Influence of Disease on a Population Model of Mid-continent Mallards

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Introduction

On numerous occasions, waterfowl deaths caused by disease were highly visible to wildlife managers and to the general public. Thousands of birds died during duck plague, avian botulism and avian cholera outbreaks. Undoubtedly, some disease occurred in waterfowl populations throughout their evolution; however, knowledge of disease epizootiology primarily developed during the past 40–50 years (Wobeser 1981) for diseases that cause massive die-offs (e.g., avian cholera, avian botulism and duck plague). Other diseases, such as avian tuberculosis, aspergillosis, parasite infection and lead poisoning, also occur at chronic levels, but the data remain meager on many of these less spectacular causes of mortality and sublethal forms of disease. However, because chronic losses occur throughout the year, their cumulative effect, as well as the large die-offs, are a potential threat to waterfowl populations (Bellrose 1976, Wobeser 1981).

Previous studies (Anderson 1975) demonstrated that 50 percent of the annual mortality in mallard (Anas platyrhynchos) populations is from nonhunting causes. In addition to disease, these causes include predation, accidental deaths, inclement weather and other factors (Stout and Cornwell 1976), which can be confounded by disease. Determination of mortality rates from diseases has been difficult because many biases and inconsistencies are associated with the available data. Assessment of disease prevalence and magnitude of losses is complicated by the spatial and temporal variability of many diseases, the logistic difficulty of studying highly mobile waterfowl populations, and the potentially confounding influences of predation and scavenging on detecting disease-related mortality. Unless losses are so extensive that they direct attention to a particular area, mortality from disease is easily overlooked (see Zwank et al. 1985). Even when die-offs are evident, mortality from disease may be underestimated because sick waterfowl become debilitated, seek seclusion in dense cover and are removed by efficient predators or scavengers prior to human detection.

Our objective was to evaluate the possible effects of three of the most common diseases (Friend 1985), avian cholera, avian botulism and lead poisoning, on the population dynamics of mid-continent mallards. We used data from disease outbreaks to develop preliminary estimates of mortality rates and their temporal pattern. A computer model was used to integrate these mortality estimates with other mallard life history characteristics, evaluate the potential effects of these diseases on mallard demographics and assess the need for better information on the effects of disease on mallards.
Mallard Population Model

The Mallard Annual Cycle Model (Koford et al. 1992) was developed to integrate information on factors affecting the population dynamics of mallards in the mid-continental United States. The model considers only female mallards, because males do not seem to be limiting to productivity. Females use 15 geographic areas, with several types of nesting and foraging habitat. Attributes for each individual female in the model include age, body mass, reproductive status, molt status, geographic location, lead exposure and others. Most attribute values change stochastically, and geographic transitions are influenced by individual attributes and environmental conditions (e.g., habitat, temperature, water conditions). Stochastic and deterministic events cause mortality or other attribute changes on a daily basis for each individual. The daily probability of mortality is the sum of the daily probabilities from hunting, crippling, predation, lead poisoning, botulism and avian cholera. Thus, the model considers that all sources of mortality are additive and does not presently incorporate a mechanism for compensatory mortality. Because the model encompasses the entire year, it can be used to evaluate cross-seasonal population effects.

We used the model to evaluate the importance of diseases on mallard populations by comparing results from a base model (with baseline disease mortality) to those produced by changing the daily probabilities of disease mortality. Our primary interest was changes in the annual rate of population growth ($\lambda_2 - \lambda_1$) during 10-year simulations with an initial population of 1,000 birds. Each of these simulations was replicated 10 times to obtain an average measure of population growth for each scenario. Standard deviations of the population growth rates for these replicates were usually 0.01–0.02. Therefore, we considered changes of 0.03–0.04 to indicate important differences. In addition to population trend, we also evaluated other changes in model results, including crude rates of cause-specific mortality and lead exposure, changes in body mass, and changes in nesting success.

