EPIZOOTIOLOGY OF AVIAN CHOLERA IN WILDFOWL

Author: Botzler, Richard G.

Source: Journal of Wildlife Diseases, 27(3) : 367-395

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-27.3.367

Journal of Wildlife Diseases, 27(3), 1991, pp. 367–395

EPIZOOTIOLOGY OF AVIAN CHOLERA IN WILDFOWL

Richard G. Botzler
Department of Wildlife, Humboldt State University, Arcata, California 95521, USA

ABSTRACT: *Pasteurella multocida*, the cause of avian cholera, has naturally infected over 100 species of free-living birds. Among wild birds, avian cholera has its greatest impact on North American wildfowl. Epizootics usually are explosive in onset and may involve thousands of birds. The disease has been reported in every month of the year among wildfowl. Disproportionate mortality, with some species suffering proportionately greater mortality than others, has been a common feature of this disease. Presence of animal organic matter plays a significant role in the survival of *P. multocida*. There are conflicting reports or a lack of information on the role of host sex, age, body size, other physical features, genetic variation or behavioral differences, as predisposing factors to infection by *P. multocida*. There also are ambiguities on the relationship between season, precipitation, temperature, nutritional stress, water quality, other microorganisms, and environmental contaminants, and the occurrence of avian cholera in wildfowl. Two competing hypotheses for the year-round reservoir of wildfowl strains of *P. multocida* are ambient soil or water of enzootic sites, and carrier animals; most current evidence favors the role of carrier animals. Transmission most likely occurs by ingestion of contaminated water, inhalation of bacteria-rich aerosols, or both. While many techniques have been proposed to prevent or control avian cholera, none have been rigorously tested to determine their effectiveness.

Key words: Avian cholera, *Pasteurella multocida*, epizootiology, waterfowl, review article.

INTRODUCTION

It is estimated that about 3.4 million hunters in North America north of Mexico kill approximately 20 million waterfowl each year, and that an equal amount of waterfowl die from nonhunting mortality (Friend, 1981). Stout and Cornwell (1976) estimated that diseases made up about 88% of the total nonhunting mortality studied among fledged North American waterfowl. As our wildfowl become increasingly concentrated on a diminishing habitat, transmissible diseases become ever greater concerns for waterfowl managers. One such threat is avian (fowl) cholera, an explosive disease found among waterfowl and associated species, upland game birds, and domestic fowl (Rosen, 1971; Wetzel and Rieck, 1972; Rhoades and Rimler, 1984).

In 1950, Rosen and Bischoff (1950) summarized current perceptions about avian cholera (*Pasteurella multocida* infection) among North American wildfowl populations. Since then, much research has been done (Wilson, 1979; Mulcahy et al., 1988), but critical information about many significant features of avian cholera in wildfowl still is lacking. There are no environmentally sound techniques for preventing avian cholera, nor for controlling epizootics in wildfowl populations. The effects of ambient environmental conditions, land use, and management practices on the initiation and course of the disease are not clear. It is my hope in this review paper to stimulate a continued interest in the study of avian cholera.

INFECTIVE AGENT

Pasteurella multocida, the bacterial agent causing avian cholera, has been reported from a wide variety of birds and mammals (Blackburn et al., 1975; Brogden and Rhoades, 1983). The genus name of *Pasteurella* was chosen to honor Louis Pasteur; the species epithet, *multocida*, reflects the broad host range of this parasite. Among birds, this bacterium was called *Pasteurella avicida* and *P. aviseptica* in earlier literature (Rhoades and Rimler, 1984).

Pasteurella multocida is an encapsulated Gram-negative bacterium whose shape may vary from a rod to a coccobacillus. A bipolar staining characteristic is evident

with methylene blue, Wrights stain, Giemsa stain and Gram stain (Wobeser, 1981).

Recently, Mutters et al. (1985) proposed three subspecies of *Pasteurella multocida*: *P. multocida multocida*, *P. multocida gallicida*, and *P. multocida septica*; these are distinguished by their differing actions in dulcitol, sorbitol, arabinose and trehalose. Most *P. multocida* strains from California (USA) wildfowl are classified as *P. multocida multocida* (63%), followed by *P. multocida gallicida* (37%), and *P. multocida septica* (<1%) (Hirsh et al., 1990).

Currently 16 serotypes of *P. multocida* are recognized in the Heddleston scheme (Rimler et al., 1984). While Heddleston (1976) found no consistent relation between serotype, biochemical characteristics, and host species of origin, most *P. multocida* strains isolated from wildfowl are serotype 1 in the Pacific, Central, and Mississippi Flyways of North America, with serotypes 3 and 4 predominating in the Atlantic Flyway (Brogden and Rhoades, 1983; Windingstad et al., 1983). In the Atlantic Flyway, avian cholera epizootics largely have been caused by serotype 3 *P. multocida*, until recently when serotype 1 strains have been isolated from eiders (*Somateria* spp.) (National Wildlife Health Research Center, Madison, Wisconsin 53711, USA, unpubl. data). More detailed descriptions of the biochemical and serological characteristics of *P. multocida* are reported by Rosen (1971), Rhoades and Rimler (1984), and Adlam and Rutter (1989).

HOST RANGE

With over 100 wild avian species known to have been naturally infected (Table 1), it is evident that *P. multocida* has a broad host range, as its species epithet implies. If one were to include all captive, zoo, and domestic birds from which *P. multocida* has been isolated, and the unpublished reports from agency files, the host range would be even greater. Probably most or all bird species are susceptible to avian cholera under the right circumstances, although it is noteworthy that infections are not yet reported among vultures (Cathartidae).

The citations in Table 1 are the first appearing in refereed literature for which it is reasonably certain that *P. multocida* was isolated from each species of free-living bird. This is not always the earliest date *P. multocida* was observed in that species; for example, Heddleston et al. (1972) and Brogden and Rhoades (1983) reported earlier isolation dates among some bird species.

Both McDiarmid (1962) and Rosen (1971) cited Suarez and Ilazabal (1941) as reporting avian cholera among puffins in Chile. There is no evidence that puffins occur naturally in Chile, although five species of shearwater (*Puffinus* spp.) are present (Johnson, 1965, 1967). Still, I found no mention of either puffins or shearwaters in Suarez and Ilazabal (1941). Keymer (1958) referred to an unidentified report of *P. multocida* in puffins (*Fratercula arctica*), but provided no details.

Avian cholera was reported in Japan among a population of wild ducks, presumably rosy-billed pochards (*Netta peposaca*), associated with a zoological garden (Fujihara et al., 1986). Although described by Fujihara et al. (1986) as wild birds originating in the U.S., rosy-billed pochards occur naturally only in temperate South America (Johnsgard, 1978).

Hinshaw and Emlen (1943) received a personal communication from J. E. Shillinger that bobwhite quail (*Colinus virginianus*) suffered epizootics of avian cholera, and Rosen (1971) reported a personal communication from L. N. Locke of mortality among common ravens (*Corvus corax*), but details are not available on these host extensions.

PATHOLOGY

Many signs associated with avian cholera are caused by an endotoxin produced by *P. multocida* (Heddleston, 1972). Managers seldom see sick birds among infected

wildfowl owing to a rapid death following onset of symptoms. At the agonal stage, convulsions and torticollis may occur. Several milliliters of a thick nasal discharge rich in *P. multocida* often are found among infected birds after death.

Internal lesions vary. Because birds die quickly, they often appear to be in good physical condition. Petechial and ecchymotic ("paintbrush") hemorrhages commonly occur on the myocardium and heart fat (Rosen, 1971). Focal necrosis may occur in the liver and other internal organs. Mucoid enteritis is often found, and this mucoid material may contain large numbers of *P. multocida*. Detailed descriptions of the signs, pathology and histologic changes associated with avian cholera are summarized by Rosen (1971) and Rhoades and Rimler (1984, 1989).

HISTORICAL BACKGROUND AND GEOGRAPHIC DISTRIBUTION

Avian cholera may have occurred in Italy as early as 1600, but was first confirmed in France in 1780 to 1782 (Gray, 1913). Among free-living birds, Sticker (1888) reported this disease from wild pheasants, presumably *Phasianus colchicus*, associated with domesticated pheasants in the vicinity of Berlin.

Epizootics of avian cholera among wild birds other than wildfowl (see Table 1) are uncommon, and the remaining focus of this review is on avian cholera among wildfowl and associated species. The first occurrence of avian cholera among wildfowl may be the mortality of about 40 wild Egyptian (*Alopochen aegyptiacus*) and spur-winged geese (*Plectropterus gambensis*) on Lake Nakuru, Kenya, in February 1940 (Hudson, 1959), although evidence that these birds died from avian cholera is indirect. Some geese were examined in the laboratory, but no bacteriological analysis was reported for them. But *P. multocida* was isolated from domestic birds dying a few days later on the lake (Hudson, 1959). Subsequently, avian cholera was reported among marine ducks, pelicans, and gulls in Chile in February 1941 (Suarez and Ilazabal, 1941); scientific names of affected birds were not provided.

Avian cholera is believed to have been introduced to the U.S. about 1880–1882 (Gray, 1913). The first known wildfowl epizootics in North America occurred during the winter of 1943–1944 in both Texas (Quortrup et al., 1946) and northern California (Rosen and Bischoff, 1949). The occurrence in California immediately followed an epizootic among domestic ducks. Currently, avian cholera occurs among all major flyways of North America (Friend, 1987).

Avian cholera was found in Holland among migrating mallards (*Anas platyrhynchos*) and green-winged teal (*A. crecca*), as well as gulls (*Larus* spp.) in September 1945; the epizootic occurred shortly before avian cholera was reported among domestic poultry of the same regions (Van den Hurk, 1946). It is not clear whether the rapid sequence of reports of avian cholera among the world's waterfowl resulted from a new and widespread awareness of the disease, or from a rapid introduction of avian cholera to the world's wildfowl. However, it seems likely that any earlier large epizootics of avian cholera among wildfowl would have been recognized, since the disease was well known among domestic birds.

Subsequently, avian cholera has been reported among gulls (*Larus dominicanus*) in South Africa (Kaschula and Truter, 1951), and wild ducks in Japan (Fujihara et al., 1986). Avian cholera occasionally has been reported among European waterfowl (Bezzel, 1979), but most reports among European wildlife are in doves, *Corvus* spp., and sparrows (Wetzel and Rieck, 1972).

Despite its occurrence on most major land masses, avian cholera seems best described as having a limited distribution and significance for most wildfowl populations. North American wildfowl are an exception. The disease occurs in all major flyways, and is a potentially serious problem for wild populations.

TABLE 1. The natural occurrence of *Pasteurella multocida* in free-living birds.[a]

Scientific name	Common name	First citation
Penguins		
Eudyptes chrysocome	Rockhopper penguin	de Lisle et al., 1990
Loons, Grebes		
Gavia immer	Common loon	Montgomery et al., 1979
Podilymbus podiceps	Pied-billed grebe	Klukas and Locke, 1970
Podiceps auritus	Horned grebe	Montgomery et al., 1979
Podiceps nigricollis	Eared grebe	Rosen, 1971
Aechmophorus occidentalis	Western grebe	Brogden and Rhoades, 1983
Pelicans		
Pelecanus occidentalis[b]	Brown pelican	Suarez and Ilazabal, 1941
Cormorants		
Phalacrocorax auritus	Double-crested cormorant	Montgomery et al., 1979
Wading Birds, Herons, Flamingo		
Ixobrychus exilis	Least bittern	Brogden and Rhoades, 1983
Ardea herodias	Great blue heron	Rosen and Bischoff, 1949
Casmerodius albus	Great egret	Raggi and Stratton, 1954
Egretta thula	Snowy egret	Oddo et al., 1978
Egretta caerulea	Little blue heron	Brogden and Rhoades, 1983
Phoenicopterus ruber	Greater flamingo	Brand and Duncan, 1983
Waterfowl—Swans		
Cygnus columbianus	Tundra swan	Rosen and Bischoff, 1949
Cygnus buccinator	Trumpeter swan	Gritman and Jensen, 1965
Cygnus olor[c]	Mute swan	Korbel, 1990
Waterfowl—Geese, Shelgeese		
Anser albifrons	Greater white-fronted goose	Rosen, 1969
Chen caerulescens	Snow goose	Rosen and Morse, 1959
Chen rossii	Ross' goose	Rosen, 1971
Branta canadensis	Canada goose	Petrides and Bryant, 1951
Alopochen aegyptiacus[d]	Egyptian goose	Hudson, 1959
Plectropterus gambensis[d]	Spur-winged goose	Hudson, 1959
Waterfowl—Ducks		
Aix sponsa	Wood duck	Rosen, 1971
Anas crecca[e]	Green-winged teal	Petrides and Bryant, 1951
Anas rubripes	American black duck	Vaught et al., 1967
Anas platyrhynchos[e]	Mallard	Quortrup et al., 1946
Anas acuta	Northern pintail	Quortrup et al., 1946
Anas discors	Blue-winged teal	Klukas and Locke, 1970
Anas cyanoptera	Cinnamon teal	Rosen, 1969
Anas clypeata	Northern shoveler	Rosen and Bischoff, 1949
Anas strepera	Gadwall	Vaught et al., 1967
Anas americana	American wigeon	Rosen and Bischoff, 1949
Netta peposaca[f]	Rosy-billed pochard	Fujihara et al., 1986
Aythya valisineria	Canvasback	Rosen and Bischoff, 1949
Aythya americana	Redhead	Wobeser et al., 1979
Aythya collaris	Ring-necked duck	Brogden and Rhoades, 1983
Aythya marila	Greater scaup	Rosen, 1971
Aythya affinis	Lesser scaup	Petrides and Bryant, 1951
Somateria mollissima	Common eider	Gershman et al., 1964
Clangula hyemalis	Oldsquaw	Locke et al., 1970
Melanitta nigra	Black scoter	Montgomery et al., 1979
Melanitta perspicillata	Surf scoter	Locke et al., 1970

TABLE 1. Continued.

