Spring 1982

The Effects Of Lead Emissions On Livestock In The Area Of The East Helena ASARCO Smelter

Randy Sacry
Carroll College

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THE EFFECTS OF LEAD EMissions
ON LIVESTOCK IN THE AREA OF THE EAST HELENA ASARCO SMELTER

Submitted in Partial Fulfillment of the Requirements for
Graduation with Honors to the Department of Biology
at Carroll College, Helena, Montana

Randy Lee Sacry
25 March 1982
This thesis for honors recognition has been approved for the Department of Biology by:

Dr. James J. Manion, Director

Dr. John A. Christenson, Reader

Robert J. McCarthy, Ph.D., Reader

23 March 1982
carroll of montana
March 16, 1982

Dear Thesis Board Members:

Some years ago a Theologian at Carroll College was asked to be a reader for a Thesis in the department of Physics. Dutifully, the kind reader struggled through the text; he then affixed a paragraph:

"Nothing herein seems to be contrary to faith and morals."

Randy Sacry and I have been good friends during his years at Carroll College. Thus I am pleased to have been a reader of his Thesis. I found the presentation very interesting and I am sure that it involved a great deal of work and laboratory diagnosis.

I have made some corrections in typographical errors, etc. For the most part it is very well done. I have suggested that the typing of the "tables" would make that part more presentable.

I would recommend "acceptance with distinction".

With kind personal regards,

Very sincerely yours,

Robert J. McCarthy, Ph.D.
Department of Theology
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I would like to give my thanks and appreciation to the many people who made my thesis a true learning experience.

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Many thanks go to ranchers Don Burnham, David Diehl, Curt Gardner and Paul Kleffner for their patient help in obtaining samples from their livestock.

I'd like to thank veterinarians Robert Painter and Lucy Dayton for their donations of references and sampling apparatus and also for steering me to Billy LaFromboise, who taught me the proper method of drawing blood.

I'd also like to thank Stan Sternberg for his contribution of reading material and the wind roses and my father-in-law, Bill Muldowney, for sharing his artistic talent.

The generosity and genuine helpfulness of everyone assisted in making my research and data collection go very smoothly. Again, Thank-you to all of you.
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The East Helena AGARCO smelter is a lead refining plant and its effects on livestock and humans in the area have long been a controversy. In an attempt to shed some light on this issue, blood samples were taken from livestock in the East Helena area and then analyzed for their lead content. Also noted in this study were such things as food type, food source, wind direction, time of year of sampling and the number of years the animal has been in the area. While the study shows the blood lead levels being higher around the smelter, they do not reach critical stages and any contamination that now occurs is likely due to improper ranch management.
Lead poisoning has been a part of history since before the time of Christ, yet even with the increased awareness we have today it is still a common toxicant in large and small animals.

Oral consumption is the major route of poisoning in animals. The major sources of lead for ranch animals are paints, old batteries, old oil and grease from tractors, pipes and galvanized metals. These sources of lead are usually picked up by the animals licking old metal and cans in junk piles or old buckets of oil and grease. Grease may be an especially potent source since it may contain as much as fifty percent lead, but the most common source is old paint and oil.

In horses most cases of poisoning have involved contaminated pastures near smelters. Airborn particulate lead lands on grass and forage and is ingested during consumption of these natural foods. Cattle were found to be unaffected by the same levels that caused problems in horses.

Industrial contamination may cause isolated problems where fumes or residue runoffs from lead reclaiming or processing plants are located in the immediate vicinity.

Minor sources are lead pipes or pipes with soldered joints used to carry drinking water. Lead arsenate pesticides are a cause of poisoning in cattle, although the problem here is usually due more to the acute toxicity of the arsenic.
TOXICITY

Lead accumulates in the body, so chronic exposure to small amounts of lead may lead to toxicosis.

In the case of chronic exposure, a dosage in the range of 1-3 g/day is toxic to calves, 6-7 mg/Kg of body weight per day is toxic to cattle, and 2-7 mg/Kg of body weight per day in horses is considered critical.

