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Authors: Bollinger, Trent K., Mineau, Pierre, and Wickstrom, Mark L.

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TOXICITY OF SODIUM CHLORIDE TO HOUSE SPARROWS (*PASSER DOMESTICUS*)

Trent K. Bollinger,[1,4] Pierre Mineau,[2] and Mark L. Wickstrom[3]

[1] Canadian Cooperative Wildlife Health Centre, Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatoon, Saskatoon, Saskatchewan, Canada S7N 5B4
[2] National Wildlife Research Centre, Canadian Wildlife Service, Carleton University Campus, Ottawa, Ontario, Canada K1A 0H3
[3] Toxicology Centre, University of Saskatchewan, Saskatoon, Saskatchewan, Canada S7N 5B3
[4] Corresponding author (email: trent.bollinger@usask.ca)

ABSTRACT: Sodium chloride (NaCl) is widely used as a deicing agent on roadways. There are numerous anecdotal reports of poisoning of passerine birds by road salt in the United States and Canada, but little is known about the toxicity of NaCl to songbirds. The objectives of this study were to determine the lethal dose range for NaCl in a representative passerine species (house sparrow [*Passer domesticus*]); to determine the clinical, physiologic, and pathologic effects of sublethal and lethal oral NaCl exposure; and to assess the potential for recovery after exposure to granular salt or highly concentrated salt solutions. The up-and-down method was used in a pilot study to estimate the lethal oral dose of granular NaCl in wild caught house sparrows. The toxicity of highly concentrated NaCl solution also was investigated. This was followed by an acute dose response study in which house sparrows were dosed orally with granular NaCl at 0, 500, 1,500, 2,500, or 3,500 mg/kg. Sparrows were deprived of water for 6 hr postexposure (PE) in an attempt to mimic specific winter conditions. Groups of three birds at each dose were euthanized at 1, 3, 6, and 12 hr PE, and samples were collected for histopathology and brain and plasma electrolyte analyses. Results indicated an approximate mean lethal dose (LD_{50}) of 3,000–3,500 mg/kg in water-deprived birds, which is similar to mammalian values. House sparrows dosed with a concentrated solution of NaCl generally died at doses of 8,000 mg/kg. Clinical signs observed at ≥1,500 mg/kg included rapid onset (<30 min) of depression (indicated by reduced activity and reduced response to visual and auditory stimuli), ataxia, inability to fly or perch, and death in as little as 45 min. Birds that survived for 6 hr usually recovered. Plasma Na concentrations >200 mmol/l were consistently associated with clinical signs. Pathologic lesions consisted of edema and distension of the caudoventral thin muscled region of the gizzard and were observed 1 hr PE in most birds dosed with ≥500 mg/kg. Brain Na concentrations in clinically ill sparrows and those that died of NaCl toxicity ranged from 1,297 to 1,615 (mean=1,450; SD=115) ppm wet weight or 5,603 to 6,958 (mean=6,367; SD=454) ppm dry weight, which differed significantly from control birds. No histologic lesions were observed in brain sections of exposed birds, likely reflecting the acute nature of the exposure. However, fluid accumulation beneath the koilin layer of the gizzard was observed in the majority of birds at high dosage levels. These results indicate that passerines ingesting relatively small numbers of road salt granules or small quantities of highly concentrated NaCl solutions are at risk of sodium poisoning.

Key words: Brain sodium, house sparrows, *Passer domesticus,* plasma sodium, road salt, salt poisoning, sodium chloride, sodium toxicity.