Scientific name	Common name	First citation
Melanitta fusca	White-winged scoter	Locke et al., 1970
Bucephala clangula[g]	Common goldeneye	Locke et al., 1970
Bucephala albeola	Bufflehead	Montgomery et al., 1979
Mergus merganser	Common merganser	Brogden and Rhoades, 1983
Mergus serrator	Red-breasted merganser	Montgomery et al., 1979
Oxyura jamaicensis	Ruddy duck	Petrides and Bryant, 1951
Eagles, Hawks, Falcons		
Haliaeetus leucocephalus[h]	Bald eagle	Rosen, 1972
Circus cyaneus	Northern harrier	Rosen and Morse, 1959
Accipiter nisus[b,i]	Sparrowhawk	Jaksik et al., 1964
Buteo jamaicensis[f]	Red-tailed hawk	Brodgen and Rhoades, 1983
Aquila rapax[j]	Tawny eagle	Waddington, 1944
Aquila chrysaetos	Golden eagle	Rosen et al., 1973
Falco tinnunculus[k]	Eurasian kestrel	Curtis, 1979
Falco sparverius	American kestrel	Rosen, 1971
Gallinaceous Birds		
Perdix perdix[l]	Gray partridge	Jennings, 1954
Phasianus colchicus[l,m]	Ring-necked pheasant	Rosen and Morse, 1959
Lagopus lagopus[n]	Willow ptarmigan	Jennings, 1955
Bonasa umbellus	Ruffed grouse	Green and Shillinger, 1936
Callipepla californica	California quail	Hinshaw and Emlen, 1943
Meleagris gallopavo	Common turkey	Williams et al., 1987
Cranes, Rails, Coots		
Rallus aquaticus	Water rail	Korbel, 1990
Gallinula chloropus	Common moorhen	Brogden and Rhoades, 1983
Fulica americana	American coot	Rosen and Bischoff, 1949
Grus canadensis	Sandhill crane	Rosen, 1971
Shorebirds		
Vanellus vanellus	Northern lapwing	Curtis, 1979
Himantopus mexicanus	Black-necked stilt	Hirsh et al., 1990
Haematopus palliatus	American oystercatcher	Blus et al., 1978
Tringa melanoleuca	Greater yellowlegs	Brogden and Rhoades, 1983
Calidris minutilla	Least sandpiper	Rosen and Bischoff, 1949
Limnodromus scolopaceus[o]	Long-billed dowitcher	Brogden and Rhoades, 1983
Scolopax rusticola	Eurasian woodcock	Smit et al., 1980
Phalaropus sp.	Phalarope	Rosen and Bischoff, 1949
Skuas		
Catharacta skua	Great skua	Parmelee et al., 1979
Gulls		
Larus spp.	Gulls	Suarez and Ilazabal, 1941
Larus ridibundus	Common black-headed gull	Curtis, 1979
Larus canus	Mew gull	Brogden and Rhoades, 1983
Larus delawarensis	Ring-billed gull	Locke et al., 1970
Larus californicus	California gull	Rosen and Bischoff, 1949
Larus argentatus	Herring gull	Heddleston et al., 1972
Larus occidentalis	Western gull	Rosen and Bischoff, 1949
Larus dominicanus	Kelp gull	Kaschula and Truter, 1951
Larus glaucescens	Glaucous-winged gull	Rosen and Bischoff, 1949
Larus marinus	Great black-backed gull	Montgomery et al., 1979
Auks		
Uria aalge[p]	Common murre	Macdonald, 1963

TABLE 1. Continued.

Scientific name	Common name	First citation
Pigeons, Doves		
Columba palumbus[g,k]	Woodpigeon	Smit et al., 1980
Columba livia	Rock dove	Macdonald et al., 1981
Streptopelia decaocto	Collared dove	Smit et al., 1980
Owls, Nightjars		
Tyto alba	Common barn owl	Korbel, 1990
Otus asio	Eastern screech owl	Faddoul et al., 1967
Bubo bubo	Eagle owl	Jöst, 1915
Nyctea scandiaca	Snowy owl	Hunter, 1967
Athene noctua	Little owl	Smit et al. 1980
Athene cunicularia[f]	Burrowing owl	Brogden and Rhoades, 1983
Strix aluco	Tawny owl	Curtis, 1979
Asio flammeus	Short-eared owl	Rosen and Morse, 1959
Swifts		
Apus apus	Common swift	Korbel, 1990
Woodpeckers		
Picoides major	Great spotted woodpecker	Korbel, 1990
Colaptes auratus	Northern flicker	Wickware, 1945
	Woodpecker	Jaksic et al., 1964
Martins		
Delichon urbica	Common house-martin	Korbel, 1990
Jays, Crows		
Garrulus glandarius[b]	Eurasian jay	Jaksic et al., 1964
Pica pica[k]	Black-billed magpie	Windingstad et al., 1988
Corvus frugilegus	Eurasian rook	Novikov, 1954
Corvus corone[q]	Carrion crow	Keymer, 1958
Corvus brachyrhynchos	American crow	Zinkl et al., 1977a
Nuthatches		
Sitta europaea	Nuthatch	Korbel, 1990
Thrushes, Thrashers		
Muscicapa striata	Spotted flycatcher	Curtis, 1979
Erithacus rubecola[q]	Robin	Keymer, 1958
Turdus merula[q,r]	Eurasian blackbird	Keymer, 1958
Turdus pilaris	Fieldfare	Korbel, 1990
Turdus philomelos	Song thrush	Smit et al., 1980
Turdus migratorius	American robin	Bivins, 1955
Mimus polyglottos	Northern mockingbird	Heddleston, 1976
Waxwings, Starlings		
Bombycilla cedrorum	Cedar waxwing	Locke and Banks, 1972
Sturnus vulgaris	European starling	Bivins, 1953
Sparrows		
Passerculus sandwichensis	Savannah sparrow	Brogden and Rhoades, 1983
Zonotrichia leucophrys[q]	White-crowned sparrow	Snipes et al., 1988
Blackbirds, Orioles, House Sparrow		
Euphagus cyanocephalus[q]	Brewer's blackbird	Snipes et al., 1988
Quiscalus quiscula	Common grackle	Bivins, 1955
Icterus galbula	Northern oriole	Faddoul et al., 1967
Passer domesticus	House sparrow	Heddleston, 1976
Finches		
Carduelis chloris	European greenfinch	Korbel, 1990
Carduelis pinus[f]	Pine siskin	Heddleston et al., 1972
Coccothraustes vespertinus	Evening grosbeak	Faddoul et al., 1967

IMPACT OF AVIAN CHOLERA ON WILDFOWL POPULATIONS

Efforts to identify the impact of avian cholera on North American wildfowl are, at best, incomplete. This is made more difficult by the inconsistency in the annual severity of the disease among North American populations. Rosen (1971) estimated that 2% fewer ducks and 6% fewer swans migrated northward to the breeding grounds in some years, as a result of avian cholera. However, these mortality figures seem high, even for wildfowl populations in California, where the disease is a recurrent annual problem.

Known avian cholera losses in California alone typically range from 10,000 to 25,000 birds each year, and were estimated to reach 70,000 in the winter of 1965–1966 (Titche, 1979). Even the 70,000 deaths reported for 1965–1966 would encompass less than 1% of the ducks in California. Rosen (1972) gave specific mortality estimates for the 1970–1971 epizootic in California; of about 7.5 million wildfowl wintering in California, approximately 0.2% of the ducks, 1.5% of the geese, 3.9% of the swans and 2.4% of the American coots (*Fulica americana*) died from avian cholera. When one considers estimated avian cholera mortality for all North American wildfowl, it probably is substantially lower than the earlier estimates given by Rosen (1971), particularly for ducks.

While avian cholera is an important mortality factor among wildfowl, it probably is less important than habitat destruction and hunting. Nevertheless, on some sites, losses from avian cholera surpass those of local hunting mortality (Moore and Simpson, 1981; Sacramento Valley National Wildlife Complex records, U.S. Fish and Wildlife Service, Willows, California 95988, USA; Mensik, 1986). More importantly, avian cholera is one additional mortality factor of an unpredictable impact that wildfowl populations must face annually.

CHARACTERISTICS OF AVIAN CHOLERA EPIZOOTICS

Avian cholera epizootics usually are explosive in wildfowl, appearing with little or no warning (Rosen and Bischoff, 1949; Rosen and Morse, 1959). While this is usually the case among domestic birds as well, some variation is known. Hendrickson and Hilbert (1932) found that *P. multocida* was

←

[a] Taxonomy based on American Ornithologists' Union (1983) checklist, and Clements (1981).
[b] Probable host identification; bird species not definitively identified in text.
[c] Also reported by Jennings and Soulsby (1956), but species of *Pasteurella* not clearly identified, and basis of bacterial identification not presented.
[d] Probable host for *Pasteurella multocida*; birds examined in lab, but not clear that *P. multocida* isolated. Bacteriological diagnosis of avian cholera made in domestic birds dying in the same area a few days later.
[e] Van den Hurk (1946) described avian cholera-like lesions in this species and observed bacilli in blood smears with a methylene blue stain, but did not report isolating *P. multocida*.
[f] Not clearly established as a free-living bird.
[g] Macdonald (1963) diagnosed pasteurellosis (fowl cholera) in this species, but provided no basis for bacterial identification.
[h] Coon et al. (1970) probably isolated *P. multocida* from this species, but bacterial identification was incomplete. Locke et al. (1972) also reported *P. multocida* from a bald eagle.
[i] Januschke (1915) earlier isolated *P. multocida* from a probable captive bird of this species.
[j] Waddington (1944) described avian cholera-like lesions in this species and observed bipolar staining bacilli in the blood and bone marrow, but did not attempt to isolate *P. multocida*.
[k] Jaksic et al. (1964) probably found *P. multocida* in this species earlier, but their bird identification was not definitive.
[l] Sticker (1888) reported avian cholera from this species; however, the bacteria identification was not definitive and it is not clear that the birds were wild.
[m] Shillinger and Morley (1942), and Hudson (1944) reported avian cholera among pheasants earlier, but either the birds were not clearly wild, or the basis of identifying *P. multocida* was lacking.
[n] Klein (1889) described bacteria from this bird species which Gratzl and Köhler (1968) interpret to be *P. multocida*.
[o] Although reported as a captive bird by Brogden and Rhoades (1983), the date and site given suggest this to be the same free-living bird later reported by Mensik and Botzler (1989).
[p] Basis of bacterial identification not clear, and species of *Pasteurella* not clearly identified.
[q] Apparently healthy, or died from causes other than pasteurellosis.
[r] Later case report of death from *P. multocida* (Curtis, 1979).

detectable in the blood of domestic chickens up to four days prior to their death from avian cholera, and there was a progressive increase in the numbers of bacteria in the blood up to the time of death; *P. multocida* was observed in the blood of two chickens for 18 and 47 days, respectively, before they died from avian cholera. Among wildfowl it has been noted that *P. multocida* strains were less virulent at the end of some epizootics, as evidenced by the increasing presence of sick birds (Rosen and Bischoff, 1949; Rosen and Morse, 1959).

Most reported avian cholera mortality among wildfowl probably occurs during extensive epizootics, involving hundreds to thousands of birds. However, there also is evidence that many smaller pockets of mortality occur which never develop to extensive epizootics (Bivens, 1953, 1955; Macdonald, 1963, 1965; Rosen, 1969, 1972; Blus et al., 1978; Wobeser, 1981; Humburg et al., 1983; Brand, 1984). In Humboldt County, California, avian cholera incidents involving less than 60 wildfowl were observed on five occasions between 1972 and 1990; on three of those occasions only a single bird with avian cholera was observed, at sites containing large numbers of susceptible wildfowl (R. Botzler, unpubl.)

On evaluating avian cholera epizootics in California, Rosen (1969) calculated a relative mortality index by comparing the observed mortality of each given species in an epizootic, to the estimated occurrence of that species in the live populations on a site. Based on this index, he found that some species suffered disproportionately greater mortality than others. For example, compared to mallards over a 9-yr period, American coots, American wigeon (*Anas americana*), white-fronted geese (*Anser albifrons*), snow geese (*Chen caerulescens*), cackling Canada geese (*Branta canadensis minima*), and northern shovelers (*Anas clypeata*) often suffered a disproportionately high mortality, whereas northern pintails (*Anas acuta*), green-winged teal (*Anas crecca*), and ruddy ducks (*Oxyura jamaicensis*) often suffered comparatively less. But Rosen (1969) found that this relative species susceptibility could vary considerably between years.

There may be greater consistency in mortality by species at a single site. For example, at Centerville on the north coast of California, American coots consistently made up a greater proportion (>80%) of the total wildfowl mortality than one would expect based on their proportion (45 to 55%) in the total live wildfowl populations; in contrast, duck species consistently died in fewer numbers than one would expect, based on their live populations in the area (Hazlewood et al., 1978; Oddo et al., 1978; Mensik and Botzler, 1989).

Another characteristic reported for avian cholera among wildfowl is sequential mortality. During two epizootics at Centerville, in California, American coots died first, followed by deaths among swans and American wigeon; northern pintails, northern shovelers, mallards and teal died late in the epizootics (Fig. 1) (Oddo et al., 1978; Mensik and Botzler, 1989). At a different site in California, Rosen and Morse (1959) noted that American coots were the first and principal species affected, and that the disease spread next to other ducks, primarily American wigeon. American coots also appeared to be the first species to die during epizootics in the Everglades of Florida (Klukas and Locke, 1970) and Chesapeake Bay (Pursglove et al., 1976). Brand (1984) noted that mortality in snow geese was followed by deaths among mallards at two sites evaluated in the Central and Mississippi flyways.