For acute exposure, the lethal levels are usually considered to be 400-600 mg/Kg of body weight in calves and 600-900 mg/Kg of body weight in adult cattle. Horses tend to be more susceptible with acute dosage being 500-750 grams total dosage. This is a result of horses eating closer to the ground and tending to pull up plants by the roots. In this, they may receive additional exposure from contaminated soils.

All species of animals are susceptible to lead but there are certain factors that influence toxicity.

A large factor is age, as is shown in the chronic and acute dosages considered to be critical. The younger the animal the more susceptible it is.

Eating habits and sensitivity of different species also play a role as in the case of horses. The extra body sensitivity coupled with the fact that horses eat closer to the ground gives them a much greater susceptibility. This would be increased even further in the case of sheep and other animals that eat still closer to the ground.
Another important factor is the reproductive state of the animal. If the adult female is pregnant, the risks will be much higher due to the added stress pregnancy puts on the body. Also, the fetus may be endangered by the passage of lead across the placental membrane. In the case of sheep, amounts as low as 1 mg/Kg of body weight per day were shown to be lethal, whereas for non-pregnant ewes the dosage was 3 mg/Kg of body weight per day. 

In general, large acute doses of lead are more of a problem in animals than small chronic ones, and the form of the lead also accounted for some differences. For example, lead salts are most easily absorbed. 

The route of entry plays an important role in toxicity. The main route of entry is through ingestion, however, the percentage of absorption in this situation is only 1-2 percent. For inhaled particulates the fractional absorption may be considerably higher. 

Although the mechanism is not known, the presence of other toxins in the body seems to enhance the effects of the toxicity of the lead. Also, if the animal is in poor general health the effects of the toxicity are increased. If the mucosa of the gastrointestinal tract is damaged the absorbance in the body systems is facilitated.

Past exposure will have an effect in that very low chronic exposure will build a lead tolerance, but previous acute exposure increases susceptibility to a recurrence of poisoning.

A major role in lead toxicity is played by bone. As the
blood passes through bone, unbound lead is bound to bone substance and immobilized. These binding sites are particularly concentrated in the bone growth regions.
After lead is absorbed across the mucosal membrane, a large proportion of it is carried on the erythrocytic membrane. In cattle this has been shown to be as high as 63-70 percent. Unbound lead is in dynamic equilibrium with lead bound to the erythrocytes and serum albumin. It is the unbound portion that is thought to be responsible for the toxic effects of lead.

Lead is moved through the liver and excreted with the bile and also by the kidneys. Because of this the liver may contain large amounts of lead and so may the renal cortex of the kidney. Lead is also excreted with milk and hence may be harmful to unweaned calves.

Lead is found initially in the soft tissues, but later is deposited in bones, especially the growth areas as cited before. The bones may then be carrying 90-98 percent of the lead burden of the body. Bone is an important detoxification area for chronic lead exposure. On saturation of the bone, signs of toxicosis appear due to a rise in the levels of lead in soft tissue and the blood. If exposure ceases, concentrations in the bones will decrease and blood lead concentrations may rise for 1-2 months until the lead is carried out of the body. It is this high concentration of lead in the bones that makes the study of the effects and mechanisms of lead exposure so difficult.
LEAD is relatively insoluble and even soluble forms like lead acetate form insoluble compounds in the gastrointestinal tract. This accounts for the poor absorbance of 1-2 percent mentioned earlier. However, even though a small percent is absorbed, a large proportion of that is deposited in soft tissue and later in bone. Blood lead levels may then rise 2.5-4 ppm within twelve hours after ingestion. This will be followed by a decline of 1-1.5 ppm within forty-eight to seventy-two hours as lead is deposited in the bone. This slow decline in blood lead emphasizes the slow excretion of lead in untreated animals and the role of lead as a chronic cumulative toxicant. 4

