), Diseases of Poultry, 5th edn. Iowa State University Press, Ames: 633–674.
- Carson, R., 1962. Silent spring. Houghton-Mifflin Co., Boston: 368 pp.
- Dahl, T. E., 1990. Wetland losses in the United States 1780s to 1980s. U.S. Fish and Wildlife Services, Washington, DC.: 13 pp.
- Da Silva, E. & M. Iaccarino, 1999. Emerging diseases: a global threat. Biotech. Adv. 17: 363–384.
- de Buys, W. & J. Myers, 1999. Salt Dreams. University of New Mexico Press, Albuquerque: 307 pp.
- Epstein, P. R., B. H. Sherman, E. Spanger-Siegfried, A. N. Langston, S. Prasad & B. McKay, 1998. Marine ecosystems: emerging disease as indicators of change. Center for Health and the Global Environment, Harvard Medical School, Boston: 85 pp.

- Ewald, P. W., 1995. The evolution of virulence: a unifying link between parasitology and ecology. J. Parasitol. 81: 659–669.
- Ficken, M. D., 1991. Necrotic enteritis. In Calnek, B. E., H. J. Barnes, C. W. Beard, W. M. Reid & H. W. Yoder, Jr. (eds), Diseases of Poultry, 9th edn. Iowa State University Press, Ames: 264–267.
- Friend, M., 1992. Environmental influences on major waterfowl diseases. Trans. N. am. Wildl. and Natural Resour. Conf. 57: 517–525.
- Friend, M., 1995a. Increase avian disease with habitat change. In LaRoe, E. T., G. S. Farris, C. E. Puckett, P. D. Doran & M. J. Mac (eds), Our Living Resources: A Report to the Nation on the Distribution, Abundance, and Health of U.S. Plants, Animals, and Ecosystems. U.S. Dept. Interior, Natl. Biol. Ser., Washington D.C.: 401–405.
- Friend, M., 1995b. Conservation landmarks: Bureau of Biological Survey and National Biological Service. In LaRoe, E. T., G. S. Farris, C. E. Puckett, P. D. Doran & M. J. Mac (eds), Our Living Resources: A Report to the Nation on the Distribution, Abundance, and Health of U.S. Plants, Animals, and Ecosystems. U.S. Dept. Interior, Natl. Biol. Ser., Washington D.C.: 7–9.
- Friend, M. & C. J. Franson (eds), 1999. Field Manual of Wildlife Disease. General Field Procedures and Diseases of Birds. U.S. Dept. Interior, U.S. Geological Survey, Information and Technology Report 1999–1001, Washington, D.C.: 426 pp.
- Friend, M. & D. O. Trainer, 1970. Polychlorinated biphenyl: interaction with DHV. Science 170: 1314–1316.
- Friend, M. & D. O. Trainer, 1974a. Experimental DDT-duck hepatitis virus interaction studies. J. Wildl. Manage. 38: 887–895.
- Friend, M. & D. O. Trainer, 1974b. Experimental dieldrin–duck hepatitis virus interaction studies. J. Wildl. Manage. 38: 896– 902.
- Giltner, L. T. & J. F. Couch, 1930. Western duck sickness and botulism. Science 72: 660.
- Gonzalez, M. R., C. M. Hart, J. R. Verfaillie & S. H. Hurlbert, 1998. Salinity and fish effects on Salton Sea microecosystems: water chemistry and nutrient cycling. Hydrobiologia 381: 105–128.
- Gratz, N. G., 1999. Emerging and resurging vector-borne disease. Annu. Rev. Entomol. 44: 51–75.
- Hanson, R. P. (ed.), 1964. Newcastle Disease Virus: an Evolving Pathogen. University of Wisconsin Press, Madison: 353 pp.
- Haschek, W. M. & C. G. Rousseaux, 1998. Fundamentals of Toxicologic Pathology. Academic Press, San Diego: 563 pp.
- Hillis, D. M., 2000. Origins of HIV. Science 288: 1757-1758.
- Hobmaier, M., 1930a. Duck disease caused by the toxin of *Clostridium botulinum*, type C. Proc. Soc. Exp. Biol. Med. 28: 339–340.
