Large Animal VETERINARY Rounds

Now available on the Internet
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OCTOBER 2002
Volume 2, Issue 8

AS PRESENTED IN THE ROUNDS OF THE DEPARTMENT OF LARGE ANIMAL CLINICAL SCIENCES OF THE WESTERN COLLEGE OF VETERINARY MEDICINE, UNIVERSITY OF SASKATCHEWAN

# Lead poisoning in cattle: Implications for food safety

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**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 WCVM; 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.**

## Lead poisoning: a history

The toxicity of lead has been recognized for 4000 years. Lead poisoning or plumbism was a disease of the wealthy in Ancient Rome. 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.[1] 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.[2-6] Herd outbreaks of lead poisoning are usually seen during the spring and summer,[2,3] beginning shortly after turnout 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 pastures 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.

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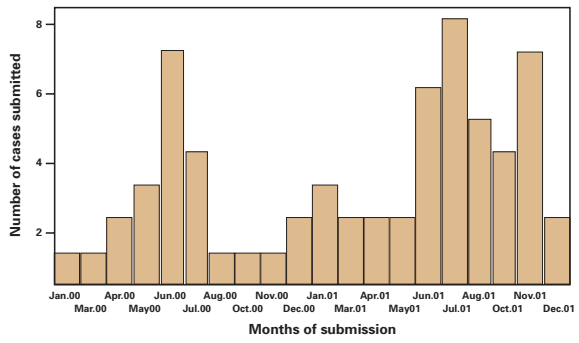
The Canadian Veterinary Medical Association recognizes the educational value of this publication and provides support to the WCVM for its distribution.

**Figure 1: Cases of lead toxicity submitted to Prairie Diagnostic Services during 2000-2001**




## 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, low-level 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.[7,8] 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:

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

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

* Wet Weight

- *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 a proteinate 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.[8] The gastroenteritis of the subacute 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.

## 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. Corticosteriods may potentially reduce cerebral edema. Radostits et al[7] discuss the newer treatment and dosage regimens in detail in their textbook, *Veterinary Medicine*.

## Prevention

Prevention of lead poisoning is very important and should be emphasized. The risk of exposure in cattle can be reduced with a few simple precautions:

- Check all pastures prior to turnout
- Do not use pasture for garbage disposal or old vehicles.
- Fence off high-risk areas or remove high-risk items.

Burning does not reduce the amount or toxicity of lead. The lead is still present in the ash and cattle commonly ingest this.

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

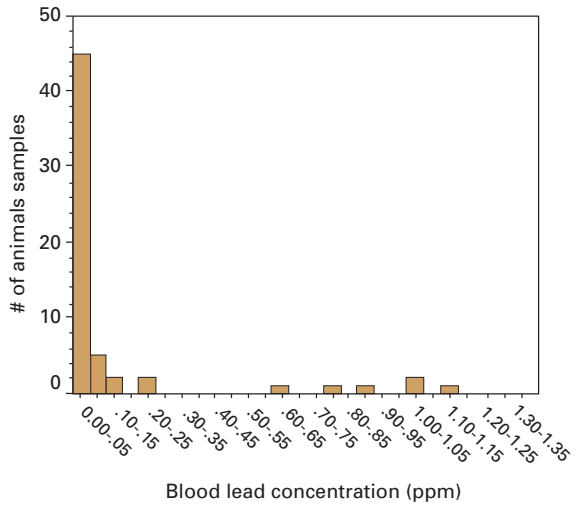
Of all confirmed cases of lead poisoning diagnosed at the toxicology laboratory of Prairie Diagnostic Service (PDS) in 2001, 14 of 64, or 22% of new submissions, involved herd exposure to a lead source. Field management of lead exposure in groups of beef cattle has not been well documented although some summaries are available.[9] The concentration of lead in the blood of exposed animals does not correlate well with the presence of clinical signs.[8] 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.

### *Herd 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[6] had toxic concentrations of lead in the blood

**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[19]**

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(>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).

