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**LEAD POISONING IN CATTLE; CASE STUDY IN JIROFT**

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**ABSTRACT**

Lead poisoning is one of the most important metal toxicity in domestic animals. In cattle, at Intake levels below those which are associated with clinical signs, there are metabolic changes accompanied with a decreased rate of growth. In this study, blood lead concentration of cattle has been measured in Jiroft. Clinical lead poisoning in cattle is an important cause of economic loss in beef and dairy herds. Lead poisoning is a common toxicity and individual animal cases often present to veterinary practitioners. The potential for lead residues within exposed, but asymptomatic cattle should also be considered. Sometimes, herd outbreaks of toxicity are seen after herd exposure to a lead source. Veterinarians commonly submit blood or tissue samples to confirm the diagnosis in individual animals with clinical signs; however, they do not routinely screen asymptomatic animals within these herds for evidence of exposure. Blood samples from 3 groups of cattle with confirmed exposure to lead were analyzed as part of a research project at the WCVI; summaries of the analyses, as well as a discussion of how these animals were treated and monitored are presented in this issue. All groups contained some asymptomatic cattle with elevated concentrations of lead in the blood. In herd exposures to lead that result in clinical signs, all the cattle should be tested prior to sale or slaughter to reduce the potential for entry of lead into the food chain.

**Keywords: Cattle, Blood, Lead, Jiroft**

**INTRODUCTION**

The toxicity of lead has been recognized for a disease of the wealthy in Ancient Rome. 4000 years. Lead poisoning or plumbism was Lead salts were used in cosmetics,

sweeteners, cooking pots, wine urns, and aqueducts. The daily lead intake of the ancient Romans was estimated to be 35-250 mg/day compared with 0.3 mg/day for people residing in the USA in the 1980s. Today, lead poisoning is primarily associated with lower socio-economic status. The majority of human lead exposure comes from environmental sources such as old paint, PVC blinds, ceramics, and lead pipes in older homes. Lead is a common cause of toxicity in cattle and may cause significant economic loss. Herd outbreaks of lead poisoning are usually seen during the spring and summer; beginning shortly after turn out to pasture (Figure 1). The relatively high frequency of lead ingestion in cattle is likely associated with their natural curiosity, propensity to lick, and lack of oral discrimination. Young cattle are most commonly affected. Lead is found

in plates inside vehicle batteries, in used motor oil or grease from engines that burned leaded gas (pre-1990), and in particles of metallic lead such as lead shot, solder, and lead windows. Other common sources of lead include old paint, paint cans, and caulking compounds (pre-1950), roofing shingles, and ash from locations where painted lumber or garbage containing a lead source was burned. High levels of lead in plants and soil are sometimes found near areas with oil field spillage, smelters, battery reclamation plants, and lead-mine tailings. Animals grazing in pasture bordering highways of countries where leaded gasoline is still used have higher background lead concentrations. Accidental herd exposures occasionally occur where large numbers of animals have ingested high amounts of lead.

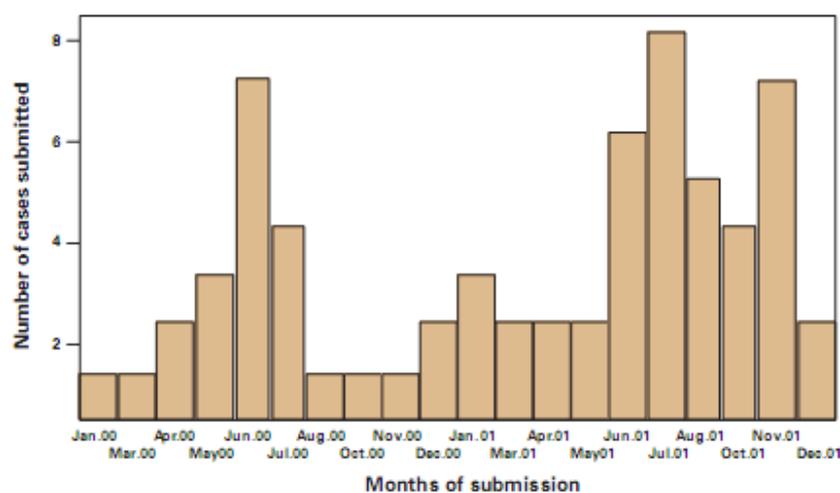


Figure 1: Cases of lead toxicity submitted to Prairie Diagnostic Services during 2013-2014

