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LETTER TO THE EDITOR . . .

Avian Cholera and Waterfowl Biology

In a review of the ecology of *Pasteurella multocida* infection in waterfowl, Botzler (1991) indicated that many fundamental questions remain unanswered; yet "the extreme virulence of avian cholera makes its control one of the highest priorities for waterfowl management in North America" (Bolen et al., 1989). The review by Botzler (1991) was particularly valuable because it proposed a model for the disease, lack of which has hampered research and development of management strategies. Research has centered on local phenomena, such as defining characteristics of wetlands where birds were found dead, while the population ecology of the disease has received little attention. Waterfowl management has, in general, focused on production of birds, with comparatively little attention to reduction of non-hunting mortality. There is increasing interest in managing habitat for migrating and wintering waterfowl (Weller, 1987; Smith et al., 1989), but management to control avian cholera is reactive, consisting primarily of collecting and disposing of carcasses when outbreaks occur. In this letter I hope to relate habitat and management changes to avian cholera, with the intent of further stimulating interest in this disease.

It is useful to consider the interaction between *P. multocida* and a susceptible bird (Fig. 1) as a starting point. Even in the most devastating outbreak not all birds die, so the interaction is not all-or-nothing. Exposure is the coming together of bird and bacterium. If a bird is not exposed, disease will not occur. The probability of exposure is related to the density and distribution of bacteria and birds, the amount of time they spend together, and viability of the bacterium in the environment. The bird's resistance has no effect on the rate of exposure. Infection involves invasion and growth of bacteria in the bird. Not all exposed individuals become infected. In the laboratory, birds can be infected by injection of a few *P. multocida* organisms (Hunter and Wobeser, 1980); however, under natural conditions a larger number of organisms is likely required to result in infection. The probability of infection is related to the route, duration and degree of exposure, virulence of the organism, and the bird's resistance. Disease consists of physical changes and signs of illness produced by infection. Avian cholera is considered an acute, highly fatal infection but the actual proportion of waterfowl that develop disease following infection is unknown, as is the proportion of diseased birds that recover. The probability that an infected bird will become diseased and subsequently die or recover is influenced by its resistance to the agent, as well as by the presence of factors that influence the ability of a sick bird to survive long enough to recover. Thus, the likelihood of an individual bird being exposed, infected, diseased, and dying of avian cholera is the product of many separate probabilities related to exposure and resistance. The likelihood of an outbreak occurring introduces another range of probabilities.

A major unknown in the ecology of avian cholera is the source of *P. multocida*. I believe that avian cholera is perpetuated by carriers of *P. multocida* among waterfowl although evidence for existence of carriers in wild waterfowl is inconclusive. *Pasteurella multocida* has been recovered from clinically healthy waterfowl (Vaught et al., 1967; Korschgen et al., 1978; Titche, 1979) but the virulence of the bacteria isolated from these birds was not reported. The importance of carrier birds in chickens has been known for many years (Hughes and Pritchett, 1930; Iliev et al., 1964; Curtis and Ollerhead, 1981). Carriers have been reported in domestic geese

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and these apparently served as a source of infection for chickens (Heller, 1958). Investigators searched unsuccessfully for carriers in domestic turkeys until Carpenter et al. (1989) demonstrated that they were common among survivors of outbreaks. Special techniques, as used by Carpenter et al. (1989), should be applied to the search for carriers among waterfowl and any *P. multocida* isolated must be characterized fully.

The most likely routes of transmission among birds are through bird-to-bird contact and via contaminated water. Different routes of transmission may explain different disease patterns. Avian cholera occurs among northward migrating geese in Saskatchewan each spring (Wobeser et al., 1979) but only a few birds are affected in a population of many thousands. Disease is confined almost entirely to lesser snow (*Chen c. caerulescens*) and Ross' geese (*C. rossii*), although other species share wetlands with these birds, suggesting that spread occurs within the population of white geese through direct contact. Windingstad et al. (1988) described a similar pattern among mallards in western Nebraska and Botzler (1991) included reports of scattered mortality that "never develop to extensive epizootics." This form of "smouldering" avian cholera may go undetected, unless a special effort is made to search for dead birds, and is probably more common than recognized currently. For example, we have found geese dead of avian cholera in Saskatchewan each spring since 1977 but during this 14-year period dead geese have been reported by members of the public only once. The explosive outbreak, involving hundreds or thousands of birds of many species, is the more generally recognized form of avian cholera. The pattern in many such outbreaks suggests simultaneous exposure to a common source of infection, probably contaminated water. Major outbreaks are distinctly seasonal, peaking in late winter to early spring (Friend, 1987). Mortality declines when birds disperse from the epizootic site,

