SITE-SPECIFIC LEAD EXPOSURE FROM LEAD PELLET INGESTION IN SENTINEL MALLARDS

TONIE E. ROCKE,[1] U.S. Fish and Wildlife Service, National Wildlife Health Research Center, 6006 Schroeder Rd., Madison, WI 53711, USA
CHRISTOPHER J. BRAND,[1] U.S. Fish and Wildlife Service, National Wildlife Health Research Center, 6006 Schroeder Rd., Madison, WI 53711, USA
JOHN G. MENSIK, U.S. Fish and Wildlife Service, Sacramento National Wildlife Refuge, 752 County Rd. 99W, Willows, CA 95988, USA

Abstract: We monitored lead poisoning from the ingestion of spent lead pellets in sentinel mallards (*Anas platyhrynchos*) at the Sacramento National Wildlife Refuge (SNWR), Willows, California for 4 years (1986–89) after the conversion to steel shot for waterfowl hunting on refuges in 1986. Sentinel mallards were held in 1.6-ha enclosures in 1 hunted (P8) and 2 non-hunted (T19 and TF) wetlands. We compared site-specific rates of lead exposure, as determined by periodic measurement of blood lead concentrations, and lead poisoning mortality between wetlands with different lead pellet densities, between seasons, and between male and female sentinels. In 1986, the estimated 2-week rate of lead exposure was significantly higher ($P < 0.005$) in P8 (43.8%), the wetland with the highest density of spent lead pellets (>2,000,000 pellets/ha), than in those with lower densities of lead pellets, T19 (18.1%; 173,200 pellets/ha) and TF (0.9%; 15,750 pellets/ha). The probability of mortality from lead poisoning was also significantly higher ($P < 0.01$) in sentinel mallards enclosed in P8 (0.25) than T19 (0) and TF (0) in 1986 and remained significantly higher ($P < 0.001$) during the 4-year study. Both lead exposure and the probability of lead poisoning mortality in P8 were significantly higher ($P < 0.001$) in the fall of 1986 (43.8%; 0.25), before hunting season, than in the spring of 1987 (21.6%; 0.04), after hunting season. We found no significant differences in the rates of lead exposure or lead poisoning mortality between male and female sentinel mallards. The results of this study demonstrate that in some locations, lead exposure and lead poisoning in waterfowl will continue to occur despite the conversion to steel shot for waterfowl hunting.

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Key words: lead exposure, lead pellets, lead poisoning, lead toxicity, mallards, mortality, Sacramento National Wildlife Refuge, sentinels, waterfowl, wetlands.

Since Bellrose (1959) estimated that between 2 and 3% of North American waterfowl died annually from lead poisoning, numerous surveys of hunter-killed birds have documented the prevalence of ingested lead pellets in waterfowl gizzards (Sanderson and Bellrose 1986). More recent surveys used lead concentrations in tissues (blood, liver, bone) or protoporphyrin concentrations in blood as a measure of lead exposure in waterfowl (Dieter 1979, Stendell et al. 1979, Scanlon et al. 1980, Anderson and Havera 1985). Unfortunately, the site of lead pellet ingestion could not be determined conclusively. Lead exposure detected in a population at one location may represent lead pellet ingestion at any number of other locations. Little is known about the site-specific risk of pellet ingestion in relation to the density and availability of pellets in the environment.

The use of sentinel animals in disease studies provides a method for obtaining site-specific information on exposure to disease agents in a controlled population. In 1986–89, we used captive-reared mallards as sentinels to monitor site-specific exposure to the toxin causing avian (Type C) botulism in wetlands in the Central Valley of California (Rocke and Brand 1994). During these studies, lead exposure and lead poisoning mortality were detected in sentinel mallards in certain wetlands. To learn more about the dynamics of lead exposure and lead poisoning in mallards where the site of lead pellet ingestion was known, we monitored blood lead levels, ingestion rates and mortality of sentinel mallards. Our objectives were to determine site-specific rates of lead exposure and lead poisoning mortality in sentinel mallards, seasonal variations in these rates, and rate of exposure in relation to lead pellet density in wetland sediments.