**Clinical signs**

Clinical signs of lead poisoning in cattle may be acute or subacute because of differences in the dose of lead consumed and the resulting severity of symptoms. In acute poisoning, animals are often found dead without any signs of illness. Affected animals may demonstrate staggering, muscle tremors of the head and neck, twitching of the face and ears, champing of the jaws, blindness, aggression, head pressing, and possibly tonic-clonic convulsions. Subacute lead poisoning is more common, with animals showing symptoms associated with cerebral edema, including blindness with a diminished or absent palpebral reflex, incoordination, staggering, anorexia, teeth grinding, and rumen stasis. Cattle may initially show constipation that progresses to diarrhea. Mid- to late-gestational abortion has been reported. Chronic lead poisoning from long-term, lowlevel exposure may potentially result in anemia and decreased hemoglobin synthesis, but this is rare in cattle. Differential diagnoses for cattle showing blindness include polioencephalomalacia, rabies, hypovitaminosis A, and ophthalmitis. Other differential diagnoses associated with tremors and ataxia without blindness include

hypomagnesemia, ketosis, arsenic poisoning, and meningoencephalitis.

In most cases, the top two differentials are lead poisoning and polioencephalomalacia. They affect similar areas of the brain and both can cause ataxia and convulsions, head pressing, and a central blindness with intact (though sometimes diminished) pupillary light reflexes. Diagnosis of the disease is usually based on clinical signs in an individual animal and the confirmation of lead toxicity with a laboratory analysis of lead levels in the blood, kidney, or liver. It is important to initiate a careful search of the environment, since the owner is often unaware of the lead source. Gross necropsy may provide little definitive information, although it is important to search the rumen and reticulum carefully for lead particles or even parts of lead plates.

**Laboratory analysis**

Various samples can be collected for laboratory diagnosis and evaluated for lead concentration, including:

- Whole blood. Collect in a lithium heparin or sodium heparin (green top) vacutainer tube. Serum is not a good diagnostic sample because lead is transported as protein on red blood cell membranes.

- Fresh or frozen kidney. Collect approximately 50 grams. Freeze the sample if it cannot be submitted promptly and keep cool.
- Fresh or frozen liver. Collect approximately 50 grams. Freeze the sample if it cannot be submitted promptly and keep cool.
- Contents of the reticulum. May be collected for close examination to identify lead particulate matter.

Reference values for samples are shown in Table 1. Lead accumulates in liver, kidneys,

and bone. It is filtered from the blood by the liver and kidneys, and is then gradually transferred to bone through equilibration. Only 2% to 10% of ingested lead is absorbed in the gastrointestinal tract. The gastroenteritis of the sub acute form is caused by the direct effect of lead salts on the alimentary mucosa or by vehicles such as oil, which carry the lead. After lead ingestion, lead is excreted in the urine, feces, milk, and bile. Lead can also be transferred across the placenta.

**Table 1: Values of lead in ppm found in cattle samples as classified by the toxicology laboratory at Prairie Diagnostic Services**

	<b>Blood</b>	<b>Kidney*</b>	<b>Liver*</b>
<b>Background/normal</b>	Usually < 0.10	0.2 to 2.0	0.1 to 1.0
<b>High normal</b>	0.10 to 0.35	2.0 to 5.0	2.0 to 5.0
<b>Toxic</b>	> 0.35	> 5.0	> 5.0

### Treatment

Treatment of clinical cases is questionable unless the ultimate fate of affected animals can be tracked after remission of clinical symptoms in order to prevent lead from entering the food chain. There are currently no guidelines to follow for assuring that such an animal will be safe for consumption when it reaches slaughter. It is difficult to determine when, or even if, treated animals should enter the food chain. Calcium disodium edetate (CaEDTA) is a chelating

agent that removes lead from bone. It is no longer commercially available, but may be formulated by some pharmacies. Use of this agent is considered “extra-label” pharmaceutical use. Clinical symptoms may initially worsen with CaEDTA due to transient increases in the concentration of lead in the blood following mobilization from the bone. Renal toxicity, intestinal irritation by CaEDTA, and depletion of essential minerals are potential side effects of treatment. Thiamine may be used in addition

to CaEDTA because it helps reduce the deposition of lead in kidney, liver, and neurological tissue. Alone, thiamine does not decrease lead concentrations in tissue, although some alleviation of symptoms may be seen. Other treatments that might limit the absorption of lead include rumenotomy to remove lead pieces and particles, and cathartics such as magnesium sulfate to precipitate soluble lead. Corticosteroids may potentially reduce cerebral edema. Radostits et al discuss the newer treatment and dosage regimens in detail in their textbook, *Veterinary Medicine*.