INTRODUCTION

The environmental impact of chemical deicers has been a subject of debate and scientific study for nearly 50 yr (D'Itri, 1992). Sodium chloride (NaCl) is the most commonly used deicer on roadways in North America, with approximately 4–5 and 15 million tons used each year in Canada and United States, respectively (Salt Institute, 2003). Wildlife mortality along roadsides where salt has been applied has been documented (Trainer and Karstad, 1960; Clark, 1981; Canadian Cooperative Wildlife Health Centre, unpubl. data). Such mortality often goes unreported or is attributed to vehicle strikes, with salt acting as a "fatal attractant" (Baker, 1965; Clark, 1981). Mortality among songbirds is reported most commonly in cardueline finches (crossbills, grosbeaks, and siskins) (Tozer, 1994). These birds appear to be attracted to salted roads, possibly in response to a dietary sodium deficiency (Fraser, 1985). Repeated observations of abnormal behavior in exposed birds along

roadsides (Trainer and Karstad, 1960; Baker, 1965) suggest that salt toxicity per se may be contributing to the vulnerability of songbirds to road traffic and may be a direct cause of mortality in some birds.

The objectives of this study were to determine the lethal dose range for NaCl in a representative passerine species (house sparrow [*Passer domesticus*]); to determine the clinical, physiologic, and pathologic effects of sublethal and lethal oral NaCl exposure; and to assess the potential for recovery after exposure to granular salt or highly concentrated salt solutions.

MATERIALS AND METHODS

Animals

Wild house sparrows were captured September through November using mist nets and live traps. Birds (males and females; predominately juveniles; mean body weight=28.1±0.24 g) were randomly assigned to treatment groups and housed in wire mesh cages under ambient light conditions, with ad libitum access to fresh water and a wild bird seed mix. They were acclimatized to captivity for at least 1 wk prior to testing.

Lethal dose estimation

Initially, four house sparrows were manually restrained and dosed orally with NaCl granules (2–3 mm diameter) at 3,000 mg/kg. Two of the birds had free access to water, while the other two birds had water withheld for 6 hr postexposure (PE) to simulate potential winter conditions. Birds were monitored for clinical signs of toxicosis, onset of signs, and time to death. Results of the initial exposure were used to select the subsequent dose, using the up-and-down method to estimate the lethal dose (Bruce, 1985). Dosages used were 2,000, 2,500, 3,000, 3,500, 4,000, 4,500, and 5,500 mg/kg. Water was withheld for 6 hr PE in all cases, and two house sparrows were exposed at each dosage level except at 4,500 and 5,500 mg/kg, when three sparrows were used. Birds were monitored continuously for the first hour, every half hour for the next 7 hr, and then at least every 10 hr until 72 hr PE. Birds that died were immediately necropsied. Sparrows still alive 72 hr PE were euthanized with halothane and then necropsied. The mean lethal dose (LD_{50}) was calculated using the trimmed Spearman-Karber estimate.

In another exposure trial, house sparrows were exposed to NaCl dissolved in MilliQ-filtered water. Birds were manually restrained and given an oral dose of NaCl solution using a stainless steel gavage tube inserted into the crop. Birds were randomly assigned to treatment groups consisting of four birds each. Birds in the three treatment groups received 4,000, 6,000, or 8,000 mg/kg NaCl respectively. Half of the birds in each treatment group were allowed free access to water, whereas the other two birds in each treatment group were water deprived for 12 hr PE and then given free access to water. All birds were monitored as above.