HOST FACTORS AFFECTING SUSCEPTIBILITY TO AVIAN CHOLERA

Host susceptibility to a disease may be influenced by such features as sex, age, other physical characteristics, genetic variation, behavioral differences among the hosts, and variation in immune status re-

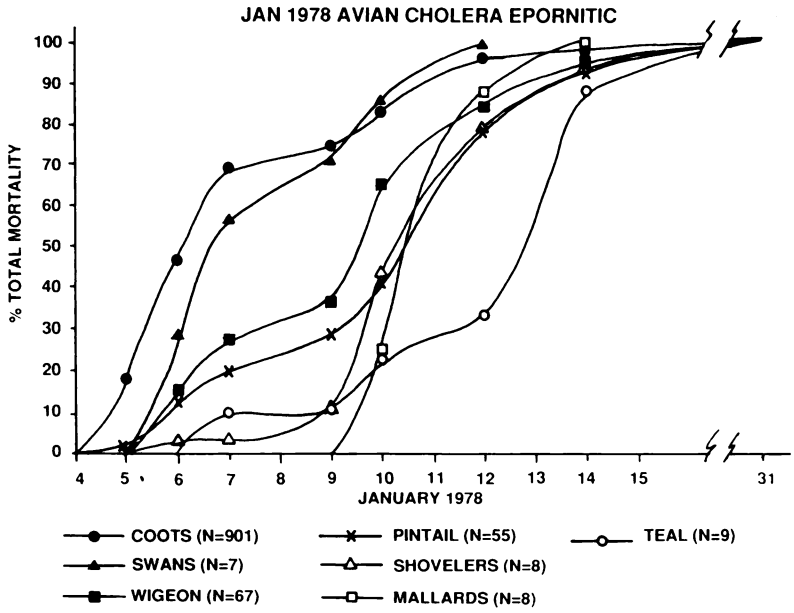

FIGURE 1. Sequence of mortality observed during the January 1978 avian cholera epizootic, Centerville, Humboldt County, California, USA.

sulting from past exposures to infective agents.

Host sex has been considered a possible predisposing factor to avian cholera. Korschgen et al. (1978) found that over 90% of observed mortality among common eiders (*Somateria mollissima*) in Maine (USA) occurred among nesting females; however, there is no evidence that the males, which move offshore after mating, were ever monitored (L. Locke, pers. comm.). Montgomery et al. (1979) found that more male than female diving ducks succumbed to avian cholera during an epizootic on the Chesapeake Bay, but speculated that more males occurred in the live population at that time. McLandress (1983) observed a higher frequency of male Ross' (*Chen rossii*) and male lesser snow geese (*C. caerulescens caerulescens*) among birds dying from avian cholera, compared to hunter-killed birds. Mensik and Botzler (1989) found no significant differences in the sex ratios of coots dying from avian cholera and those killed by gunshot. Based on these limited studies, the role of host sex may be significant in some cases, but has no consistent effect.

Host age may affect susceptibility to avian cholera. Hoffman and Stover (1942) found the prevalence of avian cholera among chickens to be directly correlated with age; *P. multocida* was reported in <0.1% of all necropsied chickens under 12 wk of age, and peaked at 9.4% of all diagnosed diseases for chickens ≥2 yr old. Hungerford (1968) found that chickens under 16 wk of age were not susceptible by cloacal inoculation to doses of *P. multocida* that consistently killed older birds.

Among wildfowl, Rosen and Bischoff (1950) reported that 4-wk-old ducklings (no species given) died from avian cholera. Hunter and Wobeser (1980) observed that 16- and 18-wk-old mallard ducks were less susceptible to avian cholera than 5- and 11-wk-old ducklings. Mensik and Botzler (1989) found no significant difference in the age structures between American coots that were shot and those dying from avian cholera. Overall, there is no consistent evidence among wildfowl for differential susceptibility to avian cholera by host age.

To date there is no consistent evidence that susceptibility to avian cholera varies consistently with the physical features of

the host. Petrides and Bryant (1951) reported that susceptibility to avian cholera in Texas was correlated to the average species weight of affected wildfowl, but Rosen (1969) was unable to confirm this observation in California. Comparing 19 physical features between American coots dying from avian cholera, and apparently healthy coots that were shot, Mensik and Botzler (1989) found significant differences only among heart, liver, and spleen weights; the authors believed these differences were due to sequelae of the disease rather than prior differences existing between the birds.

Host density may be a predisposing factor to avian cholera epizootics. One can distinguish two levels at which host density may influence the effects of disease: an increased risk of an epizootic starting as the density of a susceptible host population increases, as well as a higher frequency of deaths in more dense host populations (density-dependent mortality). Overcrowding of wildfowl in relation to available habitat, drought conditions, faulty water management, or inclement weather often have been speculated to increase wildfowl susceptibility to avian cholera (Petrides and Bryant, 1951; Vaught et al., 1967; Rosen, 1969; Klukas and Locke, 1970; Zinkl et al., 1977a). Van Es and Olney (1940) reported that epizootics among domestic birds increased when their densities increased. Combs (1988) reported that wildfowl species maintaining larger flock sizes died earlier in avian cholera epizootics than species with small flock sizes, suggesting that increased host density may increase susceptibility to avian cholera.

Rosen (1969) compared population estimates of live tundra swans (*Cygnus columbianus*) with tundra swan mortality from avian cholera over a 13-yr period in California's Central Valley, and concluded that avian cholera functioned in a density-independent fashion. One shortcoming of this comparison was that it presumed an even distribution of swans over the study area; no effort was made to monitor local variations in swan densities over this 13-yr period. While the total area covered in these surveys was similar, there was no mention of annual variation in available habitat as determined by rainfall and local flooding. Likewise, it ignored the role of other waterfowl populations in contributing to a density-dependent effect of the disease.

Susceptibility to avian cholera varies among bird species, both in the laboratory and in the field. Some variation may be due to genetic differences between species. Among domestic animals, chickens and turkeys vary greatly in susceptibility to *P. multocida* when inoculated by the nasal cleft (Heddleston and Watko, 1965) and the palatine route (Rosen, 1969). Using intraperitoneal injections, Rosen (1969) calculated the LD_{100}'s for gulls (*Larus* sp.) and American coots to be $10^{8.7}$ and $10^{2.7}$ *P. multocida*, respectively. However, the intraperitoneal route is not likely to be involved in natural transmission of *P. multocida*. Also, LD_{100} calculations can vary greatly with the presence of one or a few very resistant host animals.

Rosen (1969) also suggested that differences in behavior and habitat use may affect susceptibility to avian cholera. Combs (1988) assessed wildfowl behavior and habitat use in relation to susceptibility to avian cholera at the Centerville site in California and found that wildfowl at greatest risk to avian cholera used land areas together during the avian cholera season (January), and commonly grazed on land or at the water surface.

In susceptible hosts, the presence of other infections may be a predisposing factor to infection with *P. multocida* (Eveleth et al., 1949; Hutyra et al., 1949; Collins, 1977). Rimler and Rhoades (1986) found that exposure to *Bordetella avium* reduced the systemic immunity conferred by an avian cholera vaccine in turkeys.

ENVIRONMENTAL FACTORS AFFECTING EPIZOOTICS

Among North American wildfowl, avian cholera initially was described as a win-

ter disease (Rosen, 1971). More recently it has been observed on the spring staging grounds in Nebraska, USA (Zinkl et al., 1977a; Windingstad et al., 1984, 1988), and during spring migration in Saskatchewan (Wobeser et al., 1979). Wildfowl epizootics in both Kenya (Hudson, 1959), and Chile (Suarez and Ilazabal, 1941) occurred during the summer (February). Avian cholera also has been reported as an early summer disease among nesting common eiders in Maine and in the St. Lawrence Seaway region (Gershman et al., 1964; Reed and Cousineau, 1967; Korschgen et al., 1978), and as a late summer disease of snow geese on their breeding grounds (Brand, 1984). Based on published and unpublished accounts, avian cholera now has been reported in every month of the year among North American wildfowl. While most reports of avian cholera still are made on winter habitat or spring staging grounds, the concentrations of birds at these sites make mortality more noticeable, and thus do not necessarily reflect the true seasonal distribution of cases. There have been no systematic studies of wildfowl mortality along migratory routes or on the breeding grounds.

The relationship between precipitation and avian cholera epizootics is inconsistent. Among domestic poultry, Hoffman and Stover (1942) found a direct correlation between rainfall and avian cholera mortality over a 10 yr period in California. Rosen and Bischoff (1949) stated that this relationship continued to exist when the data were extended to 20 yr, but provided no supporting information.

Wildfowl epizootics in northern California generally have begun at the start of the rainy season. Rosen and Bischoff (1950) believed the severity and spread of avian cholera was related to precipitation, but presented no supporting data. Rosen (1972) later suggested that an absence of rain and the resulting concentration of waterfowl contributed to unusually high mortality during the 1970–1971 epizootics in California. However, until careful monitoring of both rainfall and mortality is done over a period of years on several sites, this issue will remain unresolved.

Frankowski (1967) found that pasteurellosis among cattle, pigs, and fowl was correlated with stressful weather conditions. Collins (1977) reported that *P. multocida* infections in mammals often occurred only after some stress such as cold temperatures. After evaluating past case reports, Eveleth et al. (1949) inferred that changes from moderate to hot or cold weather, the occurrence of rain or snow, and changes in water supply served as predisposing factors to avian cholera epizootics in domestic fowl.

Ambient temperature may affect susceptibility to avian cholera. Simensen et al. (1980) observed a later onset and lower mortality among inoculated turkeys housed at 33 to 37 C, compared to turkeys held at 22 C or lower. Among wildfowl, Rosen and Bischoff (1950) reported that chilling did not favor infection with *P. multocida* in coots, but provided no supporting data. Windingstad et al. (1988) noted two peak periods of avian cholera mortality in Nebraska, each of which followed several days of very cold temperatures. Interestingly, Bredy and Botzler (1989) found that *P. multocida* had a very poor survival in water held at 4 C, compared to water held at 20 C. Overall, the relationship of ambient temperatures to the occurrence of avian cholera among wildfowl is ambiguous.

Nutritional stress has been suggested as a predisposing factor to avian cholera. Eveleth et al. (1949) claimed that domestic birds were more susceptible to a given dose of *P. multocida* after fasting for 48 hr than if well-fed, but presented no supporting data. Korschgen et al. (1978) speculated that food stress among nesting female common eiders during incubation exacerbated latent *P. multocida* infections, and caused the disease to spread to other susceptible eiders in the area. However, most wild birds dying during avian cholera epizootics are in good condition (Friend,

1987; Mensik and Botzler, 1989), suggesting that nutritional deficiencies do not play an important role as a predisposing factor among wildfowl.

The interactions of *P. multocida* with other microorganisms have not been assessed. In water, Bredy and Botzler (1989) observed increased survival and growth of *P. multocida* after introduction of other microorganisms.

Water quality may affect susceptibility to avian cholera (Eveleth et al., 1949). For example, no avian cholera has been observed at Centerville, California, since 1979, coinciding with a change in the water source used to flood the waterfowl ponds (R. Botzler, unpubl.). Windingstad et al. (1984) found differences in specific conductance, calcium, magnesium, chloride, sulfate and sodium levels between areas affected by avian cholera, and areas not experiencing epizootics in the Rainwater Basin of Nebraska. Windingstad et al. (1988) quoted J. Price (National Wildlife Health Research Center) as noting that some of these factors may influence the survival of *P. multocida* in water. Later, Gordon (1989) showed that many of the differences observed by Windingstad et al. (1984) persisted over time at these sites.

Survival of *P. multocida* is enhanced by animal organic matter. Hendrickson and Hilbert (1932) found that *P. multocida* survived in chicken carcasses for 11 days at room temperature and for at least two months when placed in an icebox; the temperatures of neither the room nor the icebox were reported. Rosen and Bischoff (1950) reported persistence of *P. multocida* for 120 days in buried coot hearts. Olson and Bond (1968) isolated *P. multocida* for 60 days from turkey carcasses. Titche (1979) found that *P. multocida* survived longer in marsh water when carcasses were present. Following an avian cholera epizootic in Nebraska, S. Hurley (1979, National Wildlife Health Research Center unpublished files) found that *P. multocida* remained on the site as long as organic animal matter was present, generally not more than 5 wk. Bredy and Botzler (1989) found that the addition of animal protein significantly enhanced the survival of *P. multocida* in water.

There is little information on whether environmental contaminants are predisposing factors to avian cholera. Pier (1981) provides a general overview on the immunosuppressive effects of aflatoxins. Friend and Trainer (1970) suggested a connection between avian cholera and insecticide use in California. Rocke et al. (1984) reported that resistance to challenge by *P. multocida* was significantly lowered among mallards exposed to sublethal levels of fuel oil, crude oil, and fuel oil mixed with an oil dispersant.

The immunosuppressive effects of lead are well known (Franson, 1986). In a field study in Central California, Gordus (1985) compared lead levels per dry weight of tissue between snow geese dying from avian cholera and hunter-killed geese; lead levels were insignificantly lower both in kidneys ($P < 0.10$) and livers ($P < 0.30$) of birds dying from avian cholera. For all birds collected, Gordus (1985) also compared the frequency with which avian cholera occurred among geese with ≤ 30 ppm lead, and among geese with >30 ppm lead; there was no significant difference in the frequency with which *P. multocida* was isolated from geese in these two groups. In a laboratory study, Wobeser (1986) also reported slightly less mortality from avian cholera among mallards dosed with lead shot, compared to birds ingesting steel shot or receiving no shot. Additional work is needed to clarify the relationship between exposure to lead and susceptibility to avian cholera.

There has been little effort to understand the epizootiology of avian cholera in the context of habitat characteristics and land use. Brown et al. (1983) found that vegetation at an avian cholera site in Nebraska was characterized by low species diversity, while a site with little avian cholera had high species diversity. In contrast, Gordon (1989) found no apparent pattern

between the occurrence of avian cholera and variation in emergent vegetation in Nebraska. In the same areas, Smith et al. (1989) found no relation between avian cholera mortality and surface water drainage among wetlands, wetland classifications, and land use.

RESERVOIRS OF *PASTEURELLA MULTOCIDA* FOR WILDFOWL

The term "reservoir" is defined as a dependable, nonclinical source of a pathogen; a place where the infective agent can survive on a year-round basis. Two major reservoirs have been proposed for avian cholera in wildfowl: ambient soil or water of enzootic sites, and carrier birds.

Kitt (1887) noted many cases of avian cholera in dometic bird flocks where no sick animals had been introduced. He concluded that *P. multocida* was a saprophytic organism, independent of animal association.

Among wildfowl, the consistent recurrence of avian cholera at some sites such as the Muleshoe National Wildlife Refuge in Texas (USA) and in northern California could be interpreted as evidence that soil or water might be an important reservoir. This view was strengthened by Rosen's (1969) observation that avian cholera may act as a density-independent mortality factor among tundra swans of California.