- Hobmaier, M., 1930b. Duck disease caused by the poison of Bacillus botulinus, California Fish & Game 16: 5–21.
- Hobmaier, M., 1932. Conditions and control of botulism (duck disease) in waterfowl. California Fish and Game 18: 5–21.
- Holmes, S. W., 1933a. November 16 letter to U.S. Department of Agriculture, Bureau of Biological Survey, Washington, D.C., National Archives, Washington, D.C.: 1 pp.
- Holmes, S. W., 1933b. November 17 letter to H.P Sheldon, Bureau of Biological Survey, Washington, D.C., National Archives, Washington, D.C.: 2 pp.
- Horvitz, S., 1999. Salton Sea 101 an introduction to the issues of the Salton Sea – California's greatest resource. California State Parks: 38 pp.
- Hosier, D. J., 1975. The ecology of avian botulism at the Salton Sea, California. M.S. Thesis, Calf. State Polytechnic University, Pomona: 66 pp.

- Jehl, J. R., Jr., 1996. Mass mortality events of eared grebes in North America. J. Field Ornithol. 67: 471–476.
- Kalmbach, E. R., 1930. Western duck sickness produced experimentally. Science 72: 658–660.
- Kalmbach, E. R., 1934. The Salton Sea refuge with respect to possible future outbreaks of duck sickness and their control. U.S. Bureau of Biological Survey, Denver, CO.
- Kalmbach, E. R., 1935a. Botulism and the proposed enlargement of the Salton Sea refuge. U.S. Bureau of Biological Survey, Denver, CO., March 20, National Archives, Washington, D.C.: 2 pp.
- Kalmbach, E. R., 1935b. Supplementary statement with respect to the development of the Salton Sea refuge and its relation to botulism. U.S. Bureau of Biological Survey, Denver, CO., March 28, National Archives, Washington, D.C.: 2 pp.
- Kalmbach, E. R., 1938. Botulism, a recurring hazard to waterfowl with notes on recent outbreaks in the United States, Canada, and Australia. Wildlife Research and Management Leaflet BS–120, Bureau of Biological Survey, Washington, D.C.: 8 pp.
- Kalmbach, E. R., 1968. Type C botulism among wild birds a historical sketch. Bureau Sport Fisheries and Wildlife, Special Scientific Report, Wildlife No. 10, Washington, D.C.: 8 pp.
- Kalmbach, E. R. & M. F. Gunderson., 1934. Western duck sickness, a form of botulism. U.S. Dept Agriculture, Tech. Bull. 411: 82 pp.
- Kennan, G., 1971. The Salton Sea: an Account of Harriman's Fight with the Colorado River. Macmillan, New York: 106 pp.
- Laflin, P., 1995. The Salton Sea California's Overlooked Treasure. The Periscope, Coachella Valley Historical Society, Indio: 56 pp.
- Lockhart, A. B., P. H. Thrall & J. Antonovics, 1966. Sexually transmitted disease in animals: evolutionary implications. Biol. Rev. 71: 415–471.
- Lloyd, S., 1995. Environmental influences on host immunity. In B. T. Grenfell & A. P. Dobson (eds), Ecology of Infectious Disease in Natural Populations. Cambridge University Press, Cambridge: 327–361.
- May, R. M., 1933. Ecology and evolution of host-virus associations. In Morse, S. S. (ed.), Emerging Viruses. Oxford University Press, New York, Oxford: 58–68.
- Metcalf, H. E. D. W. Luchsinger & W. C. Ray, 1994. Brucellosis. In Beran, G. W. & J. H. Steele (eds), Handbook of Zoonoses 2nd edn. Section A: Bacterial, Rickettsial, Chlamydial, and Mycotic. CRC Press, Boca Raton: 9–39.
- O'Neill, E. J., 1999. From behind the blue goose sign Graphic Press, Klamath Falls: 412 pp.
- Price, J. I. & C. J. Brand, 1984. Persistence of *Pasteurella multocida* in Nebraska wetlands under epizootic conditions. J. Wildl. Dis. 20: 90–94.