Disease Mortality Factors

Lead Poisoning

Hazardous levels of lead ingested and absorbed into body tissues results in lead poisoning. Lead pellets from shot shells are the most common source of lead poisoning in migratory birds (Friend 1987a). Mortality depends primarily on the dose (number of pellets ingested) and the diet (U.S. Fish and Wildlife Service [USFWS] 1986). Lead poisoning can cause death in waterfowl within 17–21 days after ingestion of a lethal dose (1–2 pellets) of lead, but acute mortality also may occur in birds that consume an overwhelming dose of lead. Mallards that survive lead ingestion usually void the lead within 20 days and remain at risk to subsequent exposure. Lead poisoning in waterfowl usually increases during autumn migration, peaks after the hunting season and remains prominent during the winter and early spring (Bellrose 1959, Sanderson and Bellrose 1986, USFWS 1986, National Wildlife Health Research Center [NWHRC] unpublished data).

Ingestion of lead shot by mallards harvested in the Mississippi flyway is among the highest reported, and has been consistently documented at about 8 percent between 1938–54 (Bellrose 1959) and 1974–82 (Sanderson and Bellrose 1986). Ingestion of
lead shot by mallards has been lower (3–4 percent) in wintering areas of the Central Flyway (Sanderson and Bellrose 1986, USFWS 1986). However, estimates of lead exposure derived from gizzard analysis may provide conservative estimates of lead exposure (Anderson and Havera 1985) and these estimates require correction for the increased vulnerability of lead-exposed mallards to hunting. Bellrose (1959) estimated a correction factor of 1.65 from band returns, and other researchers estimated hunting vulnerability at 2–3.5 (M. Heitmeyer personal communication), 2.0 for geese (DeStefano 1989:23–24), and 1.0 (Ankney and Dennis 1982). For our analysis, we used a 2.0 correction factor to obtain an estimated 4.0 percent natural rate of exposure to lead. We combined mortality estimates for number of ingested shot (Bellrose 1959) with the distribution of ingested shot (Bellrose 1959) to obtain a weighted probability of mortality of 0.18 for all mallards ingesting lead shot. The resulting 20-day mortality rate (0.04 × 0.18 = 0.0072) was converted to an average daily probability of mortality (0.0004), which was then adjusted for seasonal changes in lead exposure (Table 1). In addition to mortality from lead poisoning, ducks that ingest lead also lose body mass (Sanderson and Bellrose 1986). We estimated mass loss for ducks that did not die from lead ingestion at 0.84 percent of body mass per day for 20 days (Sanderson and Bellrose 1986:18–19).

Avian Botulism

Avian botulism is caused by ingestion of a neurotoxin produced by the bacterium Clostridium botulinum type C, which is widely distributed in wetlands and found in marsh invertebrates (Jensen and Allen 1960, Duncan and Jensen 1976). Waterfowl inadvertently ingest type C botulism toxin while feeding, and die from paralysis or from drowning (Rosen 1971a). Avian botulism has been recognized as a major killer of wild waterfowl since the early 1930s (Giltnert and Couch 1930, Kalmbach and Gunderson 1934), and continues to cause die-offs which vary greatly among years, sites and species. Losses of more than 1,000,000 birds have occurred during localized outbreaks in a single year (Locke and Friend 1987). In endemic areas, losses of 5,000 birds or more can frequently occur. Avian botulism occurs almost yearly, and losses are extensive in mallard breeding areas on the prairies (Locke and Friend 1987). Frequent losses also occur in Minnesota, Wisconsin, Illinois, Iowa and Nebraska. Avian botulism is the primary disease affecting mallards during the post-breeding phase of the annual cycle. Outbreaks occur primarily during July through September, but can occur in December and January or occasionally during early spring (Locke and Friend 1987). Control of this disease has focused on regulating water levels and on collecting avian carcasses to prevent further botulism toxin production. Recent experimental studies indicated that, during some outbreaks daily carcass pickup can reduce botulism mortality rates for mallards four to five times (Reed and Rocke 1992).