The survival and growth of *P. multocida* in soil has been assessed by several authors. Hutyra et al. (1949) referred to a study by T. Kitt in which *P. multocida* survived three months in garden soil. Dimov (1964) found that *P. multocida* survived up to 113 days in soils held at 3 C, and from 15 to 100 days in soils held at 20 C. Olson and Bond (1968) found that *P. multocida* survived up to 21 days in soils held at 26 C, and 18 days in soils stored at 4 C. Awad et al. (1976) reported survival of *P. multocida* in soil for 27 days. Backstrand and Botzler (1986) found that *P. multocida* survived less than 20 days after inoculation into soil of an enzootic site during an avian cholera epizootic. In the same study, no *P. multocida* were recovered from uninoculated soils natural to the area before, during, or after an avian cholera epizootic occurring on this site.

Rosen and Bischoff (1950) buried the hearts of coots dying from avian cholera in soil; avirulent *P. multocida* could be recovered after 120 days, but not after 240 days. They speculated that survival of *P. multocida* in soil was inversely related to its virulence. Little information about the physical or chemical characteristics of the soils used was presented in these studies. Taken as a whole, there is little evidence to support the hypothesis that soil can serve as a year-round reservoir of *P. multocida*.

Water bodies of enzootic avian cholera sites may be a reservoir of *P. multocida*. Natural waters may contain high levels of *P. multocida* during epizootics; Rosen and Bischoff (1949) observed that 0.5 ml of water from a pond with an ongoing epizootic contained enough virulent *P. multocida* to kill a mouse in four hours after intraperitoneal injection.

The survival of *P. multocida* in water has been assessed by numerous authors. Hutyra et al. (1949) reported a study in which *P. multocida* survived in water with the exclusion of air for 18 days at 5 to 6 C. On one occasion, Rosen (1969) observed that *P. multocida* was recovered from water 3 wk after 100 snow geese died on a pond, with no waterfowl being observed on the area in the intermittent period. Bendheim and Even-Shoshan (1975) observed that at 18 C, *P. multocida* survived for 3 days in distilled water, 12 days in tapwater, 28 days in deionized water, and 99 days in water contaminated with turkey litter. Titche (1979) found that survival of *P. multocida* ranged from 6 days in "saline" to 30 days in marsh water adjacent to an opened carcass of a duck dying from avian cholera.

At Centerville, California, *P. multocida* survived in water for 13 days beyond the end of one epizootic (R. G. Botzler, unpubl.). In a later study on this site, *P. mul-*

tocida was not recovered from natural pond water 4 days before an avian cholera epizootic, but was isolated at 3 and 10 days after the epizootic started; it was not isolated at 17 days or later (Backstrand and Botzler, 1986). In the same study, *P. multocida* survived less than 6 days in pond water inoculated with the organisms (Backstrand and Botzler, 1986).

Except for Bendheim and Even-Shoshan (1975), who used tryptose blood agar base, all tests for *P. multocida* in water were by mouse inoculation. However, Rosen (1972) found that Das' medium was superior to mouse inoculation in some cases for establishing the presence of *P. multocida*.

Bredy and Botzler (1989) found that *P. multocida* could survive for >1 yr in water under specific conditions, including warm terperatures (18 to 20 C), the addition of protein, 0.5% NaCl, and the presence of other microorganisms; in contrast, variation in pH, clay content and sucrose level had little effect on survival of the pasteurellae. It is not clear how pertinent these findings are to the year-round survival of *P. multocida* in water of natural wildfowl habitats. Overall, the majority of studies do not support the hypothesis that water is an important reservoir for *P. multocida*, but the issue is not yet resolved.

Carrier animals may be an important reservoir. *Pasteurella multocida* occurs in the respiratory tracts and mouths of a great variety of mammals. Examples include the respiratory tracts of domestic sheep (Foreyt and Jessup, 1982), the mouths of Norway rats (Schipper, 1947), domestic dogs and cats (Owen et al., 1968; Hubbert and Rosen, 1970; Arnbjerg, 1978), as well as from numerous wild mammals (Owen et al., 1968; Rosen, 1970; Bond et al., 1972; Quan et al., 1986).

Pasteurellosis in both mammals and birds can be initiated by the bites of infected mammals. Human cases of *P. multocida* infection commonly have followed bites of dogs, cats and other mammals (Hubbert and Rosen, 1970). Bergerud (1971) showed that orally infected lynx (*Felis lynx*) transmitted *P. multocida* to caribou (*Rangifer caribou*) calves during unsuccessful predation attempts. Similarly, Smit et al. (1980) reported the transmission of *P. multocida* to birds by cat bite. Korbel (1990) found *P. multocida* infections in 54% of 92 wild birds that had been bitten by cats. Gregg et al. (1974) found that domestic turkeys could be infected with *P. multocida* through the bite of raccoons (*Procyon lotor*) and speculated that wild racoons might serve as reservoirs of *P. multocida* for turkeys that move onto open rangelands. Rhoades and Rimler (1989) pointed out that among *P. multocida* originating from mammals, isolates from cattle and sheep were nonpathogenic for poultry, whereas those from pigs, cats, rats and mice were considered possible sources of avian cholera.

Quan et al. (1986) evaluated 243 *P. multocida* isolates collected from culturing tissues from wild rodents, lagomorphs, and carnivores, as well as flea pools from these animals, over a 12-yr period during sylvatic plague studies. It is of interest that most *P. multocida* isolants were serotypes 1A and 3A, the same serotypes generally found in wildfowl during avian cholera epizootics.

After isolating *P. multocida* serotype 1 during an avian cholera epizootic in Nebraska, Windingstad et al. (1988) isolated *P. multocida* from nasal swabs in 35 of 37 feedlot cattle. Eighty percent of the *P. multocida* isolates from cattle were serotype 3 or serotype 3 with cross-reactivity; only one weak serotype 1 cross-reactor was found. No *P. multocida* was recovered from cloacal or pharyngeal swabs among 20 domestic ducks and geese on a farm adjacent to the wildfowl epizootic. Thus, there appeared to be little connection between domestic animals and the occurrence of avian cholera in wildfowl examined during this study.

Pritchett et al. (1930a, b) and Pritchett

and Hughes (1932) found that a high frequency of recovered chickens became carriers, and proposed that birds surviving avian cholera from 1 yr provided the reservoir for the next year's epizootics. Using radio-labelled *P. multocida*, Iliev et al. (1965a) found that orally ingested pasteurellae were inactivated in the proventriculus of fowl. They also concluded that feces from established carrier birds could not maintain *P. multocida* and were unimportant sources of environmental contamination; rather, established carriers were significant sources of *P. multocida* only through oral and nasal discharges, especially into water. Iliev et al. (1965b) further noted that fowl infected through their oral mucous membranes could shed nonpathogenic *P. multocida* of mammalian origin for 50 to 60 days. In contrast, fowl similarly infected with nonpathogenic *P. multocida* of avian origin could become permanent carriers, but the *P. multocida* never reverted to pathogenic strains in the infected birds.

Heddleston (1972) believed that the life of a carrier bird was the only limit to the duration of a chronic carrier state in domestic fowl. Among domestic fowl, Curtis and Ollerhead (1981) inferred that infected carrier birds occurred only in flocks with a past history of avian cholera.

Heddleston and Watko (1963) assessed a variety of wildlife, farm animals and laboratory animals as potential carriers of *P. multocida*, and as potential sources of avian cholera for domestic birds. They found that pigeons, sparrows, mice, and rabbits died of acute septicemia after intranasal exposure to a *P. multocida* strain isolated from a chicken. Rats, ferrets, guinea pigs, a sheep, a pig and a calf had no noticeable response after intranasal infections, but the pig and calf had inapparent *P. multocida* infections in their nasal passages when tested 34 days after inoculation. After being fed viscera from chickens dying from avian cholera, one of five rabbits developed a nasal infection, one of two mink developed pneumonia, and 11 of 19 mice contracted a fatal septicemia. Thus, *P. multocida* appeared to transmit readily between domestic birds, wildlife, and a variety of other animals.

Serdyuk and Tsimokh (1970) infected sparrows, pigeons, and rats with *Pasteurella* sp., and were able to transmit avian cholera from infected to healthy chickens in each of the chicken-wildlife-chicken sequences. None of the sparrows and pigeons carrying the organisms developed signs of infection, whereas 10% of the infected rats developed pasteurellosis. They suggested that wild birds or rodents might be a reservoir for domestic poultry.

Snipes et al. (1988) reported *P. multocida* from a variety of wild mammals and birds on turkey farms experiencing avian cholera epizootics in the preceding two to eight months. The somatic serotypes of isolates from wildlife were the same as those affecting the turkeys on only two of seven premises checked, suggesting little connection between the avian cholera in turkeys and *P. multocida* in wildlife in this study.

Rhoades and Rimler (1984) suggested that wildfowl carriers may be a source of *P. multocida* for domestic birds. However, the role of carriers among wildfowl is not clearly established. Quortrup et al. (1946) reported that a wild-caught mallard resistant to challenge with *P. multocida* transmitted avian cholera to birds caged with it; the authors believed the exposed bird was shedding *P. multocida*. Rosen (1972) reported a snow goose that survived an apparent case of avian cholera but died of the disease 3 mo later at his laboratory, suggesting an intermittent carrier state.

Healthy appearing wildfowl have been evaluated for the presence of *P. multocida*. Vaught et al. (1967) isolated *P. multocida* from the spleens and livers of three apparently healthy coots in Missouri. However, in another Missouri study, Donahue and Olson (1969) could not isolate *P.*

multocida from nasal pharynxes of 400 wild waterfowl from the Mississippi Valley. There is some question, however, if nasal swabs are a good source of *P. multocida* in carrier animals (Wobeser, 1981).

Korschgen et al. (1978) isolated *P. multocida* from the tissues in only one of 236 apparently healthy common eiders, and from the oropharynx in one of 357 apparently healthy nesting female eiders. Titche (1979) isolated *P. multocida* from 16 (9.3%) of 172 apparently healthy wildfowl after injecting macerated lung tissue from these birds into white mice, suggesting that wildfowl carriers might be more common than previously believed. Titche (1979) later isolated *P. multocida* type 3 from 3 of 41 apparently healthy wild ducks near a waterfowl facility housing wild birds undergoing an avian cholera epizootic from type 3 *P. multocida*. Titche (1979) believed the infection may have been introduced to the wildfowl facility by infected carrier birds.

Gulls may be reservoirs of *P. multocida* (Rosen and Bischoff, 1950; Gershman et al., 1964; Korschgen et al., 1978). Titche (1979) isolated *P. multocida* from 15 of 37 California gulls (*Larus californicus*), but from none of 23 ring-billed gulls (*Larus delawarensis*) in California. In the same study, he noted that six of nine California gulls fed *P. multocida*-contaminated meat shed the *P. multocida* in their feces for up to 120 hr; *P. multocida* also was recovered from three gulls by oral swabs.

Szécsényi (1965) and Zinkl et al. (1977a) proposed that crows (*Corvus* spp.) are carriers of *P. multocida*. American crows (*C. brachyrhynchos*) were observed dead during an avian cholera epizootic in waterfowl; serotype 1 *P. multocida* was isolated from both dead crows and waterfowl (Zinkl et al., 1977a). Sanders (1938) exposed fish crows (*Corvus ossifragus*) to *P. multocida* by the eye and nasal cleft, and several birds lived up to 66 days with chronic *Pasteurella* infections in their posterior nares.

Overall, most biologists seem to favor the hypothesis that recovered carriers are the most important reservoir for avian cholera. Rosen and Morse (1959) inferred that sick birds or carriers transmitted avian cholera from Grizzly Island to Lower Klamath National Wildlife Refuge, Oregon, a distance of 275 air-miles (440 km). McDiarmid (1962) reported isolating *P. multocida* from a swallow (probably *Hirundo rustica*, no species given) that arrived in England from Africa; he suggested migration as a means of introducing this pathogen. Novikov (1954) reported avian cholera among migrating rooks (*Corvus frugilegus*) in the Soviet Union; avian cholera among domestic birds began shortly after the rook mortality.

Rosen (1972) speculated that waterfowl carry *P. multocida* north to the nesting grounds, and sustain mortality there too. Wobeser et al. (1979) provided supporting evidence by observing avian cholera among snow geese and Ross' geese during their spring migration in Saskatchewan. Avian cholera has been observed on the breeding grounds of common eiders (Gershman et al., 1964; Reed and Cousineau, 1967; Korschgen et al., 1978) and snow geese (Brand, 1984).

In one intriguing study, Brand (1984) found that avian cholera mortality in the Central and Mississippi Flyways closely followed the migration patterns of snow geese, starting from Hudson Bay; but no attempt was made to isolate *P. multocida* from the apparently healthy snow geese in this study. It has been noted that avian cholera at the Sacramento Valley Complex usually follows the arrival of snow geese (J. G. Mensik, Sacramento National Wildlife Refuge, pers. comm.).

Later, C. Brand and P. Whiteley (1988 unpublished report, National Wildlife Health Research Center files, Madison, Wisconsin) could not induce a carrier state among 101 mallards inoculated with *P. multocida*, even after exposing them to cyclophosphamide or dexamethsone. Collins (1984) induced the persistence of *P. multocida* among mice by using low doses of aztreonam.

There are still ambiguities on the respective contributions of soil or water, as

well as carrier animals. Even when *P. multocida* was isolated from carrier animals or soil and water, these isolates often were not serotyped or inoculated into susceptible birds to determine whether they could cause avian cholera. Many strains of *P. multocida* do not cause the disease in wildfowl. Much additional work is needed to clearly establish the reservoir of *P. multocida*.

TRANSMISSION

Once a clinical infection is established in susceptible animals, transmission through a susceptible population has been hypothesized to occur in several ways, including arthropods, inhalation and ingestion.

The soft tick, *Argas persicus*, can transmit *P. multocida* among domestic birds (Basu, 1930; Iovchev, 1967). Petrov (1970) found that *P. multocida* survived 33 days at 30 C and 100 days at 4 C in *A. persicus*. Metwalley et al. (1978) found that *P. multocida* survived up to 31 days in *A. persicus* at room temperature. Glukhov and Novikov (1975) reported a 1,000-fold increase of *P. multocida* in *A. persicus*.