Lead combines with erythrocytes in the blood but unless it is in very high concentrations it is not found in the plasma. Anemia may result from an increased fragility of the red blood cells. This fragility could be caused by basophilic stippling of the cell and leads to premature destruction of the red blood cells. It also causes depression of bone marrow so that fewer cells are produced. The nervous system is affected by decreased blood supply, due to damaged capillaries which result in either edema or collapse of small arteries. 4

Peripheral nerves are affected by a segmental demyelination which interferes with the nerve conduction. The "Roaring" in horses, pharyngeal or buccal paralysis in cattle, and the paralysis of masseter muscles in dogs are evidence of neurological damage of cranial nerves or brain stem nuclei."
In the kidneys lead causes degeneration and death of renal tubule cells. Acute exposure may cause necrosis of the gastrointestinal mucosa. Liver degeneration and necrosis may follow both chronic and acute exposures. In young animals the effect is on metabolically active growth centers in long bones and will result in an increased density which may be detected by X-ray. Lead crossing the placental barrier may cause the fetal liver to accumulate toxic levels. Lead exposure may also cause fetal re-absorption, abortion or sterility.

Lead is removed slowly from the body, primarily through the kidneys and therefore the effects on that organ are easily accounted for.

Subcellular effects are not fully understood. However, lead can cause rupture of lysosomes and release acid phosphatase which is required for energy production and protein synthesis. Lead also interferes with several enzymes involved in the synthesis of heme. One effect is the inhibition of amino levulinic acid dehydrase (ALA-D) needed for the conversion of amino levulinic acid (ALA) to porphobilinogen (PBG), a precursor of heme. Lead also inhibits ferrochelatase, an enzyme of the heme synthesis pathway, which catalyzes the insertion of iron in protoporphyrin IX to form heme. The blocking mechanism can be seen in Fig. 1. In general, lead interferes with thiol-containing (-SH) enzymes.

Lishluis has reviewed the literature and data and has discussed the relationship between blood lead levels and various effects on hematological and biochemical parameters. He sub-
divides the total lead burden into three separate pools which are rapid exchange of lead in blood and soft tissue, intermediate exchange of lead in skin and muscles and intermediate and slow exchange of lead in bone marrow and dense bone. The idea of pools of lead is widely accepted and substantiated by large volumes of data accrued through both animal and human experimental subjects. One could hypothesize the existence of these pools since there is a large number of lead binding sites in organs and tissues. The rates of exchange in these pools depend on the stability of the lead complexes, the abundance of lead binding sites, the concentration of competing cations and the affinity of these cations for the same binding sites. This concept is also valid in elucidating the interaction of lead and zinc on ALA-D. This enzyme, which is activated by zinc and inhibited by lead, is rich in sulfhydryl groups having fifty-six per molecule. The importance of the effect of lead on ALA-D inhibition is still uncertain since ALA-D is not a rate limiting enzyme for heme synthesis. A great deal of ALA-D must be inhibited before any effects are shown and even then treatment with ZnCl₂ restores full enzyme activity. Even though the effect of ALA-D inhibition may not be important, it is a sensitive test for lead exposure.

Detail is not known on the effect lead poisoning has on the mitochondria, but accumulation of lead is especially high in liver and kidney mitochondria. In vivo kidney mitochondria show increased swelling when compared to mitochondria suspended in KCl. This shows that respiring mitochondria have a greater uptake of
lead.

Lead inhibits the succinate oxidation in submitochondrial particles. Oxidation of NADH by submitochondrial particles is also affected, although by higher concentrations than succinate. Also, very low levels of lead have been found to be inhibitory to pyruvate oxidase in vitro. These observations suggest that pyruvate dehydrogenase and possible other Krebs cycle enzymes are more sensitive to lead than is the electron transport system. Measurement of succinate exchange indicates that lead may interfere with the transport of succinate and other substrates into the matrix space of intact mitochondria. Some respiratory sequences, however, seem to be rather insensitive to lead.