Dose response study

House sparrows were dosed orally with NaCl granules (2–3 mm diameter) at 0, 500, 1,500, 2,500, or 3,500 mg/kg. Water was withheld for 6 hr PE, and birds were monitored every half hour for clinical signs of toxicosis. Groups of three birds at each dose were anaesthetized with halothane at 1, 3, 6, and 12 hr PE, and intracardiac blood samples were collected for plasma electrolyte analysis. If birds demonstrated severe intoxication and were unlikely to survive to their predefined sampling time, they were bled and euthanized. Plasma sodium, potassium, and chloride concentrations were determined directly using ion selective electrodes and an Abbott Spectrum II analyzer (Chicago, Illinois, USA). Birds were then euthanized with an overdose of halothane and immediately subjected to complete postmortem examination. The brain was removed and halved along the median plane. One half of the brain was placed in 10% neutral buffered formalin and processed routinely for histopathologic examination using hematoxylin and eosin stain. Four transverse sections of the brain were examined histologically at the level of the medulla oblongata, cerebellum, optic lobe, and rostral and caudal cerebral cortex. The remaining half of the brain was weighed, dried with a Savant Speed Vac, and then reweighed to derive a dry weight and wet weight/dry weight ratio. Sodium concentration of the dried brain sample was determined by nitric acid and microwave digestion with concentrations determined by inductively coupled plasma (ICP) emission spectrophotometry using standard techniques (Jarrell Ash Corporation, 1995; CEM Corporation, 1995). Bovine liver with a Na concentration of 2,420±60 ppm, as determined by flame atomic emission spectrometry (National Institute of Standards and Technology, Gaithersburg, Maryland, USA; standard reference material 1577b), was analyzed with each batch of brain samples. Wet weight Na concentra-

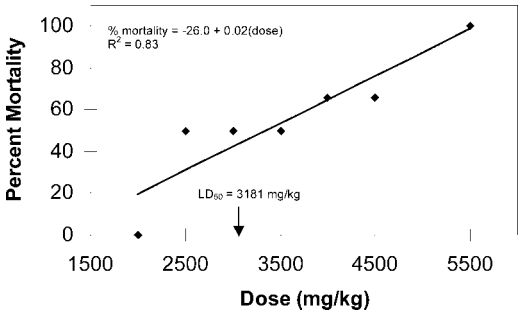

FIGURE 1. Effect of oral dose of NaCl on mortality of house sparrows.

tions were calculated using the wet weight/dry weight ratio derived for each sample.

RESULTS

Lethal dose estimation

The LD_{50} for house sparrows orally exposed to granular NaCl and deprived of water for 6 hr PE was 3,181 mg/kg (95% confidence interval = 2,504, 4,040) (Fig. 1). No clinical signs were observed at 2,000 mg/kg, but one member of the pair exposed to 2,500 mg/kg was depressed and reluctant to fly 45 min PE; this bird died at approximately 5 hr PE. All sparrows exposed to 3,000 mg/kg NaCl, including those with free access to water, were mildly depressed 20 min PE. The two birds with access to water appeared normal by 1 hr PE, whereas both water-deprived sparrows became increasingly depressed, developed ataxia, and eventually were unable to fly; one of these birds died, and one recovered 1–2 hr after being given access to water at 6 hr PE. Similarly, one of two birds died at dosages of 3,500 and 4,000 mg/kg. At the 4,500 mg/kg dose, two of three sparrows died and all birds showed neurologic signs at 30 min PE; one of these birds died at 45 min PE, the other at 12 hr PE. At the 5,000 mg/kg dose, all three birds died at 1.5, 4, and 5 hr PE, respectively.

Sparrows given concentrated NaCl solutions were able to tolerate higher NaCl exposures (Tables 1 and 2) than birds given granular NaCl. Sparrows survived exposures of up to 6,000 mg/kg even when water deprived for 12 hr. At the 8,000 mg/kg dose, more than half of birds died regardless of whether they had access to water. The survivors at this dose all had received a 4,000 mg/kg NaCl solution 5 days previously with no effect.

Dose response study

Plasma Na concentrations at 1, 3, 6, and 12 hr PE are plotted in Figure 2. The mean plasma Na concentration of the 12 control birds was 163.2±3.01 mmol/l, and the upper 95% confidence limit was 165.1 mmol/l. Although only three control birds were sampled at each time point, there was no obvious increase in plasma Na levels in control birds deprived of water for up to 6 hr. Plasma Na concentrations of all dosed sparrows were above the upper 95% confidence limit for control birds at 1, 3, and 6 hr PE, but concentrations returned to normal by 12 hr PE after birds had ac-

TABLE 1. Effect of oral doses of concentrated NaCl solutions on house sparrows when deprived of water for 12 hr after exposure.