Poultry mites (*Dermanyssus gallinae*) taken from rabbits dying from pasteurellosis contained *P. multocida* (Bigland, 1954). Petrov (1975) found that *D. gallinae* engorging on blood of infected birds remained carriers of *P. multocida* for 42 to 64 days, depending on the ambient temperature. Quortrup et al. (1946) recovered *P. multocida* from mites (*Dermanyssus* spp.) collected from ducks dying from avian cholera.

Derylo (1967, 1970) found virulent *P. multocida* in the gut and feces of Mallophaga (*Eomenacanthus stramineus* and *Menopon gallinae*) that fed on hens with avian cholera. Derylo (1969) transmitted *P. multocida* to hens by inoculation of louse feces containing *P. multocida* through impaired skin of the uninfected hens.

Kitt (1888) reported that domestic birds contracted pasteurellosis after ingesting flesh-fly maggots that had fed on avian cholera carcasses. He noted that maggots commonly were ingested by susceptible birds and proposed that fly maggots were an important means of avian cholera transmission (Kitt, 1888). Skidmore (1932) observed that avian cholera was transmitted to turkeys eating house flies (*Musca domestica*) which had fed on the blood of rabbits dying from *P. multocida* infections.

Krinsky (1976) reported transmission of *P. multocida* by tabanid flies. Despite their possible role with domestic birds, there is no direct evidence at this time that arthropods play an important role in the transmission of avian cholera among wildfowl.

Rosen and Morse (1959) proposed that *P. multocida* may be transmitted by inhalation of bacterial aerosols (droplet infection). Donahue and Olson (1971) found turkeys to be very susceptible to *P. multocida* by inoculation into the palatine air spaces. Simensen and Olson (1980) inferred that *P. multocida* could be transmitted readily through the air.

Virulent *P. multocida* can occur in high concentrations in water during avian cholera epizootics (Rosen and Bischoff, 1949). Bacteria often concentrate at the water surface (Potter and Baker, 1961; Potter, 1964). Further, Blanchard and Syzdek (1970) found that air bubbles breaking at an air-water interface remove bacteria concentrated in the surface microlayer and eject them into the atmosphere; the bacterial concentrations in the drops ejected from the bubbles were 10 to 1,000 times higher than that of the water from which the bubbles burst. Thus, waterfowl splashing could create bacteria-rich aerosols.

There is limited information available on the susceptibility of wildfowl to airborne transmission. Titche (1979) observed that 33 (63%) of 52 test waterfowl and coots died from avian cholera when 10^8 to 10^{11} *P. multocida* were inoculated by aerosol over time periods ranging from 15 to 120 min; but the bacterial inocula and exposure times were far greater than one would expect under natural condi-

tions. Yet, Combs (1988) observed that species such as coots, which create aerosols when taking off in large rafts by running across the water, died earlier and in greater numbers during avian cholera epizootics at Centerville, compared to species lifting off singly and vertically (e.g., pintails, shovelers, and mallards).

Ingestion is a route by which *P. multocida* may be transmitted among susceptible wildfowl. *Pasteurella multocida* was readily passed to susceptible birds by the oral route in the laboratory (Rosen and Bischoff, 1949). Quortrup et al. (1946) found that healthy ducks placed near ducks orally infected with *P. multocida* died after 28 hr if they had access to the same drinking water. Pabs-Garnon and Soltys (1971) found that susceptible turkeys were infected after sharing a common water source with experimentally-infected turkeys; *P. multocida* was isolated from the water. Other susceptible turkeys in close proximity to the infected birds, but which did not share the same water source, remained unaffected, suggesting that *P. multocida* was transmitted more readily through ingestion of contaminated water than through droplet aerosol. Zinkl et al. (1977a) reported that each of eight coots exposed to drinking water contaminated with 2.3×10^7 *P. multocida*/ml died from avian cholera within 2 days.

In the natural environment, bacteria were observed to concentrate near the surface of water, rather than deeper within the water column (Potter and Baker, 1961; Potter, 1964). Combs (1988) observed that wildfowl such as American coots and American wigeon, which grazed frequently at the water surface were the first species to die at one avian cholera site, and suffered the greatest losses.

While there is some ambiguity on the role of water as a year-round reservoir of *P. multocida*, it appears likely that water plays a significant role in its transmission. Price and Brand (1984) observed that carcasses were the source of *P. multocida* in water, and noted that the avian cholera epizootic stopped when the bacteria no longer were detectable in water.

Predation or scavenging of infected animals sometimes may play a role in transmitting *P. multocida*. At one avian cholera epizootic, Rosen and Bischoff (1949) reported a dead cat found with the remains of a coot in its stomach; however, it was not clear whether *P. multocida* was isolated from the cat. Later, Rosen and Morse (1959) reported an avian cholera epizootic in coots and ducks that was followed by a *P. multocida* epizootic among meadow mice (*Microtus montanus*). This, in turn, was followed by avian cholera deaths among gulls (*Larus* spp.), short-eared owls (*Asio flammeus*), and northern harriers (*Circus cyaneus*). Stomach contents of the owls and gulls contained mice. A dead weasel (*Mustela* spp.) also had mice in its stomach; *P. multocida* was isolated from the spleen of the weasel (Rosen and Morse, 1959). It was not stated that the same strain of *P. multocida* was isolated from all animals in this interesting study. Zinkl et al. (1977a) observed crows scavenging on infected waterfowl and dying from avian cholera in Nebraska. Taylor and Pence (1981) observed flocks of common crows scavenging on fresh duck carcasses during an avian cholera epizootic in Texas; *P. multocida* was isolated from both moribund and dead crows as well as apparently normal short-eared owls and a cottontail rabbit at this site.

Paullin (1987) observed coots cannibalizing other coots dying from avian cholera, but did not determine whether cannibalizing coots subsequently contracted the disease. Coots also have been observed ingesting gull feces (Rosen and Bischoff, 1950; Gullion, 1952), and infected gulls can shed *P. multocida* (Titche, 1979).

Seven of nine California gulls died from avian cholera after being fed contaminated tissues from wildfowl dying from avian cholera (Titche, 1979). However, Titche (1979) believed the stress of handling and captivity also may have contributed to their susceptibility. During the 1974–1975 epi-

zootic at Nebraska, L. N. Locke (pers. comm.) estimated that eight bald eagles (*Haliaeetus leucocephalus*) died from avian cholera after eating carcasses or sick birds.

Overall, there is little evidence that avian cholera is transmitted among wildfowl by arthropod bite. In contrast, inhalation, ingestion, or both may be important means of transmission. However, the possible role of fly maggots proposed by Kitt (1888) is intriguing, and deserves further consideration.

A PROPOSED MODEL FOR AVIAN CHOLERA EPIZOOTIOLOGY

A model for avian cholera epizootiology in wildfowl should identify the factors increasing the risk of susceptible hosts to infection by *P. multocida* (predisposing factors), the reservoir for the disease, the means by which the disease is first introduced to susceptible populations, the means of transmission between susceptible hosts, the events occuring in affected populations during an epizootic (disease dynamics), and the final impact of each epizootic on the host populations. The last feature, overall mortality, is the best documented aspect among wildfowl. Winter habitat of wildfowl generally is well defined and total wildfowl mortality usually can be determined if the effort is made. Even where vegetation is thick, total mortality can be estimated from recovery rates of marked carcasses. There is less certainty on the other aspects of avian cholera epizootiology, however.

Many potential predisposing factors have been evaluated, including host age, sex, and physical features, as well as environmental factors such as rainfall, ambient temperature, nutritional stress, and chemical contaminants, but with inconclusive results. At Centerville, host density, time spent on land, and feeding strategy were proposed as predisposing factors (Combs, 1988).

The reservoir of *P. multocida* is uncertain. Most studies do not support the hypothesis that soil or water are important reservoirs of *P. multocida*. Although *P. multocida* can survive >1 yr in water (Bredy and Botzler, 1989), conditions in this laboratory study were different from those of natural waterfowl habitats.

Currently, most biologists favor the hypothesis that carrier birds are the most important reservoir for avian cholera among wildfowl. It is clear that many vertebrates, including waterfowl and other birds can carry *P. multocida*, and pasteurellae can be transmitted to susceptible hosts from infected animals. Wobeser (1981) suggested that one fatal case resulting from exacerbation of a carrier state among several hundred normal birds might be sufficient to start an epizootic.

There is evidence suggesting that introduction of avian cholera to susceptible populations may occur regularly. Avian cholera has been noted in every month of the year. Single mortalities or small epizootics have been reported regularly (Bivens, 1953, 1955; Macdonald 1963, 1965; Rosen, 1969, 1972; Blus et al., 1978; Wobeser, 1981; Humburg et al., 1983; R. Botzler, unpubl.) and others probably remain unreported. I believe the introduction of avian cholera into susceptible wildfowl populations may be common, and that most incidents of avian cholera may involve only one or a few birds and remain undetected. However, one of these small events may flare into an extensive epizootic under the proper conditions such as the high wildfowl densities occurring on wintering and staging grounds. Among eiders, Korschgen et al. (1978) noted that avian cholera epizootics occurred on the nesting grounds at a time of both severe stress and high densities of susceptible birds.

Once an epizootic starts, heavy contamination of the environment, especially water, probably allows transmission of *P. multocida* among dense populations by ingestion, inhalation, or both. Mortality among infected waterfowl has varied greatly at different enzootic sites, and even between years at the same site. An analysis

of wildfowl mortality is summarized for one site, Centerville, California, as follows.

Coots suffered greater mortality than any other species, both in absolute numbers and proportion of their live population affected. There was no variation in coot mortality based on sex, age, body weights, or other physical characteristics (Oddo et al., 1978; Mensik and Botzler, 1989). Birds died in a sequence at Centerville: coots died first, followed by tundra swans, American wigeon, northern pintails, northern shovelers, mallards, and teal, respectively (Oddo et al., 1978; Mensik and Botzler, 1989), (Fig. 1). Differences in wildfowl susceptibility to avian cholera were related to differences in their activities; species dying early in the epizootics commonly grazed or fed at the water surface, maintained high density groupings, and used land areas together during the avian cholera season (Combs, 1988). These observations are of limited value without verification (or refutation) of their applicability to other sites.

The most serious short-coming in developing a viable model for avian cholera in wildfowl is not a lack of information, but rather a lack of consistency in the information. This inconsistency is due in part to the variety of species and circumstances among the studies cited, and also because the majority of studies are descriptive, without adequate controls. This inconsistency also may reflect important gaps in our understanding of the disease. Overall, much work still is needed to establish the conditions initiating avian cholera in waterfowl, and magnifying the disease into epizootics.

PREVENTION, CONTROL AND TREATMENT

Despite an increasing interest in migratory bird disease problems of North America (Friend, 1981, 1984), there still is much speculation, and little firm fact on strategies to prevent and control avian cholera epizootics. Most recommendations for prevention are oriented to reducing exposure of susceptible birds and lowering contamination of aquatic ecosystems by *P. multocida* (Friend, 1987).

A regular program of monitoring for wildfowl mortality is important to identify an epizootic at the earliest possible stage. This provides the greatest opportunity to prevent high losses, and the financial costs of control efforts are relatively small compared to those required for handling an extensive epizootic among wildfowl (Friend, 1987). Marsh surveillance and carcass removal is the strategy followed by the California Department of Fish and Game (W. Clark, pers. comm.).

A number of recommendations have been made for controlling an epizootic once it starts. Carcass collection is almost always encouraged. Athough the benefit of carcass collection has never been definitively tested, the procedure is logical. Carcasses may serve as decoys and attract other susceptible birds to a contaminated site. Wobeser et al. (1982) found evidence of seven species of birds and four species of mammals scavenging on avian cholera-killed birds during one epizootic.

Wildfowl carcasses commonly have several milliliters of fluid rich in *P. multocida* that can be discharged from their nares, and further add to the contamination of a site. Carcasses hold a substantial supply of *P. multocida* that is constantly added to the environment as the birds decompose. Survival of *P. multocida* is enhanced by the presence of carcasses, as outlined earlier (Rosen and Bischoff, 1950; Olson and Bond, 1968; Titche, 1979; Price and Brand, 1984).

Further, carcasses attract scavengers which may ingest and transmit *P. multocida* to other sites (Rosen, 1971; Wobeser, 1981). Zinkl et al. (1977a) reported that American crows suffered chronic cases of *P. multocida* infection after scavenging on carcasses of waterfowl dying from avian cholera. Friend (1987) noted that gulls, crows, and other scavengers survived from several days to 2 wk after infection with *P. multocida*. This would greatly facilitate the ability of scavengers to spread the dis-

ease to new sites, some distance from the original source.

Burning collected carcasses is preferred to burying them (Friend, 1987). A high water table occurs during the winter season at many enzootic avian cholera sites and *P. multocida* can survive for several months in water under some conditions, including the presence of animal protein (Bredy and Botzler, 1989). Thus, birds buried in a high water table may provide conditions enhancing survival of *P. multocida* at the site.

It may be beneficial to control movements of bird populations, both to contain infected birds on a site, and to prevent additional susceptible populations from moving onto an infected site. For example, Friend (1987) reported the use of aircraft to move whooping cranes (*Grus americana*) away from an avian cholera epizootic. Use of either aversive devices such as hazing, or attractants such as food have been suggested (Friend, 1987).

One extreme recommendation is depopulation of infected birds to reduce the risk that the disease will spread further among susceptible populations. Gershman et al. (1964) reported the eradication of remaining eiders, gulls, and terns after an epizootic in eiders on some islands off Maine; however, the disease later returned to the site (Korschgen et al., 1978). Pursglove et al. (1976) reported the depopulation of infected coot populations to be effective in halting an epizootic on the Chesapeake Bay. While the data presented show a decline in avian cholera mortality concomitant with the coot eradication, it is not possible to evaluate the impact of depopulation in the absence of an untreated control poulation of coots. Montgomery et al. (1979) also questioned the efficacy of this eradication program.