[1] Present address: U.S. National Biological Service, National Wildlife Health Center, 6006 Schroeder Rd., Madison, WI 53711.

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STUDY AREA

We conducted the study at SNWR, Willows, California (39°29'N, 122°20'W), a 4,300-ha refuge divided into about 70 habitat units managed primarily for wetlands and associated wildlife species. Three wetland units were used as study sites. A 1.6-ha enclosure was constructed in each study site to confine sentinel ducks as described by Rocke and Brand (1994). One site (P8) was located in an area that was open to game bird hunting; the others (TF and T19) were located in the "closed zone", but were adjacent to hunting areas on SNWR and surrounding private land. These sites were typical of refuge wetlands: water levels were <1 m, and major vegetation included scattered southern cattail (*Typha domingensis*) and Tule bulrush (*Scirpus acutus*) clumps, smartweed (*Polygonum* spp.), and swamp timothy (*Crypsis schoenoides*). Wetland sediments generally consisted of a detrital layer of decomposing vegetation and mud up to 10 cm deep overlaying a hard clay pan bottom of the Willows Series (Begg, 1968).

METHODS

Experimental Design and Data Collection

We estimated pellet densities in the sediments of each study site using a core sampler constructed of galvanized metal that measured 64-cm^2 circular surface area by 10 cm deep. Two hundred core samples were taken within the enclosure in P8 during October 1986 and May 1987, and 100 samples were taken within each of the enclosures in TF and T19 during August 1987. We randomly located sampling sites along 8 equidistant transects (25/transect) in P8 and 4 transects (25/transect) in each of TF and T19. Core samples were placed in plastic bags, radiographed or fluoroscoped to detect pellets, and rinsed through a series of sieves to recover and identify shot. We identified pellets as lead or steel with a magnet, and by pellet's resistance to crushing by shears or pliers. We estimated pellet density (pellets/ha) from the mean number of pellets and surface area for each core sample.

Captive-reared mallards (mixed sex and age), obtained from Wild Wings of Oneka (Hugo, Minn.), were used as sentinels. Each sentinel was wing-clipped; numbered poultry bands were placed on each leg, and a numbered plastic tag was attached to the left patagium. In July of each year (1986–89), about 50 sentinels were placed in enclosures in all 3 study sites and maintained through October or until hunting season began (the last week of Oct). In February through May 1987, we also placed sentinels in the enclosure in the hunted wetland (P8) to monitor lead exposure after the hunting season. Because we had no prior knowledge of sex-specific differences in lead ingestion, lead exposure or lead poisoning mortality, no attempts were made to equalize the number of male and female sentinels in each enclosure. Although all sentinels had access to natural foods in enclosures, we periodically provided supplemental waste grains (mostly rice) and grit.

At intervals in July-October 1986 and February-May 1987, sentinel birds were captured to determine the presence of ingested shot by fluoroscopy (Radifluor 360, Assoc. X-Ray Techs, Mountain Valley, Calif.). A 2-mL blood sample was collected from each bird by jugular venipuncture and placed in Vacutainer tubes containing sodium heparin (Becton-Dickinson, Rutherford, N.J.), inverted at least 10 times, and frozen for lead analysis. After thawing, aliquots of the sample were diluted tenfold in a solution containing 0.5% Triton X-100 (alkylaryl polyether alcohol; J. T. Baker Chem. Co., Phillipsburg, N.J.) and 0.2% ammonium dihydrogen phosphate and analyzed for lead concentration by atomic absorption spectrophotometry (Perkin-Elmer Model 2380; Perkin-Elmer Anal. Instruments, Norwalk, Conn.) with methods described by Fernandez and Hilligoss (1982). Birds with blood lead concentrations ≥0.2 ppm were considered exposed to lead (Friend 1985).