### **1. A case series in the field management of group exposure to lead**

Of all confirmed cases of lead poisoning diagnosed at the toxicology laboratory of Prairie Diagnostic Service (PDS) in 2001, 14 of 64, or 22% of new submissions, involved herd exposure to a lead source. Field management of lead exposure in groups of beef cattle has not been well documented although some summaries are available. The concentration of lead in the blood of exposed animals does not correlate well with the presence of clinical signs. Asymptomatic animals may contain sufficient lead in their tissues that would make them unsuitable for human consumption. After exposure to lead,

there is no documentation of how long cattle should be held before they can be safely marketed or if, in fact, they should ever be marketed for human consumption. In light of these unanswered questions, initial investigations were conducted at WCVM in 3 groups of cattle, accidentally exposed to lead sources. In all 3 groups, cattle with asymptomatic lead toxicity were discovered. The following are summaries.

#### **1.1. Case 1**

A group of 51 bred heifers, 1 steer, 8 cows, and 8 calves were placed on pasture in autumn. Four heifers were subsequently found dead over a 15-day period. The local veterinarian identified 2 other heifers showing clinical signs of acute lead poisoning and confirmed this with laboratory analysis. Their blood lead levels were 0.45 ppm and 1.12 ppm. Two old vehicle batteries were found on this pasture and the cattle were removed, but additional sick animals were identified. Overall, 8 of 68 animals died due to lead toxicity. On day 23, all exposed animals were uniquely identified, scored on body condition, examined for pregnancy, and blood samples were collected for lead analysis.

Ten percent had toxic concentrations of lead in the blood (>0.35 ppm), 3 were previously symptomatic, and 3 animals were

consistently asymptomatic. Four of these 6 animals were bred heifers. A fifth heifer with blood lead levels in the toxic range appeared to have recently aborted. The sixth animal was a calf. Four other pregnant heifers (6.7%) had blood lead levels in the high normal range (0.1 – 0.35 ppm), indicating abnormal exposure to lead (Figure 2).

### 1.1. Case 2

The herd owner found a cow and calf on pasture late in the summer showing neurological symptoms typical of lead poisoning. The cow died and analysis of the kidney confirmed lead poisoning with a lead concentration of 134 ppm. Blood from the calf was also consistent with lead poisoning

with 0.62 ppm of lead. A shed that contained old batteries had been burned down. Four of the 22 animals (18.2%) on this pasture died. On day 45 after the first case, the local veterinarian collected blood samples from 2 other animals showing clinical signs. On day 77, blood samples for lead analysis were collected from 25 potentially exposed animals, due to some uncertainty about which animals had been in that pasture. Three consistently asymptomatic animals (12%) had lead levels in the toxic range, and 10 (40%) were in the high normal range (Figure 3). One of the 10 had been previously treated.