Friend (1987) believed that population reduction of migratory birds infected with avian cholera is justified only under special conditions: (1) the outbreak must be discrete and localized rather than generalized and widespread, (2) techniques must be available that will allow complete eradication without causing widespread dispersal of potentially infected birds, (3) methods used must be specific for target species and pose no significant risk for nontarget species, (4) eradication must be justified on the basis of risk to other populations if the outbreak is allowed to continue, and (5) the outbreak represents a new geographic extension of avian cholera into an important migratory bird population.

Another suggestion for control is disinfecting small bodies of water. Rosen and Bischoff (1949) treated the water of an infected pond with copper sulfate mixed in hydrochloric acid during one avian cholera epizootic, but could not draw any conclusions on its effectiveness. Following an avian cholera epizootic among common eiders on islands off the coast of Maine, Gershman et al. (1964) reported the disinfection of small puddles and water holes with a cresylic compound in conjunction with wildfowl depopulation and incineration of carcasses, eggs, and nests; no additional mortality was observed 2 wk later, but the contributions of these different control procedures could not be independently assessed. Among domestic poultry, fumigation with methyl bromide can kill *P. multocida* on contaminated litter (Bendheim and Shoshan, 1979).

There is little information available on the treatment of wildfowl suffering from avian cholera. Queen and Quortrup (1946) and Zuydam (1952) successfully treated several species of infected ducks with penicillin. Zinkl et al. (1977b) found that infected Canada geese survived when 50 mg oxytetracycline was given intramuscularly, and also when 500 g/ton tetracycline was given in the feed. However, this procedure is not yet applicable to free-living populations.

Immunization is a potentially important tool of the future. The natural immunity of waterfowl appears to be low. Donahue and Olson (1969) sampled 400 waterfowl in the Mississippi Valley, and found two

birds with detectable antibodies to *P. multocida*. It is not known if these antibodies were protective.

Killed vaccines (bacterins) protect only against particular immunotypes, whereas live-attenuated vaccines provide cross-protection (K. R. Rhoades, pers. comm.). Turkeys can be effectively immunized with live-attenuated vaccines given orally (Collins, 1977). While vaccine overdoses may result in signs and symptoms of avian cholera, there is no evidence that outbreaks result from vaccine overdoses (K. R. Rhoades, pers. comm.).

Queen and Quortrup (1946) showed the potential for immunizing wild ducks with an autogenous bacterin. Price (1985) used a *P. multocida* isolant from a lesser snow goose to develop a beta-propiolactone-inactivated Type 1 killed vaccine effective for giant Canada geese (*Branta canadensis maxima*). This bacterin provided immunity for at least one year among a captive flock of Canada geese in contact with free-flying waterfowl during avian cholera epizootics. The vaccine requires an intramuscular or subcutaneous injection, and an effective delivery system for wild populations is not available.

Habitat manipulation may provide an effective tool in the future. Controlled drainage, water diversion and pumping operations potentially can be used to prevent wildfowl use of problem areas and attract susceptible birds to new, uninfected habitat (Friend, 1987). Based on a small control operation with a flock of Canada geese experiencing avian cholera, Zinkl et al. (1977b) proposed adding water to dilute *P. multocida* present in contaminated drinking water of a site. As the ecological requirements of *P. multocida* become more clearly defined, it may become possible to use habitat management to select against the survival of *P. multocida* in the environment.

I advocate management procedures that avoid major changes in our waterfowl habitats. Despite our shared concern for preventing the serious losses among our already troubled wildfowl populations, wildfowl are only one part of a complex fabric composing natural systems. The whole fabric must be considered in our efforts to manage this one disease. Even if techniques involving major habitat manipulation were successful, I believe that all aspects of that habitat must be considered before acting on the behalf of one favored species or group of species. Techniques whose impacts are limited to *P. multocida* or wildfowl are preferred over those with a broader environmental impact.

A major problem in assessing control strategy, is that virtually none of the recommended procedures have been rigorously tested to determine their value. At the 1988 Wildlife Disease Association Conference, Gary Wobeser proposed that assessment of the benefits of disease control procedures is one of the four major issues that must be addressed in planning appropriate disease control responses. Until the value of these proposed control procedures is established, managers lack the bases for making wise decisions.

FUTURE DIRECTIONS

Despite the extensive work done in recent years, there still is little clarity on the roles of such host factors as sex, age, physical characteristics, genetic factors, densities, and behavior on susceptibility of wildfowl to *P. multocida*. Many past studies are based on the results for one species at one site on one occasion, and are descriptive rather than controlled; this may account for many of the inconsistencies in past results. There is a need for systematic studies, including repetitions of some past work, to verify (or refute) our current perceptions.

A tool with important future implications is the use of rRNA probe and restriction endonuclease analysis to fingerprint and distinguish strains of *P. multocida* (Snipes et al., 1989). Biologists may begin to solve some of our major epizootiological problems once they can trace the origins and routes of separate *P. multocida* strains.

The apparent sudden onset of avian cholera among wildfowl in North America, and indeed in the world, is intriguing. It might be no more than simultaneous chance recognition of a problem already present in these wild populations. But the onset of reports of this problem also coincided to a period when many changes in land use practices were occurring on a broad scale. The large-scale use of pesticides began at this time. The human population has increased drastically, and land is farmed more intensively with a result that less of the historical, natural habitat remains. The birds are more crowded on the remaining habitat. Natural foods have been replaced largely with agricultural crops in the wintering habitat. Thus, avian cholera may be a problem closely linked to human-associated environmental changes. The relationship of avian cholera to water quality, land use practices, environmental contaminants, and other human-associated changes is a fruitful area for increased study. Studies such as these require a networking of skills from several disciplines, and are best conducted by teams of experts from diverse fields.

Another recommended area for study is the development of techniques to vaccinate certain high risk populations, as is being done with rabies in some carnivore populations of North America and Europe. Because of high concentration of birds in wintering areas, and the frequent occurrence of avian cholera on wintering habitat, this direction may hold some promise. An effective experimental vaccine for serotype 1 *P. multocida* already is available (Price, 1985) and should be tested for use in a wide range of wildfowl where serotype 1 strains are a threat. In particular, concern for endangered species at risk to avian cholera justify the additional testing, and eventual vaccination of individual birds.

ACKNOWLEDGMENTS

I greatly appreciate the assistance of W. Clark, L. N. Locke, and K. R. Rhoades for reviewing the manuscript, and S. W. Harris for reviewing avian taxonomy. Translation services provided by R. Becking (Dutch), R. Cornejo (Spanish), and G. Fulton (Serbo-Croatian, Russian) were of great assistance. I also am indebted to S. Gordon, R. Sathrum, and interlibrary loan librarians everywhere.

LITERATURE CITED

ADLAM, C., AND J. M. RUTTER (editors). 1989. *Pasteurella* and pasteurellosis. Academic Press, London, England, 341 pp.

AMERICAN ORNITHOLOGISTS' UNION. 1983. Checklist of North American birds. 6th ed. Allen Press, Inc., Lawrence, Kansas, 877 pp.

ARNBJERG, J. 1978. *Pasteurella multocida* from canine and feline teeth, with a case report of *Glossitis calcinosa* in a dog caused by *P. multocida*. Nordisk Veterinärmedicin 30: 324–332.

AWAD, F. I., A. A. SALEM, AND A. A. FAYED. 1976. Studies on the viability of *Pasteurella multocida* type 1 under simulated environmental conditions in Egypt. Egyptian Journal of Veterinary Sciences 13: 57–69.

BACKSTRAND, J. M., AND R. G. BOTZLER. 1986. Survival of *Pasteurella multocida* in soil and water in an area where avian cholera is enzootic. Journal of Wildlife Diseases 22: 257–259.

BASU, B. C. 1930. On the role of *Argas persicus* Oku in the transmission of *Pasteurella avicida*. The Indian Medical Gazette 65: 326–327.

BENDHEIM, U., AND A. EVEN-SHOSHAN. 1975. Survival of *Pasteurella multocida* and *Pasteurella anatipestifer* in various natural media. Refuah Veterinarith 32: 40–46.

———, AND A. E. SHOSHAN. 1979. Susceptibility to methyl bromide fumigation of *Pasteurella multocida, Pasteurella anatipestifer* and *Escherichia coli* in poultry litter. Refuah Veterinarith 35: 154–158.

BERGERUD, A. T. 1971. The population dynamics of Newfoundland caribou. Wildlife Monograph 25, The Wildlife Society, Washington, D.C., 55 pp.

BEZZEL, E. 1979. Wildenten. BLV Verlagsgesellschaft mbH., München, Federal Republic of Germany, 156 pp.

BIGLAND, C. H. 1954. A rabbit infestation with poultry mites and experimental mite transmission of fowl cholera. Canadian Journal of Comparative Medicine 18: 213–214.

BIVINS, J. A. 1953. Pasteurellosis in a starling. Cornell Veterinarian 43: 241–243.

———. 1955. Pasteurellosis in feral birds. Cornell Veterinarian 45: 180–181.

BLACKBURN, B. O., K. L. HEDDLESTON, AND C. J. PFOW. 1975. *Pasteurella multocida* serotyping results (1971–1973). Avian Diseases 19: 353–356.

BLANCHARD, D. C., AND L. SYZDEK. 1970. Mech-

anism for water-to-air transfer and concentration of bacteria. Science 170: 626–628.

BLUS, L. J., L. N. LOCKE, AND E. CROMARTIE. 1978. Avian cholera and organochlorine residues in an American oystercatcher. Estuaries 1: 128–129.

BOND, R. E., E. L. MCCUNE, AND L. D. OLSON. 1972. Isolation of *Pasteurella multocida* from wild raccoons and foxes: Preliminary report. Journal of Wildlife Diseases 8: 296–299.

BRAND, C. J. 1984. Avian cholera in the Central and Mississippi flyways during 1979–1980. The Journal of Wildlife Management 48: 399–406.

———, AND R. M. DUNCAN. 1983. Avian cholera in an American flamingo, *Phoenicopterus ruber*: A new host record. California Fish and Game 69: 190–191.

BREDY, J., AND R. G. BOTZLER. 1989. The effects of six environmental variables on the survival of *Pasteurella multocida* in water. Journal of Wildlife Diseases 25: 232–239.

BROGDEN, K. A., AND K. R. RHOADES. 1983. Prevalence of serologic types of *Pasteurella multocida* from 57 species of birds and mammals in the United States. Journal of Wildlife Diseases 19: 315–320.

BROWN, P. C., J. P. SCHERZ, AND R. M. WINDINGSTAD. 1983. Remote sensing and avian cholera in Nebraska wetlands. *In* Technical Papers of the American Congress on Surveying and Mapping. American Society of Photogrammetry, Salt Lake City, Utah, pp. 483–492.

CLEMENTS, J. 1981. Birds of the world: A checklist. Facts on File, Inc., New York, New York, 562 pp.

COLLINS, F. M. 1977. Mechanisms of acquired resistance to *Pasteurella multocida* infections: A review. Cornell Veterinarian 67: 103–138.

———. 1984. Effect of aztreonam on the growth of *Pasteurella multocida* in the lung. American Journal of Veterinary Research 45: 972–975.

COMBS, S. 1988. Behavioral ecology of coots and waterfowl at an avian cholera epornitic site, Humboldt County, California. M. S. Thesis. Humboldt State University, Arcata, California, 109 pp.

COON, N. C., L. N. LOCKE, E. CROMARTIE, AND W. L. REICHEL. 1970. Causes of bald eagle mortality, 1960–65. Journal of Wildlife Diseases 6: 72–76.

CURTIS, P. E. 1979. Observations on avian pasteurellosis in Britain. The Veterinary Record 104: 471–474.

———, AND G. E. OLLERHEAD. 1981. Investigation to determine whether healthy chickens and turkeys are oral carriers of *Pasteurella multocida*. The Veterinary Record 108: 206–207.

DE LISLE, G. W., W. L. STANISLAWEK, AND P. J. MOORS. 1990. *Pasteurella multocida* infections in rockhopper penguins (*Eudyptes chrysocome*) from Campbell Island, New Zealand. Journal of Wildlife Diseases 26: 283–285.

DERYLO, A. 1967. Rola wszolow w przenoszeniu pasterelozy u kur. Wiadomosci Parazytologiczne 13: 619–623.

———. 1969. Wszoly (*Mallophaga*) jako wektory *Pasteurella multocida*. Annales Universitatis Mariae Curie-Sklodowska, Sectio C: Biologia 24: 355–366.

———. 1970. Mallophaga as a reservoir of *Pasteurella multocida*. Acta Parasitologica Polonica 17: 301–313.

DIMOV, I. 1964. Survival of avian *Pasteurella multocida* in soils at different acidity, humidity and temperature. [In Russian with German summary.] Nauchni Trudove Vissh Veterinarnomeditsinski Institute "Prof. D-r-G Pavlov" 12: 339–345.

DONAHUE, J. M., AND L. D. OLSON. 1969. Survey of wild ducks and geese for *Pasteurella* spp. Bulletin of the Wildlife Disease Association 5: 201–205.

———, AND L. D. OLSON. 1971. Research technique. Inoculation of *Pasteurella multocida* into the palatine air spaces as an exposure method for fowl cholera in turkeys. Avian Diseases 15: 158–162.

EVELETH, D. F., A. E. GOLDSBY, AND C. I.NELSON. 1949. Fowl cholera (*Pasteurella multocida*). Veterinary Medicine 64: 73–78.

FADDOUL, G. P., G. W. FELLOWS, AND J. BAIRD. 1967. Pasteurellosis in wild birds in Massachusetts. Avian Diseases 11: 413–418.

FOREYT, W. J., AND D. A. JESSUP. 1982. Fatal pneumonia of bighorn sheep following association with domestic sheep. Journal of Wildlife Diseases 18: 163–168.

FRANKOWSKI, M. 1967. Problemy epizootiologiczne pasterelozy w województwie Warszawskim w latach 1957–1961. Polskie Archiwum Weterynaryjne 10: 493–506.

FRANSON, J. C. 1986. Immunosuppressive effects of lead. *In* Lead poisoning in wild waterfowl—A workshop, J. S. Feierabend and A. B. Russell (eds.). Cooperative Lead Poisoning Control Information Program, National Wildlife Federation, Washington, D.C., pp. 106–109.