We collected data on lead poisoning mortality during all 4 years of the study. Two or more people, on foot or in canoes and accompanied by 1–2 retrieving dogs, searched each enclosure intensively to recover sick and dead birds 5–6 days/week. Sick or dead sentinel birds were removed and replaced with healthy birds to maintain the number of sentinels in each enclosure. During each search, live sentinel birds were also

counted. Sick sentinel birds were killed by cervical dislocation or CO_2 asphyxiation. All birds that were killed or found dead were necropsied to determine the cause of morbidity or death. We recorded gross lesions. Gizzard contents were examined manually and by fluoroscopy for pellets. We tested blood samples for botulism toxin using the standard mouse protection test (Quortrup and Sudheimer 1943), and livers were cultured on blood agar media to determine the presence of bacterial pathogens. Other standard diagnostic procedures were used if the need was indicated by necropsy observations (Hitchner et al. 1980, Lennette et al. 1985). Livers for lead analysis were kept frozen until assay. After thawing, liver tissue samples were ground, ashed in a muffle furnace and then digested in a solution of nitric and hydrochloric acid. Lead concentrations were determined by atomic absorption spectrophotometry with the methods of Boyer (1984). Lead standards and blank crucibles were processed with each batch of 25–30 samples for quality control. Birds with liver lead concentrations ≥8.0 ppm (wet wt) accompanied by lesions consistent with lead toxicosis (Cook and Trainer 1966, Wobeser 1981, Friend 1985) were considered lead poisoned (Friend 1985, Friend et al. 1987). Birds with liver lead concentrations ≥2.0 ppm (wet wt), but <8.0 ppm, were considered to be exposed to lead (Bagley and Locke 1967); in these cases, lead poisoning was suspected to have contributed to morbidity or mortality only if lesions consistent with lead toxicosis were present.

Data Analysis

The probability of lead exposure in sentinel birds was determined from consecutive sampling of birds for blood lead concentrations. Because sentinel birds were sampled several times at intervals of varying length, and lead exposure is reversible (i.e., elevated blood lead levels can decline over time to levels <0.2 ppm), we could not simply calculate the cumulative proportion of birds exposed. Therefore, we used a simple first order Markov model (Bishop et al. 1975) to accommodate our sampling structure and to standardize the data for subsequent statistical analysis of lead exposure rates. Each sampling interval represented a binomial trial, conditioned on the previous lead exposure status, where the transition probabilities between unexposed and exposed states were estimated. We assumed these transition probabilities remained constant during each time period. We estimated a 2-week transition probability to standardize the exposure rates as follows. If i indicates the subject's current state (1 = not exposed, 2 = exposed) and j indicates the subject's state 2 weeks later, then the probability (P_{ij}) that an unexposed subject becomes exposed within 2 weeks is P_{12}. For our data, the interval length between sampling each bird was rounded up to the nearest number of 2-week intervals, say k. Then, a likelihood was constructed from the appropriate terms of P_{ij}^k. Iterative reweighted least squares procedures (PROC NLIN) were used to maximize the resulting likelihood to obtain the best estimates of P_{12} (2-week exposure rates) and their standard errors (SAS Inst. Inc. 1989). A Z test was used to compare lead exposure rates in sentinel birds among enclosures (P8, TF, T19). Because we hypothesized that sentinel birds enclosed in pools with higher lead pellet densities would have higher rates of lead exposure, we compared the Z-statistic to a table of critical values for a 1-sided distribution. A 2-sided Z test was used for comparing rates of lead exposure between males and females in each pool and between seasons in 1 pool (P8).

To compare lead poisoning mortality between enclosures and years and between male and female sentinels, we used cumulative probabilities of mortality (Reed and Rocke 1992). We used the staggered entry Kaplan-Meier survival estimator (Kaplan and Meier 1958, Pollock et al. 1989) that allows for new animals to be added during the study. To approximate the number of birds at risk at any particular time, any missing sentinels (birds that escaped from the enclosures or could not be accounted for) were counted as half a bird at risk during the interval between the last date they were observed and the date they were known to be missing (Harris et al. 1950, Crowley and Breslow 1984, Rocke and Brand 1994). We used a Chi-square log rank test (Pollock et al. 1989) to compare cumulative probabilities of lead poisoning mortality between enclosures. The Chi-square statistic was converted to a Z-statistic with the appropriate sign (Crowley and Breslow 1984) and compared to a table of critical values for a 1-sided distribution. Overall trends in the rates of lead poisoning between enclosures were analyzed by pooling the Z-statistic from each of 4 years to produce a composite Z-statistic (Anderson and Burnham 1976, Reed and Rocke 1992). A similar approach was used to compare

Table 1. Pellet ingestion (as determined by fluoroscopy) by captive-reared mallard sentinels held in a 1.6-ha enclosure in a hunted wetland (P8) at the Sacramento National Wildlife Refuge from July to October 1986 (before hunting season) and February to April 1987 (after hunting season).

