Journal of Wildlife Diseases, 25(1), 1989, pp. 132–135

Acute Arsenic Toxication of a Free-ranging White-tailed Deer in New York

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ABSTRACT: An adult, female white-tailed deer (*Odocoileus virginianus*) died due to acute arsenic intoxication in an intensively managed northern hardwood forest in northern New York. We hypothesize that the deer licked trees injected with Silvisar 550, which contains monosodium methanearsonate. We believe this is the first report of death of a free-ranging white-tailed deer, due to ingestion of monosodium methanearsonate.

Key words: White-tailed deer, *Odocoileus virginianus*, monosodium methanearsonate, Silvisar 550, acute toxic poisoning, case report.

The toxic effects of herbicides containing forms of inorganic arsenic to wildlife in the United States have been recognized since the early 1950's. Unusual wildlife mortality in a number of species, including white-tailed deer (*Odocoileus virginianus*), occurred in the upper peninsula of Michigan in the early summer of 1952 where a lumber company was testing sodium arsenite as a chemical debarking agent. This prompted Boyce and Verme (1954) to determine attractiveness and toxicity of sodium arsenite to white-tailed deer. They concluded that deer are attracted to solutions of sodium arsenite due to its salty taste and that small amounts of it are extremely toxic to deer. Webb et al. (1956) conducted a study in the central Adirondack Mountains in New York to test the effects of debarking agents on forest wildlife. Although during their study wildlife were not adversely impacted, they concluded that treated wood posed a threat to wildlife, if ingested, 4 to 7 days post-treatment.

In 1979 the U.S. Environmental Protection Agency (EPA) proscribed the use of a widely used herbicide, 2,4,5-T, on forested lands in the United States. At that time, the EPA designated monosodium methanearsonate (MSMA) as a substitute chemical (Midwest Research Institute, 1975). MSMA is an organic arsenical used in some herbicides and silvicides, and is a sodium salt of methanearsenic acid (Naqvi et al., 1985). It is widely used in the southern United States as a selective postemergent herbicide on cotton (Judd, 1979) and along roadsides for weed control (Naqvi et al., 1985). Silvicides containing MSMA have been used in intensively managed hardwood or coniferous forests in the northern United States. Inorganic arsenicals such as sodium arsenite, although extremely effective, were abandoned during the late 1960's because of their toxicity, reactivity and instability.

A number of studies have tested the toxicity of MSMA to various animals. Most authors primarily examined domestic animals (Dickinson, 1972; Exon et al., 1974; Abdelghani et al., 1976; Judd, 1979; Shariatpanahi and Anderson, 1984; Naqvi et al., 1985; Prukop and Savage, 1986). Hence, little is known about the effects of MSMA on free-ranging wildlife. We report on the death of a free-ranging white-tailed deer on Huntington Wildlife Forest, in the central Adirondack Mountains of New York (USA; 43°57′ to 44°04′N, 74°10′ to 74°18′W), due to the apparent ingestion of lethal levels of MSMA in Silvisar 550 (TSI Company, Flanders, New Jersey 07836, USA).

Dickinson (1972) reported the oral lethal dose (100% mortality) for cattle is 80 to 100 mg MSMA/kg body weight. Four of five animals showed symptoms of arsenic poisoning after doses of 50 to 70 mg MSMA/kg body weight had been given. Two steers succumbed after consuming a total of 100 mg MSMA/kg body weight in 10 mg/kg increments over a 10 day period. Liver and kidney tissue of one animal succumbing on day 10 contained 24 and 64 ppm arsenic residue, respectively. Liver and kidney for a second animal (near death

on day 10, euthanized on day 12) contained 30 and 23 ppm arsenic residue, respectively. Dickinson (1972) suggested that the second animal reduced the arsenic content in the kidney after the treatments ended, thus accounting for the lower kidney concentration.

The availability of organic arsenicals from vegetation and soils to wildlife has been studied in a forested ecosystem. Norris et al. (1983) found that in a coniferous forest treated by stem injection with MSMA, arsenic residues in the litter were significantly higher for 2 yr posttreatment close to treated trees. Arsenic levels in the litter decreased with distance from a treated tree, and with time. They observed no significant increases in arsenic residue in the soils, however. Thus, they concluded that MSMA does not pose a hazard to groundwater or soils through long-term accumulation.

Silvisar 550 has been used on the Huntington Wildlife Forest for the past 6 yr to control understory beech (*Fagus grandifolia*) and striped maple (*Acer pensylvanicum*), 2.5 to 15 cm diameter breast height (dbh), for timber stand improvement. The total active ingredient (MSMA) is about 48% and total elemental arsenic, in water soluble form, is 22%. The solution contains 0.72 kg MSMA/liter (Vineland Chemical Company, 1611 West Wheat Road, Vineland, New Jersey 08360, USA). The standard application is 1 to 2 ml of solution per 5 cm of dbh, injected into spaced axe cuts made in the tree bark 1 m above the ground.