Avian Cholera

Most species of waterfowl are susceptible to avian cholera, an infectious disease caused by the bacterium, Pasteurella multocida (Rosen 1971b). The earliest documented record of avian cholera in North American wild ducks was reported in Texas (Quotrurup et al. 1946), and periodic outbreaks among ducks have occurred since that time (Petrides and Bryant 1951, Rosen 1971b, Brand 1984). Avian cholera epizootics usually occur at waterfowl concentration areas, most commonly in the Pacific and
Central flyways, where mortality may exceed 1,000 birds per day, and death may follow exposure by 6–12 hours (Friend 1987b). Avian cholera mortality has occurred annually since 1975 (Zinkl et al. 1977) in Nebraska’s Rainwater Basin, where mallards are the most frequently affected species (Windingstad et al. 1984, 1988). Avian cholera mortality in mallards also was reported in northwest Missouri and southwest Iowa during 1963 (Vaught et al. 1967), and this area has become enzootic for avian cholera (Brand 1984, NWHRC unpublished data, Windingstad et al. in press). In other circumstances, however, the disease may become a chronic infection without causing mass mortality (Botzler 1991). Losses can occur at any time of the year, but typically occur during the non-breeding season when waterfowl are concentrated. Central flyway outbreaks peak in winter and continue during spring migration.

**Avian Botulism and Cholera Mortality**

We obtained reports of avian cholera and botulism losses of mallards during 1979–88 in the Central and Mississippi flyways from federal wildlife refuges and wetland management districts, state wildlife agencies, the Canadian Wildlife Service, and Ducks Unlimited—Canada. Epizootic files at the NWHRC also were reviewed to obtain additional information on losses of mallards from disease. Daily mortality rates during outbreaks were calculated from 124 reports, with estimates of the mallard population at risk, dates delimiting the start and end of a mallard die-off, and either the number of retrieved mallard carcasses or the estimated number of dead mallards. Daily mortality rates for each outbreak were estimated by the Mayfield method (Johnson 1979). We log-transformed these daily mortality rates (Heisey and Fuller 1985) to produce normal distributions and calculated seasonal means and approximate standard errors (Table 1). For botulism mortality, we also modified the seasonal mortality rates to account for a linear increase in reported botulism mortality beginning in July, peaking in mid-August and declining until late September (NWHRC unpublished data).

The estimated daily mortality rates for the Mallard Annual Cycle Model represent the conditional probability of mortality given an outbreak and, therefore, likely include two opposing biases. Estimates of disease mortality are conservative because scavengers can dispose of 80–90 percent of the available carcasses within three days (Humburg et al. 1983, Stutzenbaker et al. 1983). Furthermore, carcasses usually are difficult to find. Humburg et al. (1983) located only one-fourth of the carcasses planted in quadrats at Swan Lake Refuge, and Stutzenbaker et al. (1983) reported that less than 10 percent of planted carcasses could be located within 30 minutes. In contrast, not all mallards in the population will be in areas where disease outbreaks occur and, thus, are not exposed to the risk of mortality from disease. Unfortunately, there is no available data on the proportion of mallard populations that is annually exposed to either avian botulism or cholera outbreaks. Because avian botulism is a widespread disease throughout the breeding and post-breeding areas of mid-continent mallard populations, we assumed that the underestimates of daily mortality were approximately balanced by applying the mortality rates to all birds in the population. However, most avian cholera die-offs occurred at endemic areas and are not representative of the entire population of mallards. Therefore, we reduced the estimated avian cholera mortality during the winter (from 0.0008 to 0.0004) and used the coefficient of variation to compute an new standard error (Table 1).
Table 1. Estimated daily mortality rates from diseases in mid-continent mallards, by season and number of die-offs (n) used to estimate mortality.