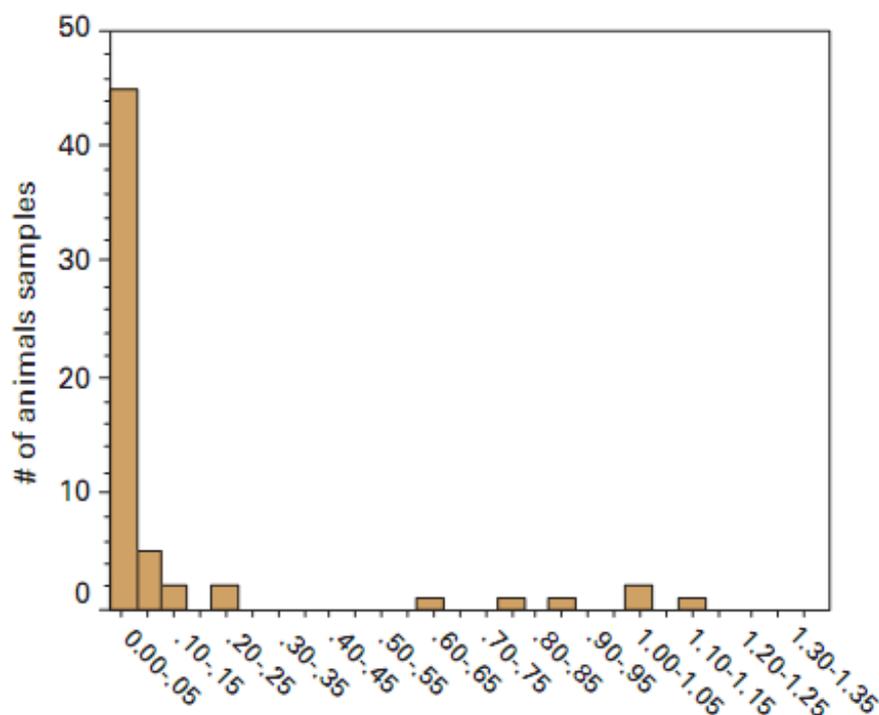


Figure 2: Distribution of lead concentrations in the blood of all potentially exposed animals from Herd 1 on Day 23 after first diagnosis and subsequent exposure was prevented

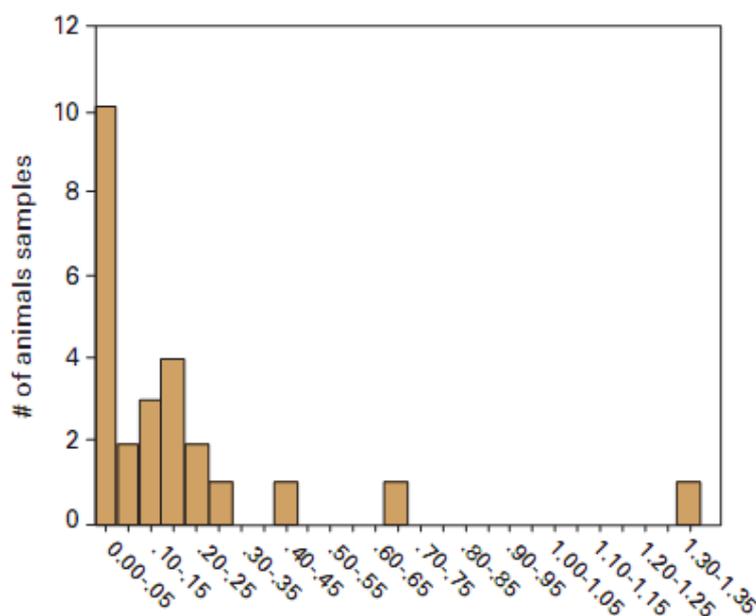


Figure 3: Distribution of lead concentrations in the blood of all potentially exposed animals from Herd 2 on Day 77 after first diagnosis and subsequent exposure was prevented

### 1.2. Changes in blood lead concentration after exposure

Veterinarians collected blood samples from several animals in both herds 1 and 2, either 2 or 3 times after the initial diagnosis to monitor the decline in the concentration of lead. The animal that had been treated was excluded from the calculations. In Herd 1, additional blood samples were collected from all animals with previously high normal or toxic blood concentrations of lead on day 92 and again on day 188. The concentrations of lead in the blood did not decrease by day 92, but had decreased by day 188. On day 188, 1 of 4 animals with initial toxic lead concentrations still had blood concentrations in the toxic range ( $>0.35$ ), and 2 of 4 still had blood levels of lead in the high normal range

(0.1-0.35 ppm). All 4 heifers with lead concentrations in the blood within the high normal range at day 23 had their lead concentration drop to normal ( $<0.1$  ppm) by Day 188.

The mean reduction in the concentration of lead in the blood of exposed animals was 0.046 ppm every 30 days (95% CI, 0.017 to 0.075 ppm). The median half-life of lead in the animals retested from herds 1 and 2 was 63 days (interquartile range, 34 to 107 days)

### DISCUSSION

This preliminary investigative field study demonstrated that between 4% and 12% of asymptomatic cattle had toxic concentrations of lead in their blood. Another 7% to 40% had concentrations of lead in the blood that would be considered high normal, suggesting

substantial lead exposure. These data suggest that when a diagnosis of lead toxicity has been confirmed, all cattle with exposure to the lead source should be tested prior to sale, even if asymptomatic. Asymptomatic animals may be unsuitable for immediate entry into the food chain. Research is necessary to develop better guidelines to determine when or if these animals should be slaughtered.

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