In preparation for winter logging activities on Huntington Wildlife Forest, a total of 3.6 liter of Silvisar 550 was applied to a 1.2 ha area on 19 and 21 May 1987. On 23 May an adult female white-tailed deer was found dead, partially emersed in a large puddle of water, on a grassy logging road. No evidence of a spill was found at the storage facility. Fecal matter suggestive of diarrhea was found among the treated trees.

The deer weighed 67.5 kg at necropsy and carried two near term fetuses. No broken bones were found, but scrapes of the integument suggested severe trauma occurred several weeks earlier. These appeared unrelated to internal pathology. The thorax and peritoneal cavities contained bloody fluid and plasma clots while the lungs were thoroughly congested and hemorrhagic. The alimentary canal contained primarily bloody fluid with little vegetation present. The rumen serosa had multiple ecchymoses, while the small intestine was inflamed and no feces were present in the large intestine. Since the rumen was full of water and the feces liquid, it appeared that the animal had consumed large quantities of water and had severe diarrhea. This was further evidenced by a protruded rectum. A slight hemorrhage appeared at the juncture of the cortex and medulla of the kidney. Gross pathological changes were compatible with those described for arsenic toxicity in cattle (Dickinson, 1972; Thatcher et al. 1985).

Standard bacterial and parasitological tests using both selective and enrichment media were conducted by the New York State Health Department (Albany, New York 12201, USA). Tests failed to identify a causative agent responsible for the gross pathological changes killing the deer. Neither *Salmonella* spp. nor *Yersinia* spp. were found in the feces, nor were there any protozoans or helminths. A Giemsa-stained blood smear had no blood parasites and the red cells were normocytic. Toxicological examination, however, indicated acute levels of arsenic in liver and kidney. These tissues were examined using inductively coupled argonne plasma spectrophotometry by Hazelton Laboratories (Madison, Wisconsin 53704, USA). The liver and kidney contained 102 ppm and 56 ppm of arsenic on a wet basis, respectively. Arsenic concentrations were 2 to 3 times higher than the lethal dose for cattle reported by Dickinson (1972).

To estimate the amount of MSMA consumed by the deer, we assumed that the assimilation rate was similar to that of cat-

tle. Because the arsenic concentration was lower in the liver than the kidney, we compared the levels of arsenic in liver and kidney tissue from the deer to those of the second steer reported by Dickinson (1972). Converting units of measure, we determined that Silvisar 550 solution contains 719 mg MSMA/ml solution. Multiplying this ratio by the ratio of the molecular weights of As/MSMA (74.9 g As/162 g MSMA) we calculated that the solution contains 332.4 mg As/ml solution. We determined the lethal dose of As/kg body weight for deer by using the concentration of As found in liver and kidney of the steer (30.3 ppm and 23.2 ppm, respectively), the known lethal dose for cattle (100 mg As/kg body weight) (Dickinson, 1972), and the concentration of As found in liver and kidney of the deer (102 ppm and 56 ppm, respectively). We estimated that the lethal dose was 241.4–336.6 mg arsenic/kg body weight. Based on the deer's weight (67.5 kg), we calculated that it consumed a minimum of 16,295–22,721 mg arsenic, or 49.0–68.3 ml of Silvisar 550 solution. We believe that these estimates are conservative and ultimately depend on the assimilation rate of the chemical. If the assimilation rate of arsenic is higher in deer than in cattle, then the deer would have consumed less solution; if assimilation rates are lower, then it would have had to consume more.

MSMA has a salty taste and could be a strong attractant to deer. Ruminants are attracted to mineral licks and consume sodium rich soils during the late spring (Weeks, 1978). Weeks (1978) suggests that white-tailed deer develop a sodium deficiency due to high levels of potassium in spring forage. Elevated levels of potassium decrease the efficiency of sodium resorption in the kidney. Past studies on the Huntington Wildlife Forest found that rock salt, mixed with soil, effectively lures deer into box traps during the spring and summer (Mattfeld et al., 1972). The only direct observation of deer licking trees was reported by Boyce and Verme (1954). They observed 12 deer lick sodium arsenite treated trees up to 208 times within the first 96 hr after application. The highest number recorded for a single deer was 75 licks.

The exact source of the chemical ingested by the deer we examined remains unknown. Previous reports suggest that the deer could have ingested the chemical directly by licking the silvicide residue from the bark of the tree, or by consuming contaminated soil. Our estimates of the total amount of MSMA ingested suggested that the deer would have licked a minimum of 25 to 34 cuts, removing all solution applied (1 to 2 ml). Despite rapid uptake of the solution during application, some solution leaks out of the cuts and runs down the bark. Consequently it is not unreasonable to surmise that this was a source of the chemical. Alternatively, solution could have been spilled and, if it penetrated the litter layer, the deer could have consumed it with soil. The supply of silvicide used to treat the area was stored in a leak-proof container, encased in a locked wooden storage box, located approximately 7 m away from the carcass. We examined the storage facility closely and found no evidence of litter or soil disturbance to suggest that this was a source. Further, applicators of the silvicide reported no spills elsewhere across the treated area.