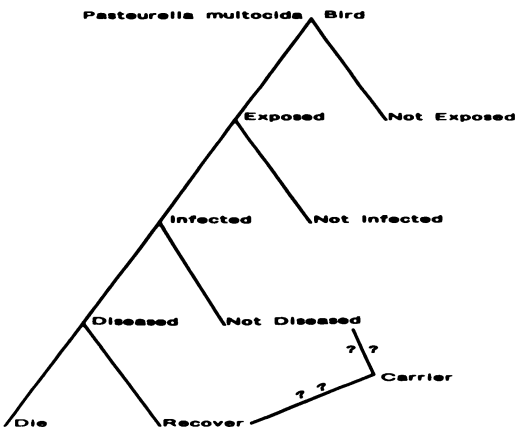


FIGURE 1. Phases in the relationship between the bacterium *Pasteurella multocida* and a bird.

but deaths continue to occur as some species migrate north. Reported summer outbreaks, with one exception, have been in colonial-nesting species such as common eider (*Somateria mollissima*) (Reed and Cousineau, 1967; Korschgen et al., 1978; Jorde et al., 1989), lesser snow and Ross' geese (Wobeser et al., 1979), and double-crested cormorants (*Phalcrocorax auritus*) (Hanson and Mutalib, 1989). The exception was an outbreak in Saskatchewan in which >4,900 ducks, primarily redheads (*Aythya americana*) concentrated for moulting, died (Wobeser and Leighton, 1988). A common feature in these outbreaks has been dense aggregation. Significant mortality among birds migrating south has been recognized twice. One instance involved birds exposed to geese returning from a die-off on the breeding grounds (Brand, 1984). The second occurred in October 1991 when >1,000 waterfowl of at least seven species died in Saskatchewan. Lesser snow geese and sandhill cranes (*Grus canadensis*) from the area of an earlier summer outbreak in the western arctic were present during the autumn outbreak.

When we consider information from the entire continent it suggests that *P. multocida* is present in at least some waterfowl populations throughout the year. The prevalence of disease is lowest in autumn,

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increasing to a peak in late winter to early spring, and then declining as birds disperse. Outbreaks occur among species concentrated during the summer and may be followed by mortality in southward migrating birds. Outbreaks on wintering and spring staging areas often involve multiple species, while mortality at other times of year is restricted to one or a few species. Colonial-nesting species, such as lesser snow geese, may maintain the bacterium over the summer because of their dense aggregation on breeding areas. There has been speculation that "certain large increasing populations of white geese may serve as reservoirs of disease that may adversely impact less abundant species or other populations" (Anonymous, 1991). Enhanced exposure and/or reduced host resistance could facilitate transition from the enzootic form maintained by bird-to-bird transmission to a water-borne epizootic.

During the past several decades there have been major habitat changes affecting wild waterfowl, as well as emergence of infectious diseases as a serious problem (Friend, 1981). The most obvious change has been continuation of the loss of wetlands that began at the time of settlement and which had resulted in loss of 53% of wetlands in the lower 48 states by the 1980's (Dahl, 1990). Stewart et al. (1987) stated "the rates of wintering habitat loss and modification have been staggering in the last 25 years, and these changes certainly are affecting the distribution and abundance of wintering waterfowl." There has also been a change in the character of wetlands, with an increase in large reservoirs (>200 ha) and a decrease in smaller wetlands (Buller, 1975; Pederson et al., 1989; Ringelman et al., 1989). Most waterfowl are gregarious, so that aggregation is normal; however, the dense concentration of birds that now occurs on staging and wintering areas (>1 million birds at some sites (Strong et al., 1991)) is probably not normal. Concentration may occur because of loss of alternate habitat, development of refuges or man-made water areas, and/or changes in agriculture that provide abundant local food but, regardless of cause, may have important effects on avian cholera. The probability of exposure to disease agents is enhanced in dense aggregations. If R_0 is defined as the average number of secondary infections attributable to a single infectious case introduced into a susceptible population, "it is intuitively reasonable to expect that R_0 should be a function of population size or density, insofar as any individual is likely to contact more individuals per unit time in a large or dense than in a small or sparse population" (Fine et al., 1982). Aggregation and changes in distribution promote mixing of populations and species that were once segregated. Recent large-scale mixing of arctic-nesting goose populations has been recognized as a problem for the management of these species (Anonymous, 1991). The prevalence of carriers of *P. multocida* among waterfowl is unknown but mixing of populations may enhance exchange of this and other agents. Outbreaks in the Central and Mississippi flyways have followed mixing of populations, some of which were known to have been previously exposed to avian cholera, and Brand (1984) suggested that carriers introduced the bacterium to new areas. *Pasteurella multocida* is not the only disease agent that may be exchanged. The effect of other agents may not be dramatic but each represents a "cost" to the host (Yuill, 1987) and the aggregate cost of exchanged parasites may render birds more susceptible to infection and/or disease when exposed to *P. multocida*.