Because lead can cross the placenta and accumulate in the fetus leading to abortion or stillbirth, the pregnancies were followed in these heifers. No abortions occurred in this group, although 1 calf was lost at birth. The lead concentration in blood samples collected from the calves during the postnatal period ranged between 0.010 and 0.095 ppm, indicating no substantial lead accumulation in the neonates. All 7 of the calves survived until weaning with no reported negative health consequences. Because lead is excreted in the milk, blood samples from these calves at weaning would have been required to determine the effect of lactation on lead accumulation in these animals, but were not available.

### *Herd 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

**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[19]**

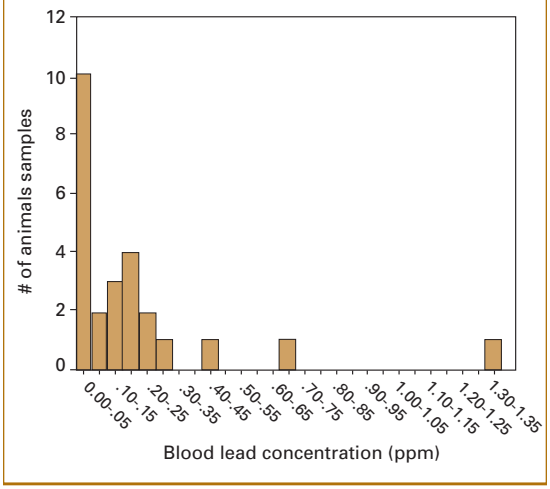


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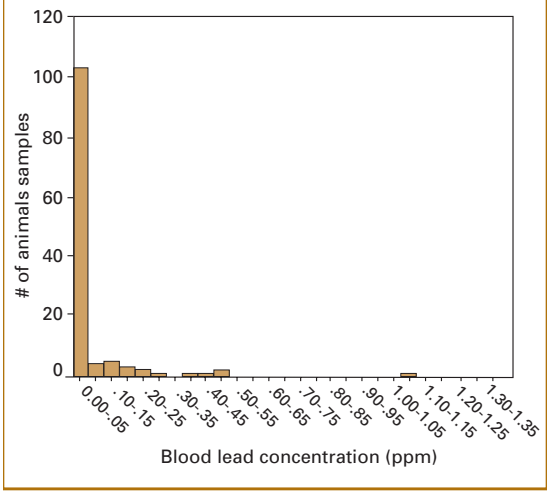
**Figure 4: Distribution of lead concentrations in the blood of all potentially exposed animals from Herd 3 on Day 7 after first diagnosis and subsequent exposure was prevented[19]**

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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.

### *Herd case #3*

A local veterinarian performed a necropsy on the fourth death within a week in a group of 128 steers on summer pasture. Analysis of liver samples confirmed lead poisoning with a toxic lead concentration of 8.92 ppm. The second day after initial diagnosis within the herd, another steer showed clinical signs of lead poisoning. This steer died despite treatment, and the lead concentration in the liver was 6.35 ppm. Overall, 5 of 128 (3.9%) animals died due to lead toxicity. Between Day 7 and 19, the remaining group of 123 had blood samples collected for lead analysis. None showed clinical signs with the possible exception of one animal that had demonstrated unusual behaviour 1 week prior to the deaths, but recovered without treatment. The analysis identified 5 more animals with blood sample lead concentrations in the toxic range (4.1%), and 11 animals in the high normal range (8.9%; Figure 4).

## 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.

In Herd 2, 10 animals with high normal or toxic concentrations of lead in their blood at the first testing were retested on day 130; 2 were still in the toxic range and 3 were in the high normal range. Three finished steers, identified as exposed on previous blood tests, were retested again on day 229. Of these 3 animals, 1 was still toxic, and the other 2 had blood lead concentrations in the high normal range.

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.