Bird ID	Dosage (mg/kg)	Dose volume (ml)	Conc. of solution (mg/ml)	Time to first clinical signs (min)	Time to improvement (hr)	Time to death (hr)
1W−	4,000	0.56	200	None	—	Survived
2W−	4,000	0.52	200	None	—	Survived
3W−	6,000	0.54	325	30	5	Survived
4W−	6,000	0.54	325	30	5	Survived
2W−	8,000	0.65	325	30	None	4
1W−	8,000	0.72	325	60	8	36
6W−[a]	8,000	0.65	325	30	12	Survived

[a] Dosed 5 days previously with 4,000 mg/kg with no adverse effects.

TABLE 2. Effect of oral doses of concentrated NaCl solutions on house sparrows given free access to water.

Bird ID	Dosage (mg/kg)	Dose volume (ml)	Conc. of solution (mg/ml)	Time to first clinical signs (min)	Time to improvement (hr)	Time to death (min)
1W+	4,000	0.52	200	None	—	Survived
2W+	4,000	0.58	200	None	—	Survived
3W+	6,000	0.50	325	30	5	Survived
4W+	6,000	0.54	325	30	5	Survived
1–29	8,000	0.61	325	20	None	45
2–29	8,000	0.61	325	20	None	45
5–20[a]	8,000	0.72	325	None	—	Survived
6–20[a]	8,000	0.56	325	None	—	Survived

[a] Dosed 5 days previously with 4,000 mg/kg with no adverse effects.

cess to water for 6 hr. At 1 hr PE, plasma Na concentrations increased linearly with dose with the following relationship: plasma Na=168.1+0.009 (dose); $P<0.001$, $R^2=0.74$. By 3 and 6 hr PE, the linear regression explained less of the variability in plasma Na ($P=0.048$, $R^2=0.29$ and $P=0.02$, $R^2=0.44$, respectively).

Brain Na concentrations at 1, 3, 6, and 12 hr PE are plotted in Figure 2. The mean brain Na concentration of the 12 control birds was 5,076±410 ppm dry weight, and the upper 95% confidence limit was 5,308 ppm dry weight. All sparrows dosed with 1,500 mg/kg or greater demonstrated elevated brain Na concentrations at 1, 3, and 6 hr PE. At 12 hr PE, after 6 hr of access to water, brain Na concentrations had returned to normal in most sparrows; however, two of six sparrows still had levels above the 95% confidence limit of control birds. The mean brain wet weight to dry weight ratio was 4.66±0.05. Mean Na concentrations of liver standards run simultaneously with each batch of brain samples was 2,218±86 ppm, resulting in a recovery percentage of 92%.

Clinical signs were observed in two of 12 sparrows dosed with 1,500 mg/kg; one died between 3 to 4 hr PE, and the other was moribund at 3 hr PE when it was euthanized. No adverse effects were observed in the 12 birds given 500 mg/kg. Signs of depression and reduced responsiveness to external stimuli occurred within 22 to 30 min in some birds, whereas in others, clinical signs did not appear for several hours. Signs progressed to include ataxia, characterized by inability to fly or perch, and occasional head tremors. Three sparrows from the 3,500 mg/kg treatment group were euthanized prior to their predefined sampling time because they showed severe clinical signs. Their plasma sodium concentrations were 215, 214, and 220 mmol/l. One sparrow with a plasma Na concentration of 200 mmol/l was clinically normal at 1 hr PE, whereas the plasma Na concentrations of three sparrows showing clinical signs at 1 hr PE were 186, 198, and 211 mmol/l. Plasma Na concentrations of >200 mmol/l were associated with severe clinical signs. The mean brain Na concentrations of birds showing clinical signs at the time of planned euthanasia ($n=6$), those that were euthanized due to severe clinical disease ($n=3$), and those that died PE ($n=2$) was 6,367±454 ppm dry weight, which was significantly different than mean brain Na concentrations of control birds ($P<0.0001$).