Month	*n*	% with ingested shot	Mean shot ingested
Aug	57	44	6.3
Sep	57	11	1.7
Oct	57	30	5.9
Feb	101	11	1.5
Mar	69	9	3.0
Apr	64	11	1.7

rates of lead poisoning mortality between male and female sentinels (of the same age group) held in the same enclosure, but the Z-statistics were compared to a table of critical values for a 2-sided distribution.

RESULTS

During October 1986, before the hunting season, we recovered 272 lead pellets from 200 sediment cores collected within the P8 enclosure (1.36 ± 0.089 pellets/core sample) and estimated a density of 2,142,200 pellets/ha; none of the pellets recovered were steel. In May 1987, we estimated 2,299,700 pellets/ha within the P8 enclosure (1.46 ± 0.107 pellets/sample); 3 of the 309 pellets recovered were steel. Within enclosures in T19 and TF in 1986, we estimated 173,200 and 15,750 pellets/ha, respectively (0.11 ± 0.034 and 0.01 ± 0.010 pellets/sample); all pellets recovered were lead.

Ingested pellets were found in the gizzards of birds in P8 (Table 1) throughout the fall of 1986 and spring of 1987. The number of pellets found in gizzards ranged from 1 to 29. In T19, only 4 of 67 birds were found with ingested pellets in the fall of 1986, and none were found in TF.

Table 2. Lead exposure in captive-reared mallard sentinels maintained in enclosures at the Sacramento National Wildlife Refuge in July to October 1986 as determined by blood lead concentrations. Birds were considered to have become exposed if the second of 2 consecutive blood samples had ≥0.2 ppm lead.

Pool	*n*[a]	No. lead-exposed	2-week exposure rate (%)[b]
P8	174	79	43.8[c]
T19	86	29	18.1[d]
TF	160	7	0.9

[a] No. of paired blood samples tested for lead.
[b] Max. likelihood estimate (%) of an unexposed bird becoming exposed to lead in a 2-week interval.
[c] The 2-week lead exposure rate for P8 was significantly different from both T19 (1-sided $Z = 2.67$, $P = 0.0038$) and TF (1-sided $Z = 11.7$, $P < 0.001$).
[d] The 2-week lead exposure rate for T19 was significantly different from TF (1-sided $Z = 1.85$, $P = 0.032$).

Table 3. Lead exposure in captive-reared mallard sentinels maintained in an enclosure in a hunted wetland (P8) at the Sacramento National Wildlife Refuge in the fall (Jul–Oct 1986), before hunting season, and in the spring (Feb–Apr 1987), after hunting season. Birds were considered lead-exposed if the second of 2 consecutive blood samples had ≥0.2 ppm lead.

Season	*n*[a]	No. lead-exposed	2-week exposure rate (%)[b]
Fall	174	79	43.8[c]
Spring	183	83	21.6

[a] No. of paired blood samples tested for lead.
[b] Max. likelihood estimate (%) of unexposed bird becoming exposed to lead in a 2-week interval.
[c] The 2-week lead exposure rate in the fall was significantly higher than in the spring (2-sided $Z = 4.07$, $P < 0.001$).

Elevated blood lead levels (≥0.2 ppm) were found in sentinel birds in all 3 enclosures in the fall of 1986 (Table 2). The maximum likelihood estimate of the 2-week lead exposure rate was significantly greater ($Z = 2.67$, $P = 0.0038$) in P8 (43.8%) than in T19 (18.1%) and significantly lower in TF (0.9%) than both P8 ($Z = 11.7$, $P < 0.001$) and T19 ($Z = 1.85$, $P = 0.032$) (Table 2). In P8, the likelihood of lead exposure was significantly higher ($Z = 4.07$, $P < 0.001$) in the fall of 1986 (43.8%) than in the spring of 1987 (21.6%) (Table 3). No sex-specific differences were detected in the rates of lead exposure in P8 ($Z = 1.57$, $P = 0.11$) or in T19 ($Z = 0.81$, $P = 0.28$). Sex-specific rates of lead exposure were not calculated for TF because there were so few exposures.