FRIEND, M. 1981. Waterfowl management and waterfowl disease: Independent or cause and effect relationships? Transactions of the North American Wildlife and Natural Resources Conference 46: 94–103.

———. 1984. Meeting migratory bird management needs by integrated disease control. Transactions of the North American and Natural Resources Conference 49: 480–488.

———. 1987. Avian cholera. *In* Field guide to wildlife diseases, Vol. 1: General field procedures and diseases of migratory birds, M. Friend (ed.). U.S.

Fish and Wildlife Service Resource Publication No. 167, Washington, D.C., pp. 69–82.

———, AND D. O. TRAINER. 1970. Some effects of sublethal levels of insecticides on vertebrates. Journal of Wildlife Diseases 6: 335–342.

FUJIHARA, Y., M. ONAI, S. KOIZUMI, N. SATOH, AND T. SOWADA. 1986. An outbreak of fowl cholera in wild ducks (*Rosybilled pochard*) in Japan. Japanese Journal of Veterinary Science 48: 35–43.

GERSHMAN, M., J. F. WITTER, H. E. SPENCER, JR., AND A. KALVAITIS. 1964. Case report: Epizootic of fowl cholera in the common eider duck. The Journal of Wildlife Management 28: 587–589.

GLUKHOV, V. F., AND V. G. NOVIKOV. 1975. Reproduction of the pathogen of poultry pasteurellosis in the body of *Argas persicus* [In Russian.] Nauchnye Trudy Stavropol'skii Sel'skokhozyaistvennyi Institut 5: 47–50.

GORDON, C. C. 1989. The relationship of wetland characteristics to avian cholera (*Pasteurella multocida*) outbreaks in the Rainwater Basin area of Nebraska. M. S. Thesis. South Dakota State University, Brookings, South Dakota, 126 pp.

GORDUS, A. G. 1985. Lead concentrations in liver and kidneys of snow geese during an avian cholera epizootic at Delevan National Wildlife Refuge, California. M. S. Thesis. Humboldt State University, Arcata, California, 58 pp.

GRATZL, E., AND H. KÖHLER. 1968. Pasteurellose (Geflügelcholera). *In* Spezielle Pathologie und Therapie der Geflügelkrankheiten. Ferdinand Enke Verlag, Stuttgart, Federal Republic of Germany, pp. 493–520.

GRAY, H. 1913. Avian cholera. *In* A system of veterinary medicine, Vol. I, E. W. Hoare (ed.). Alexander Eger, Chicago, Illinois, pp. 420–432.

GREEN, R. G., AND J. E. SHILLINGER. 1936. Progress report of wildlife disease studies for 1935. Transactions of the North American Wildlife Conference 1: 469–471.

GREGG, D. A., L. D. OLSON, AND E. L. MCCUNE. 1974. Experimental transmission of *Pasteurella multocida* from raccoons to turkeys via bite wounds. Avian Diseases 18: 559–564.

GRITMAN, R. B., AND W. I. JENSEN. 1965. Avian cholera in a trumpeter swan (*Olor buccinator*). Bulletin of the Wildlife Disease Association 1: 54–55.

GULLION, G. W. 1952. Some diseases and parasites of American coots. California Fish and Game 38: 421–423.

HAZLEWOOD, R. M., A. F. ODDO, R. D. PAGAN, AND R. G. BOTZLER. 1978. The 1975–76 avian cholera outbreaks in Humboldt County, California. Journal of Wildlife Diseases 14: 229–232.

HEDDLESTON, K. L. 1972. Avian pasteurellosis. *In* Diseases of poultry, M. S. Hofstad, B. W. Calnek, C. F. Helmboldt, W. M. Reid, and H. W. Yoder, Jr. (eds.). Iowa State University Press, Ames, Iowa, pp. 219–251.

———. 1976. Physiologic characteristics of 1,268 cultures of *Pasteurella multocida*. American Journal of Veterinary Research 37: 745–747.

———, AND L. P. WATKO. 1963. Fowl cholera: Susceptibility of various animals and their potential as disseminators of the disease. Proceedings of the U.S. Livestock Sanitation Association 67: 247–250.

———, AND ———. 1965. Fowl cholera: Comparison of serologic and immunogenic responses of chickens and turkeys. Avian Diseases 9: 367–376.

———, T. GOODSON, L. LEIBOVITZ, AND C. I. ANGSTROM. 1972. Serological and biochemical characteristics of *Pasteurella multocida* from free-flying birds and poultry. Avian Diseases 16: 729–734.

HENRICKSON, J. M., AND K. F. HILBERT. 1932. The persistance of *Pasteurella avicida* in the blood and organs of fowls with spontaneous fowl cholera. Journal of Infectious Diseases 50: 89–97.

HINSHAW, W. R., AND J. E. EMLEN. 1943. Pasteurellosis in California valley quail. Cornell Veterinarian 33: 351–354.

HIRSH, D. C., D. A. JESSUP, K. P. SNIPES, T. E. CARPENTER, D. W. HIRD, AND R. H. MCCAPES. 1990. Characteristics of *Pasteurella multocida* isolated from waterfowl and associated avian species in California. Journal of Wildlife Diseases 26: 204–209.

HOFFMAN, H. A., AND D. E. STOVER. 1942. An analysis of thirty thousand autopsies on chickens. California Department of Agriculture Bulletin 31: 7–30.

HUBBERT, W. T., AND M. N. ROSEN. 1970. I. *Pasteurella multocida* infection due to animal bite. American Journal of Public Health 60: 1103–1108.

HUDSON, C. B. 1944. Fowl cholera in ring-necked pheasants. Journal of the American Veterinary Medical Association 109: 211–212.

HUDSON, J. R. 1959. Pasteurellosis. *In* Infectious diseases of animals, Vol. 2, A. Stableforth and I. Galloway (eds.). Butterworths Scientific Publ., London, England, pp. 413–436.

HUMBURG, D. D., D. GRABER, S. SHERIFF, AND T. MILLER. 1983. Estimating autumn-spring waterfowl nonhunting mortality in north Missouri. Transactions of the North American Wildlife and Natural Resources Conference 48: 241–256.

HUNGERFORD, T. G. 1968. A clinical note on avian cholera. The effect of age on susceptibility of fowls. Australian Veterinary Journal 44: 31–32.

HUNTER, B., AND G. WOBESER. 1980. Pathology of experimental avian cholera in mallard ducks. Avian Diseases 24: 403–414.

HUNTER, B. F. 1967. Isolation of *Pasteurella multocida* from a snowy owl (*Nyctea scandiaca*), a

new host record. California Fish and Game 53: 213–214.

HUTYRA, F., J. MAREK, AND R. MANNINGER. 1949. Special pathology and therapeutics of the diseases of domestic animals, Vol. I. Fifth English edition, J. R. Greig (ed.). Alexander Eger Inc., Chicago, Illinois, 962 pp.

ILIEV, T., R. ARSOV, AND V. LAZAROV. 1965a. Can fowl, carriers of *Pasteurella*, excrete the organism in feces? [In Bulgarian with German summary.] Nauchni Trudove Vissh Veterinarnomeditsinski Institute "Prof. D-r-G Pavlov" 14: 7–12.

———, ———, E. IOVCHEV, G. GIRGINOV, AND I. DIMOV. 1965b. The carrier state in fowl infected with nonpathogenic *Pasteurella* strains. [In Bulgarian with German summary.] Nauchni Trudove Vissh Veterinarnomeditsinski Institute "Prof. D-r-G Pavlov" 14: 19–22.

IOVCHEV, E. 1967. Die Rolle von *Argas persicus* in der Epizootiologie der Geflügelcholera. Angewandte Parasitologie 8: 114–117.

JAKSIC, B. L., M. DORDEVIC, AND B. MARKOVIC. 1964. Fowl cholera in wild birds. [In Serbo-Croatian with German summary.] Veterinarski Glasnik 18: 725–730.

JANUSCHKE, E. 1915. Geflügelcholera beim Sperber (*Accipiter nisus* sc. Nisus com.). Wiener Tierärztliche Monatsschrift 1915: 272–273.

JENNINGS, A. R. 1954. Diseases in wild birds. Journal of Comparative Pathology 64: 356–359.

———. 1955. Diseases in wild birds. Bird Study 2: 69–72.

———, AND E. J. L. SOULSBY. 1956. Diseases in wild birds, third report. Bird Study 3: 270–272.

JOHNSGARD, P. A. 1978. Ducks, geese, and swans of the world. University of Nebraska Press, Lincoln, Nebraska, 404 pp.

JOHNSON, A. W. 1965. The birds of Chile and adjacent regions of Argentina, Bolivia and Peru, Vol. I. Platt Establecimientos Gráficos S. A., Buenos Aires, Argentina, 398 pp.

———. 1967. The birds of Chile and adjacent regions of Argentina, Bolivia and Peru, Vol. II. Platt Establecimientos Gráficos S. A., Buenos Aires, Argentina, 447 pp.

JÖST, E. 1915. Avian cholera in the uhu (*Bubo maximum*). [In German.] Beitrage Tierheilkunde 1915: 11 (Original not seen).

KASCHULA, V. R., AND D. E. TRUTER. 1951. Fowl cholera in sea gulls on Dassen Island. Journal of the South African Veterinary Medical Association 22: 191–192.

KEYMER, I. F. 1958. A survey and review of the causes of mortality in British birds and the significance of wild birds as disseminators of disease. The Veterinary Record 70: 713–720.

KITT, T. 1887. Die Geflügelcholera. Centralblatt für Bacteriologie und Parasitenkunde 1: 305–314.

———. 1888. Beiträge zur Kenntniss der Geflügelcholera und deren Schutzimpfung. Deutsche Zeitschrift für Tiermedicin und Vergleichende Pathologie 13: 1–30.

KLEIN, E. 1889. Ueber ein akute infektiöse Krankheit des schottischen Moorhuhnes (*Lagopus scoticus*). Centralblatt für Bakteriologie und Parasitenkunde 6: 36–41.

KLUKAS, R. W., AND L. N. LOCKE. 1970. An outbreak of fowl cholera in Everglades National Park. Journal of Wildlife Diseases 6: 77–79.

KORBEL, R. 1990. Epizootiologie, Klinik und Therapie der *Pasteurella-multocida*-Infektion beim Vogelpatienten nach Katzenbiss. Tierärtzliches Praxis 18: 365–376.

KORSCHGEN, C. E., H. C. GIBBS, AND H. L. MENDALL. 1978. Avian cholera in eider ducks in Maine. Journal of Wildlife Diseases 14: 254–258.

KRINSKY, W. L. 1976. Animal disease agents transmitted by horse flies and deer flies (Diptera: Tabanidae). Journal of Medical Entomology 13: 225–275.

LOCKE, L. N., AND R. C. BANKS. 1972. Avian cholera in cedar waxwings in Ohio. Journal of Wildlife Diseases 8: 106.

———, V. STOTTS, AND G. WOLFHARD. 1970. An outbreak of fowl cholera in waterfowl on the Chesapeake Bay. Journal of Wildlife Diseases 6: 404–407.

———, J.A. NEWMAN, AND B. M. MULHERN. 1972. Avian cholera in a bald eagle from Ohio. Ohio Journal of Science 72: 294–296.

MACDONALD, J. W. 1963. Mortality in wild birds. Bird Study 10: 91–108.

———. 1965. Mortality in wild birds. Bird Study 12: 181–195.

———, D. OWEN, K. G. SPENCER, AND P. E. CURTIS. 1981. Pasteurellosis in wild birds. The Veterinary Record 109: 58.

MCDIARMID, A. 1962. Diseases of free-living wild animals. Food and Agricultural Organization Studies No. 57. United Nations, Rome, Italy, 119 pp.

MCLANDRESS, M. R. 1983. Sex, age, and species differences in disease mortality of Ross' and lesser snow geese in California: Implications for avian cholera research. California Department of Fish and Game 69: 196–206.

MENSIK, J. G. 1986. Epizootiology of avian cholera on the Centerville Gun Club, Humboldt County, California. M.S. Thesis. Humboldt State University, Arcata, California, 46 pp.

———, AND R. G. BOTZLER. 1989. Epizootiological features of avian cholera at Centerville, Humboldt County, California. Journal of Wildlife Diseases 25: 240–245.

METWALLY, M. A. E. R., L. ELGHANAM, AND M. MILIGY. 1978. *Argas persicus* as a potential vector of fowl cholera and gallinarum-pullorum diseases in birds. Veterinary Medical Journal (Cairo) 26: 223–230.

MONTGOMERY, R. D., G. STEIN, JR., V. D. STOTTS,

AND F. H. SETTLE. 1979. The 1978 epornitic of avian cholera on the Chesapeake Bay. Avian Diseases 23: 966–978.

MOORE, R. L., AND C. D. SIMPSON. 1981. Disease mortality of waterfowl on Texas Playa lakes. Southwestern Naturalist 25: 566–568.

MULCAHY, D., P. WARPINSKI, L. BENJAMIN, AND D. HAMILTON. 1988. Avian cholera and related topics: An annotated bibliography. U.S. Fish and Wildlife Service Biological Report 88(40), Washington, D.C., 190 pp.

MUTTERS, R., P. IHM, S. POHL, W. FREDERICKSEN, AND W. MANNHEIM. 1985. Reclassification of the genus *Pasteurella* Trevisan 1887 on the basis of deoxyribonucleic acid homology, with proposals for the new species *Pasteurella dagmatis, Pasteurella canis, Pasteurella stomatis, Pasteurella anatis,* and *Pasteurella langaa*. International Journal of Systematic Bacteriology 35: 309–322.

NOVIKOV, A. 1954. Migrating rooks as carriers of fowl cholera. [In Russian.] Veterinariya (Moscow) 31: 27.

ODDO, A. F., R. D. PAGAN, L. WORDEN, AND R. G. BOTZLER. 1978. The January 1977 avian cholera epornitic in northwest California. Journal of Wildlife Diseases 14: 317–321.

OLSON, L. D., AND R. E. BOND. 1968. Survival of *Pasteurella multocida* in soil, water, carcasses and in the mouths of various birds and mammals. Proceedings of the Annual Meeting of the Livestock Sanitation Association 72: 244–246.