Lead induces passive osmotic swelling in intact mitochondria suspended in KCl, a result which indicates that the membrane has been made permeable to potassium ions and chloride ions.

Although the affects on the mitochondria are not fully understood, damage to this versatile organelle could be potentially harmful.

It seems that the major problem with the accumulation of lead is in the subsequent disruption of heme synthesis, which is well documented in cases of lead poisoning.
The role of proper nutrition is important, especially as it pertains to heavy metal emission plants.

Many nutrients have been found to have an effect on or be affected by lead toxicity. A detailed report of this may be seen in Table 1. This shows the most striking inter-relationship of dietary constituents with oral lead toxicity.

There are a number of reasons for the importance of proper nutrition, the most simple being that healthy animals are much less susceptible to lead poisoning. Another reason is inadequate nutrition contributes to insufficient amounts of zinc, copper and iron. These minerals compete with lead for the active sites on enzymes. This makes the lead unable to bind and it will pass out of the body more quickly.\textsuperscript{11}

Another important factor and one that very much comes into play around lead emission plants is the amount of supplemental food given to livestock in the winter. If animals are not fed adequate amounts during cold months they are forced to consume forage lacking in nutritional value and to graze closer to contaminated soils. That the soil around the East Helena smelter is contaminated is unquestionable.\textsuperscript{12} See Table 7.
CLINICAL SIGNS

There are two systems affected in lead poisoning in animals, the central nervous system and the gastrointestinal tract. Central nervous system problems occur in about 90 percent of the cases and the gastrointestinal complications in approximately 60 percent. These symptoms are summarized in Table II.

The first evidence of lead intoxication is often depression and anorexia. Anorexia is almost a constant occurrence in lead poisoning. Constipation followed by diarrhea, abdominal stress and "tucked" abdomen are also common symptoms.

Cattle may grind their teeth and appear to be chewing without swallowing and give the impression of excess salivation. "Blind staggering" is a bovine condition caused by the lead affecting the optic nerve. The animal cannot see to determine whether an object is mobile and will push at it continually.

Other clinical signs are eyelids that blink in a violent manner and fine muscle tremors. A rhythmic twitching of the ears or bobbing of the head may be seen as well as marked excitement and convulsive seizures. These latter symptoms are usually the final stages before death and prognosis is poor.
Blood was drawn from three sample herds in the area of the American Smelting and Refining Company's East Helena plant and from one herd in the Helena valley which was used as the control group. Sample sites may be seen on Fig. 2.

The blood drawing was quite simple. There are two main veins commonly used to draw blood. The first, the juglar, runs along the side of the cow's neck. The best procedure is to hold the hand against the vein and allow the pressure to cause the vein to bulge. Once the vein is distended, the needle is inserted and the blood may be drawn. This method requires the cow to be placed in a squeeze chute and nose tongs must be used to hold the head to the left side. The other method involves taking blood from the middle coccygeal vein under the base of the tail. I used the first method, although the "tail" method is becoming increasingly popular.

Once the blood is drawn it is injected into sterile tubes containing sodium oxalate as a preservative. Each tube holds approximately 15 ml and the sample will last for several days.

Two samples were taken from one cow in each herd to check the lab for accuracy.

The blood was sent for analysis to ASARCO Inc., Department of Environmental Sciences, Salt Lake City, Utah.

There are two main procedures for blood lead testing. The first is an extraction method using Triton X-100, ammonium pyrroolidine dithiocarbamate and methyl isobutyl ketone. An atomic
absorption (AA) spectrophotometer is then used and calculations are made using the data obtained. The second method and that employed by the A2ARCO lab is called the boat or Delves cup procedure. This also utilizes an AA spectrophotometer. However, in this case the blood is flamed in a Delves cup and absorption measured. The Delves cup method is inserted as it appears in the lab book. It should be noted that the acceptable margin of error is ±6 μg/100 g to either side of the given sample number and that concentrations of less than 10 μg/100g are not usually reported.
The location of the ranches and the wind roses with respect to the ARCO smelter are found in Fig. 2 and 3.