## When is an exposed animal safe to slaughter?

One study calculated a half-life of 10.5 days (SE, 1.1 days) for lead concentrations in the blood in a large group of cattle exposed to lead-contaminated feed.[10] In another study, the half-life of lead in the blood ranged between 48 and 2507 days.[11] This long and highly irregular excretion pattern is similar to that observed in the present study where the median half-life of lead was 63 days (interquartile range, 34 to 107 days). Variability in these half-life calculations is likely due, in part, to the inability to accurately estimate the continued absorption, distribution, redistribution, and fluctuation of lead in the rumen, reticulum, blood, liver, bile, kidney, urine, and bone of affected animals. These data suggest that it may take 6 to 12 months or more for blood concentrations of lead to return to background levels in asymptomatic animals**.** In general, the higher the initial concentration of lead in the blood, the longer the animal takes to return to background levels. Lead concentrations in the blood will vary dramatically if continued long-term absorption of lead from particles sitting in the rumen occurs; the decline is even less predictable in those cases. Slaughter of animals exposed to lead should be discussed with the Canadian global Food Animal Residue Avoidance Database (gFARAD) at their toll free number 1-866-243-2723.

Pregnant animals represent a unique concern.[12] At parturition, there is an increase in calcium requirements, leading to the mobilization of calcium from bone. Concentrations of lead in the blood increase at parturition likely due to mobilization of lead from the bone along with the calcium.[13] Lead is also excreted in milk.[14] Exposed, but healthy heifers and cows, may be suitable for replacements in the breeding herd. However, before this can be recommended, a study of long-term fertility in these animals and the distribution of lead in the tissues of their offspring is necessary.

Health Canada develops standards for residues in food. They focus on foods that are a large portion of human diets or are known to sometimes contain higher levels of lead. Guidelines for the maximum allowable levels of lead in drinking water, baby food, and evaporated milk exist. For example, the Guidelines for Canadian Drinking Water Quality list the maximum acceptable concentration for lead as 0.10 ppm.[15] There is no specific regulated amount of lead permissible in beef. The lack of specific guidelines puts the responsibility on the local veterinarian and the herd owner to determine if or when it is safe to release an exposed animal for potential entry into the food supply. The Canadian Food and Drug Act, chapter F-27, paragraph 4 (applicable portions), states: "No person shall sell an article of food that: (a) has in or on it any poisonous or harmful substance; (b) is unfit for human consumption; … (d) is adulterated…."[16] The Canadian Food Inspection Agency, is responsible for monitoring the safety of meat products processed in federal plants.

Economic losses from lead poisoning in the 3 herds described above were not limited to the acute death losses, but also included further economic losses from delayed or lost opportunity to slaughter exposed cattle. Clinical lead toxicity is only the tip of the iceberg when considering the total cost of herd exposure. The extent of asymptomatic lead toxicity in the Canadian cattle industry is unknown, but based on previous surveys of organs collected at Canadian slaughter facilities, it is extremely low.[17,18] Testing of known exposed animals prior to sale is recommended when a diagnosis of lead toxicity has been confirmed in a herd. It may take at least 6-12 months for blood level concentrations to return to background concentrations, therefore, it is important to discuss the timing for slaughter of exposed animals with gFARAD. Future efforts toward agreed-upon strategies and guidelines for long-term management and disposition of animals poisoned with lead would significantly aid in efforts to manage this disease.

***Acknowledgements:*** *The herd investigations used in this review were funded in part by the Saskatchewan Department of Agriculture and Food.*

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**References**

1. Needleman HL. *History of Lead Poisoning in the World*. 1999. *http://www.leadpoison.net/research.htm*. Accessed: September 26, 2002.
2. Blakley BR. The incidence and seasonal characteristics of veterinary toxicosis in Saskatchewan. *Can Vet J* 1984;25:17-20.
3. Yonge KS, Morden BB. Bovine lead poisoning in Alberta: A management disease. *Can Vet J* 1989;30:42-45.