Gross postmortem examination revealed fluid accumulation under the koilin layer and distension of the caudoventral, thinly muscled region of the gizzard. This change was observed at 1 hr PE in two of three birds in each of the groups given 500 mg/kg and 1,500 mg/kg, and in all three birds in each of the groups given 2,500 and 3,500 mg/kg NaCl. The proportion of birds with gizzard edema decreased with time postexposure but even at 12 hr PE one in

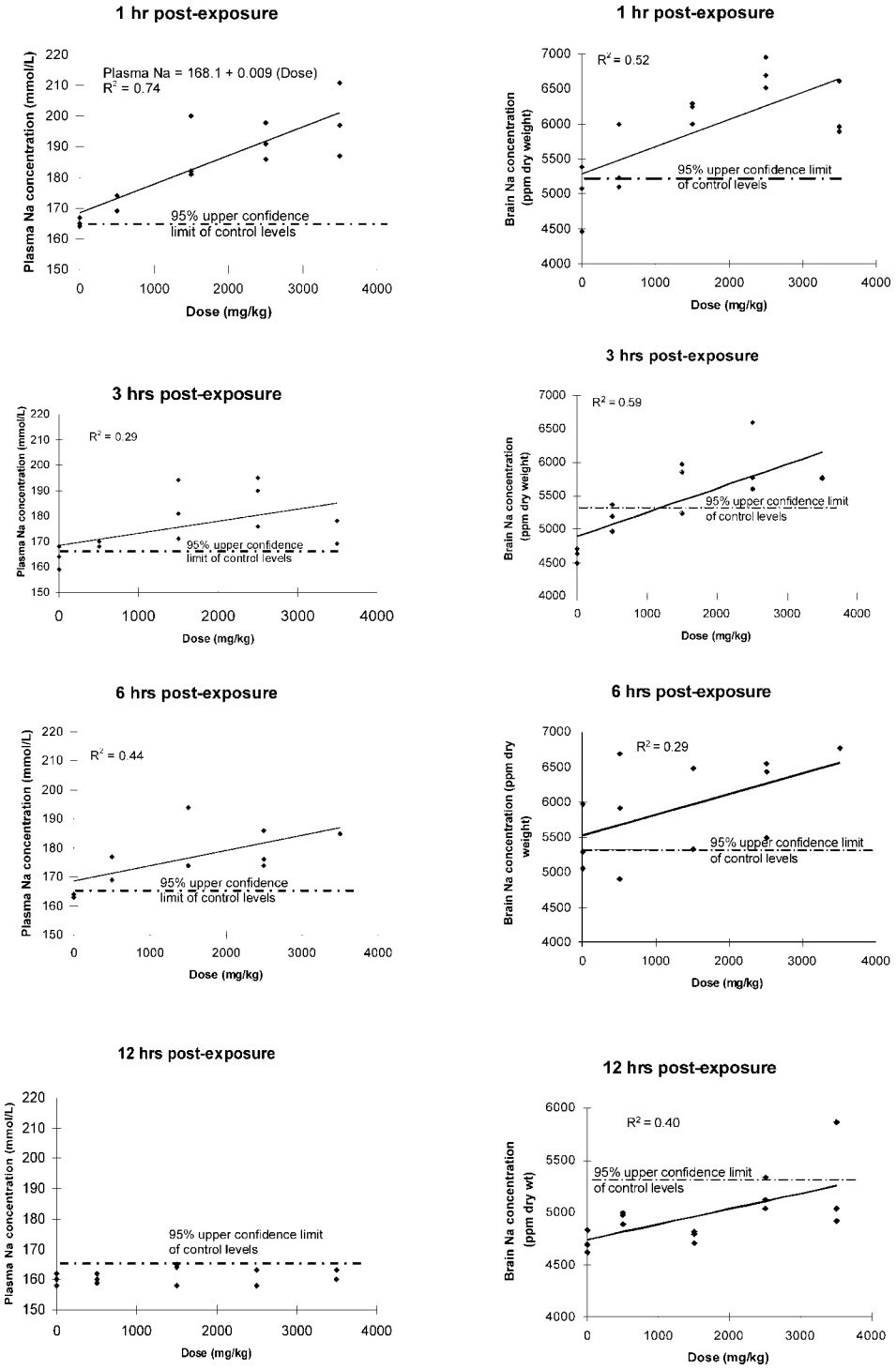

FIGURE 2. Effect of oral dose of NaCl on plasma and brain Na concentrations.

three birds in each of the groups given 1,500, 2,500, and 3,500 mg/kg NaCl still were affected. There were no histologic lesions in the brains of sparrows that died acutely of salt toxicity or those that were euthanized up to 12 hr PE.

DISCUSSION

Published oral LD_{50} values for NaCl are not available for any wild species. Reported oral LD_{50} for rats and mice are 3,000 mg/kg and 4,000 mg/kg, respectively (Bertram, 1997). Approximate lethal doses reported for pigs, horses and cattle are 2.2 g/kg body weight, 6 g/kg for sheep (Buck et al., 1976) and 4 g/kg for chickens (Austic and Scott, 1984). Although lethal dose is dependant on availability of water, these levels are similar to the LD_{50} of 3,181 mg/kg observed in this study for granular NaCl. Sparrows given a concentrated solution containing NaCl and sparrows with free access to water had a higher LD_{50}.

Exposure to elevated levels of NaCl in the recent past also appeared to increase the LD_{50}. In our study, three sparrows dosed with 4,000 mg/kg NaCl in solution 5 days previously survived subsequent oral exposure of 8,000 mg/kg in solution, whereas four other sparrows that had not been previously dosed with NaCl died when exposed to the same 8,000 mg/kg dose. In birds with salt-secreting nasal glands, prior exposure to NaCl induces hypertrophy of the gland and increased volume of secreted fluid, allowing them to more