Cattle from the Curt Gardner ranch were used as the control group. From a herd of thirty cattle, six blood samples were taken.

The Kleffner ranch is of main interest since it gets the major portion of the wind blowing from the direction of the smelter. It also has the greatest amount of particulate matter. From a herd of 40, seven samples were drawn. Two samples taken from each of two horses were also included.

The Bial ranch is also of prime interest since Dave Rich owns land in the westerly direction from the smelter and receives a great deal of wind. Dave owns a large number of cattle, but due to the extensive area over which they graze only six samples were possible.

The Don Farnham ranch is close to the smelter, but the wind is from the direction of the ranch to the smelter. Don runs a large herd of Seminatol cattle from which fifteen samples were taken.

More comprehensive data on the findings from those ranches may be found in Tables III-VI. The information includes type of food, food source, water source, number of years in the area, time of year samples were taken and, of course, blood lead levels. The time of year of sampling is important because January - March is known to be the second highest period in the year for lead poisoning.
CONCLUSION

Before beginning sample collection I talked with the ranchers and found that they had had no problems with lead poisoning in several years. I then talked with biologist Jim Steverson and found that since 1977 vast improvements have been made at the ASARCO plant. The primary improvement was the replacement of an old electrostatic precipitator, for dust removal, with a new acid plant and baghouse. The acid plant is used for making sulfuric acid and during this process the air must be pure. The baghouse is the new dust collector. Other improvements have been the purchase of sweepers, water trucks and sprinklers to decrease dust around the plant. Vent systems have also been added to draw the dust to the baghouse.

There had been some problems with a ranch not included in this study, the Cory ranch. This ranch is found in the same direction as the Kleffner ranch only farther away. From talking to ranchers and reading Dr. Painter's file, malnutrition may have played a role here.8 Since the Cory ranch is farther away than the Kleffner ranch and Kleffner cattle had no problems I would hypothesize that the issue now is not with lead emissions but with contaminated soils.

The wind roses support this hypothesis. As shown in Fig. 3 the roses indicate the wind direction with respect to the microwave tower, which is due south of the smelter four
miles. This tower is approximately the same height as the A5ASCQ emission stack.\textsuperscript{10}

These wind roses indicate the monthly wind directions for one year. The wind is predominantly out of the west, blowing mainly in the direction of the Diehl and Kleffner ranches. If the blood lead of the cattle was due to respiratory intake of particulate lead, cattle from these ranches should show higher readings than Burnham's. As can be seen from Tables III-VI this is not the case. All three ranches have similar readings. In fact, the highest single reading comes from the Burnham ranch.

The fact that the three sample sites show higher lead levels than the control site is to be expected since the smelter has been in the area for 94 years.\textsuperscript{12} The soil is thus going to be contaminated, as is shown in Table VII, and livestock will have a better chance of lead toxicity.

Levels of .35 ppm in blood are considered critical for cattle and other ruminants.\textsuperscript{4,6} However, recovery has been seen in cases where blood lead was as high as .8 ppm for a period of three weeks.\textsuperscript{2} No samples taken were near these levels and in fact most were much lower.

Most ranchers in the area feed hay or oats they have grown themselves and drinking water comes from the surrounding creeks and ranch wells. Mineral salts are often made available to livestock, but this is standard ranch management and is not done to combat lead contamination.

As stated earlier the major problem appears not to
be airborne lead particulate, but contaminated soils. Proper nutrition and adequate amounts of winter feed seem to be the major factors in keeping down lead levels.