Although no previous accidental poisonings of wildlife have been discovered since the use of Silvisar 550 began on Huntington Wildlife Forest, this observation has potential significance. We recommend that operations using silvicides or herbicides containing MSMA pay close attention to handling and application of the chemical. Dosages should be carefully controlled to minimize overflowing the axe cuts. Timing of application should be carefully considered, especially to limit its use during times when certain wildlife species display seasonal attraction to sodium. Further studies are needed to fully evaluate the effects of this chemical on free-ranging wildlife.

We sincerely appreciate the expertise

and assistance of W. B. Stone in the diagnosis and necropsy of the deer. We thank W. Winner, J. Bopp and J. Hirsch for field assistance; G. Baldassarre, B. W. Breitmeyer, D. L. Garner, R. D. Nyland, R. W. Sage, and P. F. Steblein for valuable critiques of earlier drafts; D. J. Gefell for assistance with calculations; Mehdi Shaygani, State Bacteriology Laboratory, Wadsforth Center for Laboratory and Research, New York State Health Department, Albany, New York; the Adirondack Ecological Center, Newcomb, New York; the New York Department of Environmental Conservation, Wildlife Resources Center, Delmar, New York; and Hazelton Laboratories, Madison, Wisconsin. This report was funded by the New York State Legislature, under the auspices of the Adirondack Wildlife Program.

LITERATURE CITED

ABDELGHANI, A. A., A. C. MASON, A. C. ANDERSON, A. J. ENGLANDE, AND J. E. DIEM. 1976. Bioconcentration of MSMA in crayfish (*Procambarus clarkii*). Trace Substances and Environmental Health 10: 235–245.

BOYCE, A. P., AND L. J. VERME. 1954. Toxicity of arsenite debarkers to deer in Michigan. Michigan Department of Conservation, Game Division. Report #2025. Lansing, Michigan, 10 pp.

DICKINSON, J. O. 1972. Toxicity of the arsenical herbicide monosodium acid methanearsonate in cattle. American Journal of Veterinary Research 33: 1889–1892.

EXON, J. H., J. R. HARR, AND R. R. CLAEYS. 1974. The effects of long term feeding of monosodium acid methanearsonate (MSMA) to rabbits. Nutrition Reports International 9: 351–357.

JUDD, F. W. 1979. Acute toxicity and effects of sublethal dietary exposure of monosodium methanearsonate herbicide to *Peromyscus leucopus* (Rodentia: Cricetidae). Bulletin of Environmental Contamination and Toxicology 22: 143–150.

MATTFELD, G. F., J. E. WILEY III, AND D. F. BEHREND. 1972. Salt verses browse-seasonal baits for deer trapping. The Journal of Wildlife Management 36: 996–998.

MIDWEST RESEARCH INSTITUTE. 1975. Substitute chemical program. *In* Initial scientific review of MSMA/DSMA. Environmental Protection Agency, U.S. Department of Commerce, Washington, D.C., NTIS #PB-pp, pp. 251–566.

NAQVI, S. M., V. O. DAVIS, AND R. M. HAWKINS. 1985. Percent mortalities and LC_{50} values for selected microcrustaceans exposed to Treflan, Cutrine-plus, and MSMA herbicides. Bulletin of Environmental Contamination and Toxicology 35: 127–132.

NORRIS, L. A., P. R. CANUTT, AND J. F. NEUMAN. 1983. Arsenic in the forest environment after thinning with MSMA and cacodylic acid. Bulletin of Environmental Contamination and Toxicology 30: 309–316.

PRUKOP, J. A., AND N. L. SAVAGE. 1986. Some effects of multiple, sublethal doses of monosodium methanearsonate (MSMA) herbicide on hematology, growth, and reproduction of laboratory mice. Bulletin of Environmental Contamination and Toxicology 36: 337–341.

SHARIATPANAHI, M., AND A. C. ANDERSON. 1984. Uptake, distribution and elimination of monosodium methanearsonate following long term oral administration of the herbicide to sheep and goats. Journal of Environmental Science and Health B19: 555–564.

THATCHER, C. D., J. B. MELDRUM, S. E. WIKSE, AND W. D. WHITTIER. 1985. Arsenic toxicosis and suspected chromium toxicosis in a herd of cattle. Journal of the American Veterinary Medical Association 187: 179–182.

WEBB, W. L., E. M. ROSASCO, AND S. V. R. SIMPKINS. 1956. The effect of chemical debarking on forest wildlife. *In* Chemical debarking of some pulpwood species, Technical Publication #79, H. Wilcox, F. J. Czabator, G. Gerdami, D. E. Moreland, and R. F. Smith (eds.). State University of New York, College of Environmental Science and Forestry, Syracuse, New York, pp. 35–43.

WEEKS, H. P., JR. 1978. Characteristics of mineral licks and behavior of white-tailed deer in southern Indiana. American Midland Naturalist 100: 384–395.

Received for publication 21 April 1988.