The cumulative probability of lead poisoning mortality (Table 4) was significantly higher in sentinels in P8 than in TF in 1986, 1987, 1988, and 1989 ($P \leq 0.006$). Likewise, the cumulative probability of mortality from lead poisoning was

Table 4. Cumulative probability of lead poisoning mortality in captive-reared sentinel mallards held in enclosures in wetlands at the Sacramento National Wildlife Refuge in the fall months (Jul–Oct) of 1986–89. Values with different letters are significantly different ($P < 0.05$).

Year	P8[a]	T19[b]	TF
1986	0.25 A	0 B	0 B
1987	0.11 A	0.06 A	0 B
1988	0.23 A	0.12 AB	0.04 B
1989	0.16 A	0 B	0 B

[a] One-sided composite Z tests indicated that the probability of lead poisoning was significantly higher in P8 than T19 (composite $Z = 4.79$, $P < 0.001$) and TF (composite $Z = 6.34$, $P < 0.001$) over all years.
[b] One-sided composite Z test indicated that the probability of lead poisoning was significantly higher in T19 than TF (composite $Z = 3.72$, $P < 0.001$) over all years.

significantly higher in P8 than in T19 in 1986 and 1989 ($P < 0.003$), but not in 1987 ($Z = 0.84$, $P = 0.20$) or in 1988 ($Z = 1.49$, $P = 0.07$). The cumulative probability of lead poisoning mortality was also significantly higher in T19 than in TF in 1987 ($P = 0.014$), but not in 1986, 1988 ($Z = 1.53$, $P = 0.06$) or 1989. Analyzing trends over all 4 years of the study, rates of mortality were significantly higher in P8 than either T19 or TF (composite $Z = 4.79$ and 6.34 respectively, $P < 0.001$). In addition, overall rates of mortality were significantly higher in T19 than TF (composite $Z = 3.72$, $P < 0.001$). In P8 in 1986, lead poisoning mortality rates were significantly higher ($Z = 3.54$, $P < 0.001$) in the fall (0.25) than in the spring (0.04). No significant differences ($P > 0.05$) were detected in rates of lead poisoning mortality between males and females in any enclosure in any year, nor were any trends detected over all years (composite $Z = 0.086$, $P = 0.39$).

DISCUSSION

Our findings suggest that rates of lead exposure and lead poisoning mortality in captive-reared sentinels coincided with lead pellet density in sediments at SNWR. The highest rates of lead exposure and lead poisoning mortality occurred in P8, the enclosure with the highest lead pellet density in the sediments (>2,000,000 pellets/ha), followed by T19, the enclosure with the second highest lead shot density (173,200 pellets/ha). Accordingly, the pool (TF) with the lowest density of lead pellets (15,750 pellets/ha) had the lowest lead exposure and lowest mortality from lead poisoning.

In previous studies of lead pellet deposition and accumulation in wetland sediments, a wide range of densities, up to 2,245,230 pellets per ha (Galveston Bay, Tex.; Fisher et al. 1986), have been found (Zwank et al. 1985, Anderson and Havera 1989, Roscoe et al. 1989, DeStefano et al. 1991, Guitart et al. 1994). However, sampling design, sample depth, and total area represented were not consistent among studies, making it difficult to compare pellet densities and availability between wetlands. Also, because the site of pellet ingestion could not be determined in free-flying birds, the risk of lead exposure and lead poisoning in waterfowl in relation to lead pellet density was not measured in these studies. Besides the availability of lead pellets, additional factors influence the probability of pellet ingestion and subsequent lead poisoning, including: species, sex, and age of bird; food habits and specific feeding behavior; water depth; availability of alternative grit or seed sources; wetland or soil substrate characteristics; and weather conditions. In our study with captive-reared sentinel mallards confined to enclosures, these variables were held constant among wetlands as much as possible, enabling comparison in the rates of lead exposure and lead poisoning between wetlands with different lead pellet densities, between seasons, and between males and females.