It is my conclusion that with the improvements the ASARCO plant has made in environmental safety equipment, airborne lead particulate is not the major cause of lead toxicity. The major cause is contaminated soil, but armed with this knowledge and using proper ranch management, lead toxicity can be alleviated.
Fig. 1 INTERFERENCE BY LEAD ON HEME SYNTHESIS

Krebs Cycle

Succinyl-CoA + Glycine

ALA Synthesis
S-Aminolevulinic Acid

Pb

ALA-dehydratase

Porphobilinogen (PBG)
PBG-deaminase
PBG-isomerase

Uroporphyrinogen III
Uroporphyrinogen I

Uroporphyrinogen - decarboxylase

Coproporphyrinogen III

Pb

coproporphyrinogen oxidase

Protoporphyrin-9

Pb

Fe^{2+} (Ferrochelatase)

HEME
Fig. 2 AERIAL VIEW OF EAST HELENA SAMPLING SITES

To Gardner Ranch

LAKE HELENA

BURKHAM

ASARCO

KLEFFNER

NATIONAL FOREST
**Fig. 3**

**SURFACE WIND ROSES**

**SITE:** 760903 MICROWAVE

**EAST HELENA, MONTANA**

\[\text{JANUARY 1979} \]

**HOURS 0 TO 23**

**CALM 1.0\%**

*(TOTAL HOURS 698)*

\[\text{FEBRUARY 1979} \]

**HOURS 0 TO 23**

**CALM 0.2\%**

*(TOTAL HOURS 556)*

\[\text{MARCH 1979} \]

**HOURS 0 TO 23**

**CALM 0.0\%**

*(TOTAL HOURS 666)*

\[\text{APRIL 1979} \]

**HOURS 0 TO 23**

**CALM 0.0\%**

*(TOTAL HOURS 681)*
Fig. 4  
SITE: 760903 MICROWAVE  
EAST HELENA MONTANA

MAY 1979  
HOURS 0 TO 23  
CALM 0.0%  
(TOTAL HOURS 689)

JUNE 1979  
HOURS 0 TO 23  
CALM 0.0%  
(TOTAL HOURS 625)

JULY 1979  
HOURS 0 TO 23  
CALM 0.0%  
(TOTAL HOURS 490)

AUGUST 1979  
HOURS 0 TO 23  
CALM 0.0%  
(TOTAL HOURS 456)
Fig. 5
SURFACE WIND ROSES
SITE: 760903 MICROWAVE
EAST HELENA MONTANA

SEPTEMBER 1978
HOURS 0 TO 23
CALM 0.0%
(TOTAL HOURS 708)

OCTOBER 1978
HOURS 0 TO 23
CALM 0.0%
(TOTAL HOURS 744)

NOVEMBER 1978
HOURS 0 TO 23
CALM 0.0%
(TOTAL HOURS 634)

DECEMBER 1978
HOURS 0 TO 23
CALM 0.0%
(TOTAL HOURS 678)
TABLE I

INTERRELATIONSHIPS OF DIETARY CONSTITUENTS WITH ORAL LEAD TOXICITY

Dietary Intake of Individual Nutrients

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>normal</th>
<th>deficiency</th>
<th>excess</th>
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</thead>
<tbody>
<tr>
<td>Protein</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Calcium</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>0</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>0</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>Nicin</td>
<td>?</td>
<td>0</td>
<td>?</td>
</tr>
<tr>
<td>Pyridoxine</td>
<td>?</td>
<td>0</td>
<td>?</td>
</tr>
<tr>
<td>Iron</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Zinc</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Selenium</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Copper</td>
<td>+</td>
<td>+</td>
<td>+</td>
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</tbody>
</table>

a = + = yes, - = no, O = undefined, ? = not established
b = lead effect metabolism of nutrient
c = deficiency of nutrient increases severity of lead toxicity
d = an excess of nutrient decreases lead toxicity
## Table II

**Symptoms of Lead Toxicosis in Cattle**

<table>
<thead>
<tr>
<th>System</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central Nervous System</td>
<td>Blindness, Muscular Twitching, Hyperirritability, Depression, Convulsions, Grinding Teeth, Ataxia, Circling, Pushing Against Objects</td>
</tr>
<tr>
<td>Gastrointestinal Tract</td>
<td>Excess Salivation, Anorexia, Tucked Abdomen, Diarrhea preceded by Constipation</td>
</tr>
<tr>
<td>Other</td>
<td>Acute Death, Bellowing</td>
</tr>
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### TABLE III