<table>
<thead>
<tr>
<th>Season</th>
<th>n</th>
<th>Estimated daily mortality rate</th>
<th>Mortality ± 1 SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breeding/Post-breeding</td>
<td>87</td>
<td>0.0005</td>
<td>0.00040–0.00065</td>
</tr>
<tr>
<td>(April 1–September 30)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Botulism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fall migration</td>
<td>15</td>
<td>0.0002</td>
<td>0.00010–0.00044</td>
</tr>
<tr>
<td>(October 1–December 15)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avian cholera</td>
<td></td>
<td>0.0001</td>
<td>0.00005–0.00022</td>
</tr>
<tr>
<td>Botulism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lead poisoning</td>
<td></td>
<td>0.0003</td>
<td>0.00005–0.00055</td>
</tr>
<tr>
<td>Winter</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(December 16–February 28)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avian cholera</td>
<td></td>
<td>0.0004</td>
<td>0.00024–0.00070</td>
</tr>
<tr>
<td>Lead poisoning</td>
<td></td>
<td>0.0004</td>
<td>0.00015–0.00065</td>
</tr>
<tr>
<td>Spring migration</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(March 1–March 31)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avian cholera</td>
<td></td>
<td>0.0002</td>
<td>0.00010–0.00044</td>
</tr>
<tr>
<td>Lead poisoning</td>
<td></td>
<td>0.0002</td>
<td>0.00010–0.00045</td>
</tr>
</tbody>
</table>

Simulation Results

Mortality patterns for the base model (Figure 1) show that disease mortality peaks in the summer and winter. Avian botulism is the primary cause of summer mortality. Disease mortality declines during the fall to early winter, when avian botulism is replaced by avian cholera and lead poisoning. Mortality from lead poisoning and avian cholera increases during the winter and begins to decline from late winter toward early spring. Mortality from these three diseases is at a minimum during the breeding period.

Model Sensitivity

Variation of our seasonal estimates of daily mortality rates for each disease was considerable. Sources of variation among outbreaks included annual differences, spatial differences and minor sampling variation for each outbreak. We conducted a sensitivity analysis to determine whether variation in our estimates of disease mortality had a substantial impact on base model results. The mean daily probability of mortality for each disease was modified by ± 1 SE (Table 1), which provided a range of values with a 68 percent chance of containing a mean daily probability of mortality during outbreaks. Differences in average annual population growth rates between the base and modified models were used to evaluate the relative sensitivity of model outputs. Growth rates were most sensitive to mortality rates for avian botulism and avian cholera, and marginally sensitive to mortality rates for lead poisoning (Table 2). Simultaneous changes in all three mortality factors indicated a potential for considerable variation in mortality from disease if estimates of all three factors were highly correlated.

Figure 1. Seasonal (within 28-day intervals) mortality of female mallards from avian botulism (\textbackslash\text\textbackslash), lead poisoning (\textbullet) and avian cholera (\textbar). Mortality standardized per 10,000 birds and estimated from the average annual values from two 10-year simulations with initial populations of 1,000 birds.

**Lead Poisoning**

Bellrose (1959) estimated that 4 percent of the mallards in the Mississippi flyway were lost annually to lead poisoning. Sanderson and Bellrose (1986) estimated that 30–40 percent of all ducks ingest lead in any given year. They also indicated that losses from lead poisoning occur most frequently during winter and spring. Results from our base model generally coincide with these predictions. Base model results indicated an average annual lead poisoning mortality of 4.8 percent with an additional 20.4 percent of the birds ingesting lead shot. Seasonal patterns of lead exposure and

<table>
<thead>
<tr>
<th>Mortality factor</th>
<th>-1 SE</th>
<th>+1 SE</th>
<th>Reduction in mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>50 percent</td>
</tr>
<tr>
<td>Botulism</td>
<td>+0.035</td>
<td>-0.014</td>
<td>0.048</td>
</tr>
<tr>
<td>Avian cholera</td>
<td>+0.029</td>
<td>-0.018</td>
<td>0.034</td>
</tr>
<tr>
<td>Lead poisoning</td>
<td>+0.023</td>
<td>-0.008</td>
<td>0.045</td>
</tr>
<tr>
<td>Botulism, avian cholera and lead poisoning</td>
<td>+0.062</td>
<td>-0.072</td>
<td>0.100</td>
</tr>
</tbody>
</table>
lead poisoning in the model began to increase in October, and reach a peak in late December or early January.