- Rapport, D. J. & W. G. Whitford, 1999. How ecosystems respond to stress. BioScience 49: 193–203.
- Read, A. F., S. D. Albon, J. Antonovics, V. Apanius, G. Dwyer, R. D. Holt, O. Judson, C. M. Lively, A. Martin-Löf, A. R. McLean, J. A. J. Metz, P. Schmid-Hempel, P. H. Thrall, S. Via & K. Wilson, 1995. Group report: genetics and evolution of infectious disease in natural populations. In Grenfell, B. T. & A. P. Dob-

son (eds), Ecology of Infectious Disease in Natural Populations. Cambridge University Press, Cambridge: 450–477.

- Rocke, T. E. & M. Friend, 1999. Avian Botulism. In Friend, M. & J. C. Franson (eds), Field Manual of Wildlife Disease. General Field Procedures and Diseases of Birds. U.S. Dept. Interior, U.S. Geological Survey, Information and Technology Report 1999– 2001, Washington, D.C.: 271–281.
- Rocke, T. E. & M. D. Samuel, 1999. Water and sediment characteristics associated with avian botulism outbreaks in wetlands. J. Wildl. Manage. 63: 1249–1260.
- Rosen, M. N., 1971. Avian cholera. In Davis, J. W., R. C. Anderson, L. Karstad & D. O. Trainer (eds), Infectious and Parasitic Diseases of Wild Birds. Iowa State University Press, Ames: 59–74.
- Salton Sea Science Office, 78–401 Highway 111, Suite R, La Quinta, CA 92253.
- Samuel, M. D., D. R. Goldberg, D. J. Shadduck, J. I. Price & E. G. Cooch, 1997. *Pasteurella multocida* serotype 1 isolated from a lesser snow goose: evidence of a carrier state. J. Wildl. Dis. 33: 332–335.
- Schrag, S. J. & P. Wiener, 1995. Emerging infectious disease: what are the relative roles of ecology and evolution? Tree 10: 319–234.
- Steele, J. H. & M. M. Galton, 1971. Salmonellosis. In Davis, J. W., R. C. Anderson, L. Karstad & D. O. Trainer (eds), Infectious and Parasitic Disease of Wild Birds. Iowa State University Press, Ames: 51–58.
- Stickel, W. H., L. F. Stickel & J. W. Spann. 1969. Tissue residues of dieldrin in relation to mortality in birds and mammals. In Miller, M. W. & C. C. Berg (eds), Chemical Fallout. Current Research on Persistent Pesticides, C.C. Thomas, Springfield: 174–204.
- Tonkin, G., 1933. November 26 letter to Chief, Bureau of Biological Survey, Washington, D.C., National Archives, Washington, D.C.: 2 pp.
- U.S. Fish and Wildlife Service, Sonny Bono National Wildlife Refuge Complex, 906 West Sinclair Road, Calipatria, CA 92233.
- U.S. Geological Survey, National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 537711.
- Walker, B. W. (ed.), 1961. The Ecology of the Salton Sea, California, in Relation to the Sport Fishery. Calif. Fish and Game Bull. 113: 1–204.
- Williams, J. E., 1965. Paratyphoid and Arizona infections. In Biester, H. E. & L. H. Schwarte (eds), Disease of Poultry, 5th edn. Iowa State University Press, Ames: 260–328.
- Wilson, J. E., 1950. Newcastle disease in a gannet (*Sula bassana*). A preliminary note. Vet. Rec. 62: 33–34.
- Wobeser, G. A., 1981. Disease of Wild Waterfowl. Plenum Press, New York and London: 78 p.
- Wobeser, G. A., 1994. Investigation and Management of Disease in Wild Animals. Plenum Press, New York and London: 265 pp.
- Wood, A. M., S. R. Miller, W. K. W. Li & R. W. Castenholz, 2002. Hydrobiologia 473/Dev. Hydrobiol. 161: 77–92.
- Zapata, A. G. & E. L. Cooper, 1990. The Immune System: Comparative Histophysiology. John Wiley & Sons, New York: 335 pp.