The higher rates of lead exposure and lead poisoning mortality detected before the hunting season in our study are counter to field observations of major lead poisoning outbreaks, which are most evident in the winter and spring, after hunting seasons have ended (Bellrose 1959, Sanderson and Bellrose 1986, NWHC unpubl. data). However, Sanderson and Bellrose (1986) point out that massive lead poisoning die-offs represent a small proportion of the total mortality from lead poisoning, most of which occurs as "...obscure, overlooked, day-to-day losses...." Our data, from a known and controlled population at risk with site-specific exposure to a known density of lead pellets, suggest that the risk of ingesting pellets and dying from lead poisoning is greater in the summer and fall months (Jul-Oct) than in winter and spring months (Feb-May) for mallards in the Sacramento Valley. This disparity may not occur in other locations or flyways, although Zwank et al. (1985) also reported that lead ingestion rates in mallards and pintails were greater before (Oct) than during the hunting season (mid-Nov through Jan) on 2 areas in Louisiana.

The seasonal differences in lead exposure and lead poisoning mortality rates we observed may be due to changes in diet. In the Sacramento Valley, northern pintails (*Anas acuta*) shift to foods higher in protein in the spring, particularly invertebrates (Miller 1987). A similar dietary shift in sentinel mallards may have decreased the ingestion of pellets mistaken as grain or grit. Previous studies (Bellrose 1959) indicated that ingested pellets remained in the gizzard an average of 20 days before they were either completely eroded, dissolved, or were voided. Using this figure, we estimated crude daily ingestion rates (no. of pellets ingested/bird/day) by dividing the total number of pellets by the number of exposure-days (20 × no. of birds sampled). In P8, during the summer-fall, we

estimated an ingestion rate of 7.8 pellets/100 bird-days, and during the spring, 1.0 pellet/100 bird-days. In addition, foods high in calcium and protein, such as invertebrates, have been shown to ameliorate the effects of lead exposure (Koranda et al. 1979), possibly resulting in lower spring mortality. If similar seasonal differences in lead ingestion and exposure occur in free-flying waterfowl, lead poisoning may have a greater effect on summer resident waterfowl that breed and moult on hunted wetlands in the Sacramento Valley than on winter migrating populations.

MANAGEMENT IMPLICATIONS

The pellet density of >2,000,000 shot/ha detected in P8 at SNWR is among the highest reported in the literature, representing years of lead pellet accumulation. Evidently, heavy clay soils in this wetland prevent lead pellets from settling into the sediments below the level of availability for mallards. Likewise, in T19, a wetland that is closed to hunting, the pellet density of >173,000 shot/ha represents years of accumulation and persistence of shot from adjacent hunted areas. The last year in which hunting was allowed adjacent to T19 was 1973. The relation between high lead pellet density and elevated rates of lead exposure in sentinel mallards demonstrates that lead poisoning can remain a significant problem in some wetlands for years after the conversion to non-toxic shot. Corrective action may be needed to reduce pellet density or availability in SNWR wetlands and others with similar clay bottoms.

Habitat management recommendations to reduce lead pellet density and availability include cultivation (Fredrickson et al. 1977, Esslinger 1979), the addition of gravel or crushed oyster shells as supplemental grit (Wills and Glasgow 1964, Beer and Stanley 1965), water-level management (Wills and Glasgow 1964, Bishop 1973), and vegetation manipulation (Jordan and Bellrose 1950, 1951). Tillage has been shown to alter the distribution of lead pellets in the soil profile (Fredrickson et al. 1977, Esslinger 1979), but the effect on subsequent lead poisoning in waterfowl has not been evaluated adequately. Removal of lead pellets from soils through reclamation procedures, such as gravity separation, has been suggested but does not appear to be a practical solution for large areas with current technologies. We recommend further studies at SNWR to apply and evaluate both current and new techniques to reduce lead pellet density and availability.

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