4. Hoff B, Boermans HJ, Baird JD. Retrospective study of toxic metal analyses requested at a veterinary diagnostic toxicology laboratory in Ontario (1990-1995). *Can Vet J* 1998;39:39-43.
5. Blakley BR. A retrospective study of lead poisoning in cattle. *Vet Hum Toxicol* 1984;26:505-507.
6. Bratton GR, Zmudski J. Laboratory diagnosis of Pb poisoning in cattle: A reassessment and review. *Vet Hum Toxicol* 1984;26:387-392.
7. Radostits OM, Gay CC, Blood DC, Hinchcliff KW. *Veterinary Medicine*, 9th ed. London: W.B. Saunders Company Ltd; 2000:1575-1585.
8. Osweiler GD. *Toxicology*. Philadelphia, PA: Williams & Wilkins; 1996:191-197.
9. Stair EL, Kirkpatrick JG, Whitenack DL. Lead arsenate poisoning in a herd of beef cattle. *J Am Vet Med Assoc* 1995;207:341-343.
10. Baars AJ, Van Beek H, Visser IJR, et al. Lead intoxication in cattle: a case report. *Food Addit Contam* 1992;9:357-364.
11. Rumbeiha WK, Braselton WE, Donch D. A retrospective study on the disappearance of blood lead in cattle with accidental lead toxicosis. *J Vet Diagn Invest* 2001;13:373-378.
12. Sharma RM, Buck WB. Effects of chronic lead exposure on pregnant sheep and their progeny. *Vet Toxicol* 1976;18:186-188.
13. Galey FD, Slenning BD, Anderson ML, et al. Lead concentrations in blood and milk from periparturient dairy heifers seven months after an episode of acute lead toxicosis. *J Vet Diagn Invest* 1990;2:222-226.
14. Osweiler GD, Carson TL, Buck WB, Van Gelder GA. *Clinical and Diagnostic Veterinary Toxicology* 3rd ed. Dubuque, Iowa: Kendall/Hunt Publishing Company; 1985:107-120.
15. Sous-comité fédéral-provincial-territorial sur l'eau potable (SCEP) appartenant du comité fédéral-provincial-territorial de l'hygiène du milieu et du travail; mars 2001. *www.hc-sc.gc.ca/ehp/dmh/catalogue/dpc_pubs/sommaire.pdf.* Accessed: September 26, 2002.
16. Department of Justice, Canada. *Food and Drugs Act*, Chapter F-27: *http://laws.justice.gc.ca/en/F-27/55581.html.* Accessed: September 26, 2002.
17. Korsrud GO, Meldrum, JB, Salisbury CD, Houlahan BJ, Saschenbrecker PW, Tittiger F. Trace element levels in liver and kidney from cattle, swine and poultry slaughtered in Canada. *Can J Comp Med* 1985;49:159-163.
18. Salisbury CDC, Chan W, Saschenbrecker PW. Multielement concentrations in liver and kidney tissues from five species of Canadian slaughter animals. *J Assoc Off Anal Chem* 1991;74(4):587-591.
19. Waldner C, Checkley S, Blakley B, Pollock C, Mitchell B. Managing lead exposure and toxicity in cow-calf herds to minimize the potential for food residues. *J Vet Diagn Invest* 2002;14 (In Press).

## Abstract of Interest

### A retrospective study on the disappearance of blood lead in cattle with accidental lead toxicosis

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Lead poisoning in cattle and other food animals is of public health significance because of the potential for human exposure to lead through ingestion of contaminated meat and milk products derived from lead-poisoned animals. In Michigan, lead poisoning in livestock is a reportable disease, and positive cattle are quarantined until they test negative (< 0.05 ppm blood lead). There is surprisingly little information on blood lead kinetics in cattle. The half-life has been variably reported as 9 weeks and 1-2 months. Because these data did not fit those obtained from cases received at the Michigan State University Animal Health Diagnostic Laboratory, a retrospective study was conducted to review all cases of accidental lead poisoning in cattle between 1990 and 1998. This information is needed to estimate when quarantined lead-poisoned cattle can be released. The results showed that the half-life of blood lead was quite variable and ranged from 48 to 2,507 days. The shortest half-lives (48, 56, and 57 days) were found in a lactating herd of 20-month-old heifers. The longest half-life, 2,507 days, was found in a 9-month-old castrated bull, which ingested a discarded automobile battery. Of the 24 animals monitored, only 8/24 (33%) had half-lives between 6 and 14 weeks. In conclusion, the half-life of blood lead is difficult to predict in accidental cases of cattle poisoning.

*J Vet Diagn Invest* 2001;13:373-378.

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