The long-term or continual use of individual wetlands throughout the winter may be another change affecting avian cholera. For example, snow geese on traditional wintering areas along the Gulf coast were "constantly on the move from one location to another, rarely using one feeding and resting area for more than two or three weeks," whereas many now spend the entire winter on a water area (Yancey, 1976). *Pasteurella multocida* persists in

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water for days to weeks (Botzler, 1991) so that bacteria from carriers or birds dying of the disease will accumulate over time. The probability of sufficient bacteria accumulating to result in water-borne transmission is a function of the number of birds shedding bacteria, the length of time they are present on the wetland, the number of bacteria shed, the survival time of bacteria in water and the amount of water. Bird density should be calculated to include a measure of time, such as total bird-days use of the area, to account for this factor of accumulation. Density might be calculated in relation to the volume of water although, if bacteria accumulate near the surface (Botzler, 1991), area may be more important than volume.

Concentration of birds might reduce resistance to infection. "Crowding" is often considered a stressor in relation to disease but interactions between social pressure and infectious disease are not consistent or predictable (Esch et al., 1975). In chickens, resistance to some agents improved with increasing social stress, up to a point, and then declined; resistance to other agents was inversely proportional to the amount of stress (Gross, 1984). There is little information available on waterfowl. In one study, crowded ducks retained more of an administered dose of parasites than did less-crowded controls and parasites were larger and caused greater tissue damage in the crowded birds (Ould and Welch, 1980). Stress, as a result of crowding, might have a direct effect on susceptibility to avian cholera, as well as an indirect effect through enhancing other parasitic conditions that could, in turn, influence resistance to *P. multocida*.

There has been a marked shift by some waterfowl to dependence on grains from autumn to early spring; e.g., >90% of food energy demands of mallards and sandhill cranes between October and February are supplied by grain (Clark and Sugden, 1989). The situation is probably similar for many geese. Grains are deficient in specific proteins, minerals and vitamins (Baldassarre et al., 1983; Jorde et al., 1983) and natural foods are required to maintain a balanced diet (Delnicki and Reinecki, 1986; Reinecki and Krapu, 1986; Loesch and Kaminski, 1989). Nutritional deficiencies affect resistance to infectious disease (Watson, 1984; Chandra, 1988) and one nutrient, deficient in many grains, may be of particular consequence. Vitamin A has been called the "anti-infection vitamin" (Sommer, 1990) and effects of deficiency on resistance to disease agents are well-documented (Chandra and Newberne, 1977; Gross and Newberne, 1980; Beisel, 1982; Sijtsma et al., 1989; Friedman et al., 1991). Deficiency interferes with maintenance of epithelial surfaces important as barriers to infection and with antibody production and lymphocyte proliferation (Davis and Sell, 1989; Friedman and Sklan, 1989; Friedman et al., 1991). Lesions of vitamin A deficiency have been reported in black ducks (*Anas rubripes*) wintering in Massachusetts (Hagar, 1950) and mallards (*Anas platyrhynchos*) overwintering in Saskatchewan (Wobeser and Kost, 1992) but the extent of deficiency among waterfowl is unknown. Concentration of waterfowl on wetlands for an extended period may exacerbate nutritional problems through depletion of natural foods required to supplement grain (Jorde et al., 1983).

Fungal growth on grain consumed by waterfowl might also affect disease. Some birds may be infected by fungus, resulting in the disease aspergillosis; others may be poisoned by toxins produced by fungi. Waterfowl die of mycotoxicosis (Robinson et al., 1982; Windingstad et al., 1989) and sublethal exposure to mycotoxins might impair resistance to avian cholera (Smith et al., 1990).

Demand for water has increased, particularly in western North America, so that "wet" lands must compete for a share. Water available for wildlife is often of poor quality and wetlands have become a method of disposing of effluent-laden water, e.g., the Kesterson National Wildlife Refuge in