readily respond to NaCl exposure (Schmidt-Nielsen and Kim, 1964). Although passeriform species, such as house sparrows, do not have a salt-secreting nasal gland (King and McLelland, 1984), our results suggest sparrows also are able to physiologically adapt to elevated salt intake. Recent migrants to areas containing high levels of salt, such as heavily salted roads, are likely more prone to toxicity (Windingstad et al., 1987).

The dose of granular NaCl required to elevate plasma sodium concentrations above the normal, homeostatic range is quite low (<500 mg/kg). House sparrows are known to ingest grit particles as large as 2.4-mm diameter (Gionfriddo and Best, 1995); based on our results, less than one road salt granule of this size would raise the plasma sodium concentration above the normal range for a house sparrow with an average weight of 28 g. Similarly, doses of 1,500 mg/kg consistently caused elevation of brain Na concentrations. During the dose response study, two of 12 sparrows showed clinical signs at a dose of 1,500 mg/kg, and one of these two died. There were no clinical signs in the 500 mg/kg dose group; thus, the no observed effect level falls somewhere between 500 and 1,500 mg/kg. Ingestion of a small number of road salt granules by a songbird would be sufficient to deliver a dose exceeding this level.

Diagnosis of Na toxicity in sick birds is based on demonstrating elevated plasma sodium concentrations. The 95% upper confidence limit for plasma Na in control birds was 165.1 mmol/l, and in all cases of NaCl exposure with restricted access to water, plasma Na levels exceeded this value. Severe clinical signs were observed in birds with plasma sodium levels of >200 mmol/l, but clinical signs also were observed in birds with plasma Na levels as low as 186 and 198 mmol/l. Dawson et al. (1965) reported that plasma Na concentrations of approximately 210 mEq/l (mEq/l = mmol/l for Na) were lethal in red crossbills (*Loxia curvirostra*).