The amount of lead poisoning in waterfowl and secondary poisoning in other species has generated considerable controversy in the United States. A national ban on lead shot for waterfowl hunting was instituted beginning in 1991, and should result in decreased levels of lead poisoning in waterfowl. We used the Mallard Annual Cycle Model to simulate the population effects of conversion from lead to nontoxic shot. Reductions in lead shot exposure and lead poisoning by 50 percent and 75 percent resulted in average annual population growth rates of 0.045 and 0.061, respectively, above the base model (Table 2). The lead poisoning mortality rate was reduced from 4.8 percent to 2.5 percent and 1.3 percent by the simulated reduction in available lead shot. The proportion of birds with sublethal lead exposure was correspondingly reduced from 0.204 to 0.106 and 0.055. This reduced exposure also contributed to increased average body mass (measured annually on January 15) of adult females from 1,083 gm (SD = 5.8) to 1,094 gm (SD = 3.8) and 1,100 gm (SD = 4.1) when lead exposure was reduced by 50 percent and 75 percent, respectively. However, body mass of young birds (hatched the previous year) was not substantially increased (1,021 gm to 1,028 gm) when lead exposure was reduced by 75 percent. These increases in average body mass had little cross-seasonal effect on spring productivity; nesting success (proportion of females with a successful nest) increased from 0.24 (SD = 0.01) in the base model to 0.26 (SD = 0.005) when lead exposure was reduced by 75 percent.

**Avian Botulism**

Little speculation has been offered regarding the impact of avian botulism on waterfowl populations, perhaps due to the inherent difficulties of assessing population effects (Jensen and Price 1987). The Mallard Annual Cycle Model reported annual botulism mortality of 5 percent in adult birds with a seasonal pattern that begins to increase in late June, and peaks in August to early September (Figure 1). This seasonal pattern of mortality has been reported by other studies (see Locke and Friend 1987).

Considerable effort and resources have been expended on carcass removal to control botulism (Parrish and Hunter 1969), but these efforts have received little evaluation (Wobeser 1987, Reed and Rocke 1992). We used model simulations to evaluate the effects of reducing the occurrence of avian botulism outbreaks and to evaluate the potential impact that carcass pickup efforts have on mallard populations. Reduction in the daily probability of botulism mortality by 50 percent and 75 percent had a corresponding effect on annual botulism mortality, and resulted in average annual population growth rate increases of 0.048 and 0.053, respectively, in comparison to the base model (Table 2). We also increased botulism mortality probabilities by two or three times to simulate a reduction in the current management efforts to remove carcasses or haze birds during outbreaks. These simulations showed respective decreases in the average annual population growth rate of 0.035 and 0.076.

**Avian Cholera**

Little is known about the impact of avian cholera on waterfowl populations (Botzler 1991). Our base model produced annual avian cholera mortality in 4.5 percent of the mallard population. In contrast, Rosen (1971b) estimated that 2 percent of the duck population in California was lost to avian cholera in some years. This estimate
may be high, even for duck populations in California, where avian cholera occurs annually (Botzler 1991). However, recent increases in the distribution and frequency of avian cholera into the Central flyway and western Saskatchewan makes assessment of the situation difficult. Disease mortality from the model generally follows the predicted pattern of winter and spring mortality (Figure 1). However, the Mallard Annual Cycle model currently has avian cholera mortality beginning during autumn and continuing into winter.