OWEN, C. R., E. O. BUKER, J. F. BELL, AND W. L. JELLISON. 1968. *Pasteurella multocida* in animals' mouths. Rocky Mountain Medical Journal 65: 45–46 (November 1968).

PABS-GARNON, L. F., AND M. A. SOLTYS. 1971. Methods of transmission of fowl cholera in turkeys. American Journal of Veterinary Research 32: 1119–1120.

PARMELEE, D. F., S. J. MAXSON, AND N. P. BERNSTEIN. 1979. Fowl cholera outbreak among brown skuas at Palmer Station. Antarctic Journal of the United States 14: 168–169.

PAULLIN, D. G. 1987. Cannibalism in American coots induced by severe spring weather and avian cholera. The Condor 89: 442–443.

PETRIDES, G. A., AND C. R. BRYANT. 1951. An analysis of the 1949-50 fowl cholera epizootic in Texas Panhandle waterfowl. Transactions of the North American Wildlife Conference 16: 193–216.

PETROV, D. 1970. On the carriage of *Pasteurella multocida* by *Argas persicus*. Veterinarno-Meditsinski Nauki 7: 11–16.

———. 1975. Studies on the gamasid red mite of poultry, *Dermanyssus gallinae,* as a carrier of *Pasteurella multocida* [In Russian with English summary.] Veterinarno-Meditsinski Nauki 12: 32–36.

PIER, A. C. 1981. Mycotoxins and animal health. Advances in Veterinary Science and Comparative Medicine 25: 185–243.

POTTER, L. F. 1964. Planktonic and benthic bacteria of lakes and ponds. *In* Principles and applications in aquatic microbiology, H. Heukelekian and N. C. Dondero (eds.). John Wiley and Sons, Incorporated, New York, New York, pp. 148–161.

———, AND G. E. BAKER. 1961. The microbiology of Flathead and Rogers Lakes, Montana. II. Vertical distribution of the microbial populations and chemical analyses of their environments. Ecology 42: 338–348.

PRICE, J. I. 1985. Immunizing Canada geese against avian cholera. Wildlife Society Bulletin 13: 508–515.

———, AND C. J. BRAND. 1984. Persistence of *Pasteurella multocida* in Nebraska wetlands under epizootic conditions. Journal of Wildlife Diseases 20: 90–94.

PRITCHETT, I. W., F. R. BEAUDETTE, AND T. P. HUGHES. 1930a. The epidemiology of fowl cholera. IV. Field observations of the "spontantous" disease. Journal of Experimental Medicine 51: 249–258.

———, ———, AND ——— 1930b. The epidemiology of fowl cholera. V. Further field observations of the "spontaneous" disease. Journal of Experimental Medicine 51: 259–274.

———, AND T. P. HUGHES. 1932. The epidemiology of fowl cholera. VI. The spread of epidemic and endemic strains of *Pasteurella avicida* in laboratory populations of normal fowl. Journal of Experimental Medicine 55: 71–78.

PURSGLOVE, S. R., JR., D. F. HOLLAND, F. H. SETTLE, AND D. C. GNEGY. 1976. Control of a fowl cholera outbreak among coots in Virginia. Southeastern Association of Game and Fish Commissioners 30: 602–609.

QUAN, T. J., K. R. TSUCHIYA, AND L. G. CARTER. 1986. Recovery and identification of *Pasteurella multocida* from mammals and fleas collected during plague investigations. Journal of Wildlife Diseases 22: 7–12.

QUEEN, F. B., AND E. R. QUORTRUP. 1946. Treatment of *Pasteurella multocida* (fowl cholera) infection in wild ducks with autogenous bacterin and penicillin. Journal of the American Veterinary Medical Association 108: 101–103.

QUORTRUP, E. R., F. B. QUEEN, AND L. J. MEROVKA. 1946. An outbreak of pasteurellosis in wild ducks. Journal of the American Veterinary Medical Association 108: 94–100.

RAGGI, L. G., AND G. S. STRATTON. 1954. Pasteurellosis in coots. Cornell Veterinarian 42: 229–230.

REED, A., AND J. G. COUSINEAU. 1967. Epidemics involving the common eider (*Somateria mollis-*

sima) at Ile Blanche, Quebec. Naturaliste Canadien 94: 327–334.

RHOADES, K. R., AND R. B. RIMLER. 1984. Avian pasteurellosis. *In* Diseases of poultry. 8th ed. M. S. Hofstad, H. J. Barnes, B. W. Calnek, W. M. Reid, and H. W. Yoder, Jr. (eds.). Iowa State University Press, Ames, Iowa, pp. 141–156.

———, AND R. B. RIMLER. 1989. Fowl cholera. *In* *Pasteurella* and pasteurellosis. C. Adlam and J. M. Rutter (eds.). Academic Press, London, England, pp. 95–113.

RIMLER, R. B., AND K. R. RHOADES. 1986. Fowl cholera: Influence of *Bordetella avium* on vaccinal immunity of turkeys to *Pasteurella multocida*. Avian Diseases 30: 838–839.

———, P. A. REBERS, AND M. PHILLIPS. 1984. Lipopolysaccharides of the Heddleston serotypes of *Pasteurella multocida*. American Journal of Veterinary Research 45: 759–763.

ROCKE, T. E., T. M. YUILL, AND R. D. HINSDILL. 1984. Oil and related toxicant effects on mallard immune defenses. Environmental Research 33: 343–352.

ROSEN, M. N. 1969. Species susceptibility to avian cholera. Bulletin of the Wildlife Disease Association 5: 195–200.

———. 1970. Pasteurellosis. *In* Infectious diseases of wild mammals, J. W. Davis, L. H. Karstad, and D. O. Trainer (eds.). The Iowa State University Press, Ames, Iowa, pp. 244–252.

———. 1971. Avian cholera. *In* Infectious and parasitic diseases of wild birds. J. W. Davis, R. C. Anderson, L. Karstad, and D. O. Trainer (eds.). The Iowa State University Press, Ames Iowa, pp. 59–74.

———. 1972. The 1970–71 avian cholera epornitic's impact on certain species. Journal of Wildlife Diseases 8: 75–78.

———, AND A. I. BISCHOFF. 1949. The 1948–49 outbreak of fowl cholera in birds in the San Francisco Bay area and surrounding counties. California Fish and Game 35: 185–192.

———, AND A. I. BISCHOFF. 1950. The epidemiology of fowl cholera as it occurs in the wild. Transactions of the North American Wildlife Conference 15: 147–154.

———, AND E. E. MORSE. 1959. An interspecies chain in a fowl cholera epizootic. California Fish and Game 45: 51–56.

———, K. D'AMICO, AND E. J. O'NEILL. 1973. First record of a golden eagle death due to avian cholera. California Fish and Game 59: 209–210.

SANDERS, D. A. 1938. Hemorrhagic septicemia: The significance of *Pasteurella boviseptica* encountered in the blood of some Florida cattle. Florida Agricultural Experiment Station Bulletin 322: 5–24.

SCHIPPER, G. J. 1947. Unusual pathogenicity of *Pasteurella multocida* isolated from the throats of common wild rats. Bulletin of the Johns Hopkins Hospital 81: 333–356.

SERDYUK, N. G., AND P. F. TSIMOKH. 1970. Role of free-living birds and rodents in the distribution of pasteurellosis (In Russian). Veterinariya (Moscow) 6: 53–54.

SCHILLINGER, J. E., AND L. C. MORLEY. 1942. Diseases of upland game birds. U.S. Fish and Wildlife Service Conservation Bulletin 21: 12.

SIMENSEN, E., AND L. D. OLSON. 1980. Aerosol transmission of *Pasteurella multocida* in turkeys. Avian Diseases 24: 1007–1010.

———, L. D. OLSEN, AND G. L. HAHN. 1980. Effects of high and low environmental temperatures on clinical course of fowl cholera in turkeys. Avian Diseases 24: 816–832.

SKIDMORE, L. V. 1932. The transmission of fowl cholera to turkeys by the common house fly (*Musca domestica* Linn.) with brief notes on the viability of fowl cholera microorganisms. Cornell Veterinarian 22: 281–285.

SMIT, T., L. G. MORAAL, AND T. BAKHUIZEN. 1980. *Pasteurella multocida* infecties bij vogels na een kattebeet. Tijdschrift voor Diergeneeskunde 105: 327–329.

SMITH, B. J., K. F. HIGGINS, AND C. F. GRITZNER. 1989. Land use relationships to avian cholera outbreaks in the Nebraska Rainwater Basin area. Prairie Naturalist 21: 125–136.

SNIPES, K. P., T. E. CARPENTER, J. L. CORN, R. W. KASTEN, D. C. HIRSH, D. W. HIRD, AND R. H. MCCAPES. 1988. *Pasteurella multocida* in wild mammals and birds in California: Prevalence and virulence for turkeys. Avian Diseases 32: 9–15.

———, D. C. HIRSH, R. W. KASTEN, L. M. HANSEN, D. W. HIRD, T. W. CARPENTER, AND R. H. MCCAPES. 1989. Use of rRNA probe and restriction endonuclease analysis to fingerprint *Pasteurella multocida* isolated from turkeys and wildlife. Journal of Clinical Microbiology 27: 1847–1853.

STICKER, A. 1888. Käsige Processe bei der Geflügelcholera. Archiv für Wissenschaftliche und Praktische Thierheilkunde 14: 333–347.

STOUT, I. J., AND G. W. CORNWELL. 1976. Nonhunting mortality of fledged North American waterfowl. The Journal of Wildlife Management 40: 681–693.

SUAREZ, J. G., AND L. L. ILAZABAL. 1941. Epidemia de colera en los patos marinos. Revista de Medicina Veterinaria (Buenos Aires) 23: 145–149.

SZÉCSÉNYI, I. 1965. Ujabb adatok a baromfikolera oktanához, járványtanához és a bacillusgazdakérdéshez. Magyar Allatorvosok Lapja 20: 53–56.

TAYLOR, T. T., AND D. B. PENCE. 1981. Avian cholera in common crows (*Corvus brachyrhynchos*) from the central Texas Panhandle. Journal of Wildlife Diseases 17: 511–514.

TITCHE, A. 1979. Avian cholera in California. Wild-

life Management Branch Administrative Report 79-2. California Department of Fish and Game, Sacramento, California, 49 pp.

VAN DEN HURK, C. F. G. W. 1946. Aanteekeningen bij de epizoötie van vogelcholera over Nederland in het najaar van 1945. Tijdschrift voor Diergeneeskund 71: 361–365.

VAN ES, L., AND J. F. OLNEY. 1940. An inquiry into the influence of environment on the incidence of poultry diseases. University of Nebraska Agricultural Experiment Station Research Bulletin 118: 17–21.

VAUGHT, R. W., H. C. MCDOUGLE, AND H. H. BURGESS. 1967. Fowl cholera in waterfowl at Squaw Creek National Wildlife Refuge, Missouri. The Journal of Wildlife Management 31: 248–253.

WADDINGTON, F. G. 1944. Pasteurellosis in poultry and wild birds in Tanganyika territory. Veterinary Journal 100: 187–191.

WETZEL, R., AND W. RIECK. 1972. Krankheiten des Wildes. Verlag Paul Parey, Hamburg, Federal Republic of Germany. 256 pp.

WICKWARE, A. B. 1945. Case reports of relatively infrequent diseases observed at the Poultry Pathology Laboratory. Canadian Journal of Comparative Medicine 9: 151–154.

WILLIAMS, E. S., D. E. RUNDE, K. MILLS, AND L. D. HOLLER. 1987. Avian cholera in a gyrfalcon (*Falco rusticolus*). Avian Diseases 31: 380–382.

WILSON, S. S. 1979. A bibliography of references to avian cholera. U.S. Fish and Wildlife Service Special Scientific Report—Wildlife No. 217, Washington, D.C., 18 pp.

WINDINGSTAD, R. M., R. M. DUNCAN, AND D. THORNBURG. 1983. Outbreak of avian cholera on the wintering grounds of the Mississippi Valley Canada goose flock. Journal of Wildlife Diseases 19: 95–97.

———, J. J. HURT, A. K. TROUT, AND J. CARY. 1984. Avian cholera in Nebraska's rainwater basin. Transactions of the North American Wildlife and Natural Resources Conference 49: 576–583.

———, S. M. KERR, R. M. DUNCAN, AND C. J. BRAND. 1988. Characterization of an avian cholera epizootic in wild birds in western Nebraska. Avian Diseases 32: 124–131.

WOBESER, G. A. 1981. Avian cholera. *In* Diseases of Wild Waterfowl. Plenum Press, New York, New York, pp. 47–60.

———. 1986. Interaction between lead and other disease agents. *In* Lead poisoning in wild waterfowl—A workshop, J. S. Feierabend and A. B. Russell (eds.). Cooperative Lead Poisoning Control Information Program, National Wildlife Federation, Washington, D.C., pp. 109–112.

———, D. B. HUNTER, B. WRIGHT, D. J. NIEMAN, AND R. ISBISTER. 1979. Avian cholera in waterfowl in Saskatchewan, Spring 1977. Journal of Wildlife Diseases 15: 19–24.

———, F. A. LEIGHTON, A. D. OSBORNE, D. J. NIEMAN, AND J. R. SMITH. 1982. Avian cholera in waterfowl in western Canada, 1978–1981. The Canadian Field-Naturalist 96: 317–322.

ZINKL, J. G., N. DEY, J. M. HYLAND, J. J. HURT, AND K. L. HEDDLESTON. 1977a. An epornitic of avian cholera in waterfowl and common crows in Phelps County, Nebraska, in the spring, 1975. Journal of Wildlife Diseases 13: 194–198.

———, J. J. HURT, J. M. HYLAND, N. DEY, D. STUDNECKA, AND D. D. KING. 1977b. Treatment of captive giant Canada geese affected by avian cholera. Journal of Wildlife Diseases 13: 294–296.

ZUYDAM, D. M. 1952. Penicilline als therapeuticum bij vogelcholera. Tijdschrift voor Diergeneeskunde 77: 256–257.

Received for publication 9 July 1990.