**SAMPLES AND DATA FROM GARDNER RANCH - CONTROL GROUP**

<table>
<thead>
<tr>
<th>Ranch Tag #</th>
<th>Sample #</th>
<th>Blood lead mg/100g</th>
<th>Blood lead mg/100g</th>
<th># years in area</th>
<th>Time of year</th>
<th>Type of food</th>
<th>Source of food</th>
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<td>4</td>
<td>Jan'82</td>
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<td>&quot;</td>
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<td>6</td>
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<td>0.06</td>
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<td>0.01</td>
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<td>0.05</td>
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<td>&quot;</td>
</tr>
</tbody>
</table>

### TABLE IV

**SAMPLES AND DATA FROM KLEFFNER RANCH**

<table>
<thead>
<tr>
<th>Ranch Tag #</th>
<th>Sample #</th>
<th>Blood lead mg/100g</th>
<th>Blood lead mg/100g</th>
<th># years in area</th>
<th>Time of year</th>
<th>Type of food</th>
<th>Source of food</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>1B*</td>
<td>15</td>
<td>0.15</td>
<td>1</td>
<td>Jan'82</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>-</td>
<td>2B*</td>
<td>16</td>
<td>0.15</td>
<td>1</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>85</td>
<td>3B</td>
<td>13</td>
<td>0.13</td>
<td>2</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>6A</td>
<td>4B</td>
<td>9</td>
<td>0.09</td>
<td>5mo</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>16</td>
<td>5B</td>
<td>12</td>
<td>0.11</td>
<td>5mo</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>-</td>
<td>6B</td>
<td>10</td>
<td>0.08</td>
<td>5mo</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>3F</td>
<td>7F</td>
<td>16</td>
<td>0.16</td>
<td>2</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>Pepper</td>
<td>9B</td>
<td>13</td>
<td>0.16</td>
<td>8</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>Alpo</td>
<td>9B</td>
<td>14</td>
<td>0.14</td>
<td>2</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>

* - indicates same animal
### Table V

**Samples and Data from Burkhart Ranch**

<table>
<thead>
<tr>
<th>Ranch Tag #</th>
<th>Sample #</th>
<th>Blood lead µg/100g</th>
<th>Blood lead ppm</th>
<th># years in area</th>
<th>Time of year</th>
<th>Type of Food</th>
<th>Source of Food</th>
</tr>
</thead>
<tbody>
<tr>
<td>2X</td>
<td>1C</td>
<td>12</td>
<td>.12</td>
<td>2</td>
<td>Feb. '82</td>
<td>hay ±</td>
<td>self-cultivated</td>
</tr>
<tr>
<td>23L</td>
<td>4C</td>
<td>16</td>
<td>.16</td>
<td>3</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>22J</td>
<td>5C</td>
<td>15</td>
<td>.15</td>
<td>5</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>041W</td>
<td>6C</td>
<td>6</td>
<td>.06</td>
<td>2</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>36L</td>
<td>7C</td>
<td>7</td>
<td>.07</td>
<td>3</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>745J</td>
<td>8C</td>
<td>12</td>
<td>.12</td>
<td>4</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>55K</td>
<td>9C</td>
<td>3</td>
<td>.08</td>
<td>4</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>5011</td>
<td>10C</td>
<td>13</td>
<td>.13</td>
<td>6</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>727J</td>
<td>11C</td>
<td>13</td>
<td>.13</td>
<td>5</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>68F</td>
<td>12C*</td>
<td>13</td>
<td>.14</td>
<td>8</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>68F</td>
<td>13C*</td>
<td>14</td>
<td>.12</td>
<td>8</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>810K</td>
<td>14C</td>
<td>13</td>
<td>.12</td>
<td>4</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>782J</td>
<td>15C</td>
<td>22</td>
<td>.22</td>
<td>5</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>391J</td>
<td>16C</td>
<td>12</td>
<td>.12</td>
<td>5</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>522G</td>
<td>17C</td>
<td>12</td>
<td>.12</td>
<td>7</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>