Birds that die from avian cholera often discharge large volumes of *P. multocida* organisms, which can survive outside the carcass for several months. Considerable efforts have been expended to collect and dispose of waterfowl carcasses, manipulated environmental conditions, or control bird movement to reduce avian cholera losses once an outbreak has been initiated. Although these procedures are logical, no data are available to evaluate the benefits of these activities (Botzler 1991). We simulated the possible impact of decreasing these control activities by increasing avian cholera mortality probabilities by 2 or 3 times the base rates. These simulations reduced average annual population growth by 0.044 and 0.075, respectively. We also evaluated potential management strategies (Habitat manipulation, disinfection of small bodies of water, or vaccination) aimed at preventing avian cholera outbreaks by reducing the daily probability of avian cholera mortality by 50 percent and 75 percent. These reductions in avian cholera mortality produced 0.034 and 0.056 average annual population growth increases over the base model (Table 2).

*Lead Poisoning, Avian Botulism and Avian Cholera*

The combined effects of disease may account for a large proportion of the non-hunting mortality (Bellrose 1976, Stout and Cornwell 1976). We evaluated the simultaneous reduction of all disease mortality probabilities by 50 percent and 75 percent. These reductions in mortality increased the annual population growth rates by 0.100 and 0.139, respectively (Table 2).

**Discussion**

Although diseases among wild waterfowl have long been recognized and have received increased attention in recent years, few estimates are available of the annual waterfowl mortality rates from disease. Even reported estimates were usually incomplete, based on crude extrapolations for a single disease, or educated guesses. Determination of mortality rates from diseases in mallards continues to be a difficult undertaking because of the annual variation in mortality from disease, spatial scale of disease outbreaks, the high mobility of waterfowl, difficulty in estimating number of birds that die during outbreaks, interactions between mortality from diseases, predation and scavenging, and other confounding factors. Our estimates of mortality from diseases certainly are susceptible to many of these difficulties and potential biases. Nevertheless, management of wildlife populations is a complicated task, often requiring decisions with such limited data (Cowardin and Johnson 1979). The development of simple models, and the geographic and temporal representation of these models can facilitate logical and orderly development of management and research. In this context, our results can provide a useful starting point to identify potential areas for further research on diseases, to focus on the potential effects of disease on
waterfowl and to provide a preliminary evaluation of overall management to reduce mortality from disease.

Sensitivity analyses indicated that more reliable estimates of the daily probability of mortality are needed, especially for avian botulism and avian cholera. Errors in the mean estimates of these daily probabilities could have a substantial effect on model predictions of population growth. The importance of spatial and annual variation in these mortality sources needs further investigation. In addition, improved estimates of the proportion of birds that are at risk to avian botulism and cholera outbreaks are necessary. In contrast, probability estimates of lead poisoning mortality seem sufficiently robust to provide reasonable model predictions. These findings are not surprising because more research has been conducted on the ingestion, mortality and physiological effects of lead poisoning on waterfowl than on avian botulism or avian cholera.

Efforts to manage waterfowl diseases can take several different approaches, depending on the epizootiology of the specific disease. One approach is to reduce the risk of initiating a die-off. Several general strategies for this include manipulation of the environment to produce conditions that are unfavorable to the disease agent, disinfection of disease hotspots, control of bird populations and immunization to reduce the number of susceptible birds. Model simulations indicate that moderate reductions (less than or equal to 50 percent) in mortality rates from lead poisoning, avian cholera or avian botulism are potentially beneficial to mallard population growth. Development of management strategies to achieve these results require research to identify critical factors in the epizootiology of avian cholera and avian botulism.

A second approach to control of disease is to reduce (or manage) mortality after the onset of an outbreak. This is the current method for management of waterfowl diseases and will probably continue because potentially beneficial action is better than doing nothing, and visible action demonstrates good intentions (Peterson 1991). Control actions usually are monitoring and early detection of mortality, carcass collection, water manipulation, and control of bird movement. Results from the model simulations indicated that, if present management has reduced avian botulism and avian cholera mortality by 2 times, these activities could have a noticeable benefit for mallard population levels. However, further research to develop alternatives and evaluate present management is needed.