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California (Zalm, 1986), or a lowcost "innovative wastewater treatment system" (Snyder and Snyder, 1984). The most common contaminants are associated with agricultural runoff. Direct toxicity, such as that associated with selenium (Zalm, 1986; Kadlec and Smith, 1989), may be the most obvious expression of a more widespread problem. Effects of sublethal exposure to most chemicals present in runoff water are unknown, as is the interaction between these chemicals and infectious diseases. Some contaminants increase the susceptibility of mallards to *P. multocida* (Whiteley and Yuill, 1991) but methods for studying such complex interactions are in their infancy (Porter et al., 1984). The effect of contaminants on wetlands has received little attention. Turbidity, insecticides and herbicides might negatively affect the production of nutrients needed to supplement a grain diet (Cain, 1987; Reid et al., 1989). Fertilizers might increase production of some nutrients but inorganic fertilizers have been implicated in the decline of submerged aquatic vegetation in Chesapeake Bay (Hindman and Stotts, 1989). Contaminants might also alter survival of *P. multocida* in water. Windingstad et al. (1984) found a positive correlation between water salinity and occurrence of avian cholera in ponds in the Rainwater Basin of Nebraska. Certain ions (Price and Brand, 1984) and increased protein content, increased salinity and elevated temperature (Bredy and Botzler, 1989) enhance survival of the bacterium in water. Thus, contamination of wetlands with salts, organic matter and thermal pollution might enhance survival and accumulation of *P. multocida* in water to a concentration sufficient for waterborne transmission.

Many mallards, Canada geese (*Branta canadensis*) and lesser snow geese now winter north of traditional areas (Buller, 1975; Yancey, 1976; Jorde et al., 1983; Bateman et al., 1987; Pederson et al., 1989). Cold, inclement weather and food scarcity are likely to be common stressors for northern birds and might reduce resistance to infection (Olson, 1984). These birds often crowd on small areas of water kept unfrozen by wells, turbulence or thermal pollution, or use deep reservoirs that provide little natural food. Overwintering birds may contribute to a decline in water quality favorable to survival of *P. multocida* in water; usage of wetlands during winter, as well as during spring migration, increases the potential for accumulation of the bacterium (Smith et al., 1990). Birds overwintering in the north are probably more dependent on agricultural crops and more prone to nutritional deficiencies than are birds that winter to the south. For example, Jorde et al. (1983) reported that mallards wintering in Nebraska consumed a diet containing 37% less protein than did mallards wintering in Louisiana.

If asked to design a set of conditions to promote frequent occurrence of large outbreaks of avian cholera on a site, I would include: a) a large population of birds composed of a mixture of species and sub-populations to increase the likelihood of including carriers and to promote exchange of disease agents; b) restriction of the birds to a small water area for an extended time to promote accumulation of *P. multocida* in water, a high rate of contact among birds, a high degree of social stress and depletion of natural foods; c) water conditions favorable for survival of *P. multocida* (e.g., increased salinity, organic matter and thermal pollution); d) dependence of the birds on a grain diet to result in nutritional deficiencies and expose birds to mycotoxins; and e) frequent occurrence of inclement weather, food shortage, exposure to chemicals in the water, and other stressors. These conditions seem perilously similar to those experienced by many waterfowl in North America at the present time.

The model of avian cholera proposed by Botzler (1991) and the above discussion lead to at least three hypotheses that could be tested. Hypothesis 1: *Pasteurella multocida* is maintained by carrier birds within waterfowl populations. Several ques-

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tions follow from this hypothesis. Where is the bacterium carried in the body? What is the prevalence of carriers in different species? How long is the bacterium carried by individual birds? Does the prevalence of carriers change seasonally? What factors promote shedding of bacteria by carriers?

Hypothesis 2: Waterborne transmission of *Pasteurella multocida* is involved in major outbreaks of avian cholera. Several questions also follow this hypothesis. Does *P. multocida* accumulate in the water of wetlands overwinter? What factors influence survival of the organism in wetlands? What is the threshold concentration of *P. multocida* required for transmission under various circumstances?

Hypothesis 3: Nutritional deficiencies and exposure to mycotoxins and contaminants reduce resistance to avian cholera. Questions readily raised by this hypothesis include the following. How prevalent is nutritional deficiency, particularly hypovitaminosis A, among wintering waterfowl? How do nutritional deficiencies affect resistance to avian cholera? How prevalent is the consumption of mycotoxins by waterfowl? What effect does exposure to mycotoxins and contaminants in wastewater have on resistance to avian cholera?

Botzler (1991) noted that development of a model of avian cholera was hindered more by a lack of consistency in the information than by a paucity of data. Many factors that enhance exposure or reduce resistance to infection could contribute to the occurrence of epizootic avian cholera and different combinations of factors are likely to apply in different areas. Thus, there may be a variety of patterns involving various combinations of factors rather than a single model to explain all occurrences of the disease.

It is unlikely that the amount of wetland area available for wintering birds can be increased substantially, so that dispersal of birds over a greater area is not a practical solution to the problem of avian cholera. However, it may be possible to manipulate populations, wetlands and food resources to reduce exposure of birds to *P. multocida* and to factors that interfere with disease resistance. Avian cholera is a complex disease but, as stated by Leopold (1933) in regard to wildlife diseases in general: "this very complexity increases the points of attack, one of which may some day be used for control measures."

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