House sparrows that were sick or died from Na toxicity had significantly elevated brain Na concentrations compared with control birds. The mean brain Na concentration in these clinically ill or dead birds was 6,367±454 ppm dry weight or 1,450±115 ppm wet weight (range=1,297–1,615). This is considerably lower than levels reported in other species suffering from salt toxicity (Table 3). This discrepancy may be the result of interspecies differences in normal and toxic brain sodium concentrations or interlaboratory differences in measured tissue sodium concentrations.

TABLE 3. Brain sodium concentrations of house sparrows compared with concentrations in other species reported in the literature.

Species	Control levels (ppm wet weight)	Toxic levels (ppm wet weight)	Reference
Ruddy ducks (*Oxyura jamaicensis*)			Gordus et al. (2002)
Mean	1,090[a]	2,580	
n	5	7	
SE	1.03	1.08	
Range	988–1,150	1,890–3,670	
Mallards (*Anas platyrhynchos*)			Meteyer et al. (1997)
Mean	1,305 and 1,403[b]	>2,000	
n	5 and 5	>10	
Canada geese (*Branta canadensis*)			Windingstad et al. (1987)
Mean	1,653[c]	1,993	
n	4	4	
Range	1,600–1,710	1,900–2,100	
Sandhill cranes (*Grus canadensis*))			Franson et al. (1981)
Mean	1,267[d]	2,104	
n	6	1	
SD	37	37	
House sparrows (*Passer domesticus*)			Present study
Mean	1,079	1,450	
n	12	11	
SD	113	115	
Range	910–1,313	1,297–1,615	

[a] Ducks shot approximately 104 km from wetland with salt poisoning.
[b] Experimental birds given free access to fresh drinking water and housed on land adjacent to hypersaline playa lakes.
[c] Hunter shot geese, origin unknown.
[d] Sandhill cranes given free access to fresh water for 14 days after previously being exposed to toxic levels of saline water.

Histologic lesions were not observed in brain sections of sparrows acutely poisoned with NaCl in this study; therefore, the diagnosis is dependant on demonstrating elevated concentrations of Na in brain and/or plasma. Gizzard edema was observed at necropsy in birds exposed to high concentrations of NaCl in their diet but disappears with time PE. Fluid accumulation under the koilin layer of the gizzard also was reported by Trainer and Karstad (1960) in pheasants experimentally exposed to high concentrations of NaCl in their diet.

Songbirds may ingest road salt granules in response to a dietary sodium deficit or incidentally while foraging for grit on roadways. Although we did not test whether road salt was ingested as grit by house sparrows, research has shown that house sparrows chose grit with characteristics similar to that of road salt. House sparrows and northern bobwhite (*Colinus virginianus*) prefer angular/oblong grit over rounded/spherical (Best and Gionfriddo, 1994) and yellow or white colored grit over black and blue colors (Gionfriddo and Best, 1996). Gionfriddo and Best (1995) reported grit size in free-ranging house sparrows ranged from 0.1 mm to 2.4 mm (mean=0.5±0.1 [SD] mm) and the number of grit particles in the gizzard ranged from 0 to 3,204 (mean=580.3); grit size and counts varied depending on diet. Ingestion of granular formulated pesticides as grit is a documented cause of mortality in birds (Augspurger et al., 1996; Stafford and Best, 1999). Results of this study indicate that wintertime mortality may be a direct result of sodium toxicity, since only five 2.4-mm salt granules would represent a LD_{50} dose to an average size house spar-

row. In addition, NaCl induced neurobehavioral effects, such as ataxia and decreased response time as a result of elevated plasma and brain sodium levels, may increase the vulnerability of songbirds to vehicle strikes along salted roads. Sparrows exposed to sublethal levels of NaCl that went on to recover continued to show clinical signs anywhere from 5 to 8 hr PE.

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