The primary approach to control of lead shot ingestion and poisoning has been a nationwide conversion to nontoxic (steel) shot, beginning in 1991. Conversion to steel shot can increase the number of waterfowl with ingested steel and presumably decrease the number with lead shot (Calle et al. 1982, DeStefano et al. 1991). However, spent shot on some areas will remain in the environment and continue to be consumed by waterfowl (Mauer et al. 1990, DeStefano et al. 1991). Furthermore, poor compliance with nontoxic shot regulations (Simpson 1989) and continued use of lead shot in Canada (e.g., DeStefano et al. 1991, Schwab and Daury 1989) will provide new sources of lead shot. Habitat manipulations (Sanderson and Bellrose 1986) may be required to reduce the availability of lead shot in selected areas. Model simulations indicated that a 50–75 percent reduction in lead poisoning potentially benefitted mallard population growth. Concurrent reductions in lead exposure also increased mean body mass. An increased body mass of waterfowl is believed to result in increased survival (Haramis et al. 1986, Hepp et al. 1986) and earlier molt
Management to reduce the risk of disease outbreaks or control mortality in mallards will undoubtedly have many positive benefits on other wild bird species. In addition to mallard populations, many other waterfowl, nongame and endangered species are susceptible to avian botulism, avian cholera and lead poisoning. Waterfowl, shorebirds and some mammals commonly are affected by type C botulism (Locke and Friend 1987). Avian cholera naturally infects over 100 wild avian species, and most bird species are probably susceptible (Botzler 1991). As wild waterfowl become increasingly concentrated on a limited habitat base, infectious diseases, such as avian cholera, become an ever greater concern for waterfowl managers. Dabbling ducks, especially the mallard and pintail (Anas acuta), have been the primary victims of lead poisoning, although diving ducks, geese and swans also have suffered significant mortality. Furthermore, eagles frequently die from lead poisoning after ingesting lead shot embedded in the flesh of their prey (Friend 1987a).

Our modelling has several important limitations that must be emphasized. Our estimates of mortality from disease (especially avian cholera and avian botulism) are, at best, preliminary. Biases in the magnitude of these estimates could have important implications for our conclusions about the potential population effects of reductions in these mortality sources. In particular, our estimates of avian cholera mortality may be excessive, and represent a worst-case scenario in which a substantial proportion of the mid-continent mallard population is at risk during avian cholera outbreaks. Modelling results of avian botulism also may represent above average years of botulism outbreaks. Because of these limitations, our results should not be used to represent the average effects of disease on the mid-continent mallard population. Further research and data collection are necessary to validate or improve these mortality estimates. Nor should the present model be viewed as encompassing all diseases of mallard populations. To a limited extent, other diseases also are represented in the estimates associated with avian cholera and botulism. This situation is inevitable because determination of the cause of mortality of all individuals in a die-off is not practical. Whereas other diseases are probably of lesser importance, their potential effect on mortality and population growth in mallards may have been underestimated.

Finally, the role of disease is part of a complex web involving many other ecological factors. In addition to causing direct mortality, diseases may increase the risk of other mortality factors, including hunting and predation (Johnson et al. 1987). The Mallard Annual Cycle Model does not consider such relationships among the various mortality sources. This is an important limitation that results from the paucity of available data on the nature of compensatory mortality in mallards, the specific functional relations between different mortality factors and the importance of density dependence in disease mortality. If density dependence occurs for disease or other mortality sources, our model simulations could exaggerate the benefits of reducing disease loss on mallard population growth. Evidence to support the concept of compensatory hunting mortality for female mallards is presently inconclusive (Burnham et al. 1984), but may be partially compensatory (Johnson et al. 1987). Conroy and Krementz (1990) indicated that predicted relationships between nonhunting mortality and hunting mortality, or population density for the additive or compensatory mor-

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tality hypotheses have not been tested. They recommend that experimental management to evaluate hunting mortality (e.g., Anderson et al. 1987) also examine the relationships among harvest, nonhunting mortality and population density. We believe that experimental programs to evaluate the effect of hunting also should consider the importance of different sources of nonhunting mortality (e.g., disease, predation and weather), the density dependent nature of these factors, and identification of factors that can be managed to enhance mallard and other waterfowl populations.

Acknowledgments


References


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