* - indicates same animal

+ - indicates mineral mix food supplement containing calcium, high phosphorus, magnesium, vitamin A, iodine, zinc
TABLE VI
SAMPLES AND DATA FROM DIEHL RANCH

<table>
<thead>
<tr>
<th>Ranch Tag #</th>
<th>Sample #</th>
<th>Blood lead mcg/100g</th>
<th>Blood lead ppm</th>
<th># years in area</th>
<th>Time of year</th>
<th>Type of Food</th>
<th>Source of Food</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>1D</td>
<td>10</td>
<td>.10</td>
<td>3</td>
<td>Late Feb., 82</td>
<td>†hay</td>
<td>†straw</td>
</tr>
<tr>
<td>-</td>
<td>2D</td>
<td>14</td>
<td>.14</td>
<td>2</td>
<td>††</td>
<td>††</td>
<td>††</td>
</tr>
<tr>
<td>-</td>
<td>3D</td>
<td>10</td>
<td>.10</td>
<td>3</td>
<td>††</td>
<td>††</td>
<td>††</td>
</tr>
<tr>
<td>-</td>
<td>4D</td>
<td>9</td>
<td>.09</td>
<td>3</td>
<td>††</td>
<td>††</td>
<td>††</td>
</tr>
<tr>
<td>-</td>
<td>5D*</td>
<td>12</td>
<td>.12</td>
<td>7</td>
<td>††</td>
<td>††</td>
<td>††</td>
</tr>
<tr>
<td>-</td>
<td>6D*</td>
<td>14</td>
<td>.14</td>
<td>7</td>
<td>††</td>
<td>††</td>
<td>††</td>
</tr>
</tbody>
</table>

* - indicates same animal
† - indicates mineral mix food supplement containing calcium, high phosphorus, magnesium, vitamin A, iodine, zinc

TABLE VII
EXPECTED LEAD CONTENTS OF UNCULTIVATED SOILS

<table>
<thead>
<tr>
<th>Element</th>
<th>Depth of soil, in.</th>
<th>Concentration (ppm) at indicated distance from stack, mi</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Lead</td>
<td>0 to 1</td>
<td>4000</td>
</tr>
<tr>
<td></td>
<td>2 to 4</td>
<td>490</td>
</tr>
<tr>
<td></td>
<td>6 to 10</td>
<td>74</td>
</tr>
</tbody>
</table>
TABLE VIII
WATER SOURCES

<table>
<thead>
<tr>
<th>Name of Ranch Owner</th>
<th>Water Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Don Burnham</td>
<td>Prickly Pear, BLM Drainage</td>
</tr>
<tr>
<td>Dave Diehl</td>
<td>Helena Drainage, Holmes Gulch</td>
</tr>
<tr>
<td>Curt Gardner</td>
<td>Ranch wells</td>
</tr>
<tr>
<td>Paul Kleffner</td>
<td>Ranch well</td>
</tr>
<tr>
<td></td>
<td>Prickly Pear</td>
</tr>
</tbody>
</table>
BLOOD LEAD DETERMINATIONS

General Procedure:

A Perkin-Elmer 503 equipped with deuterium arc background corrector, peak reader and Delves cup modifications is used for determinations.

10 μl of blood are pipeted into each Delves cup, dried on a hot plate, ashed at burner edge and inserted into the flame. Readings are taken directly in concentration from peak reader.

Analysis:

Blood sample must be collected with anticoagulants. We use BD 7 ml tubes with sodium oxalate.

Sample Preparation; (Prepare unknowns in duplicate)

1: Mix samples for at least 1 hour using a rotating mixer.

2: Pipet 10 μl of blood into Delves cup. Rinse pipet tip with two 10 μl portions of H₂O, adding rinses to cup.

3: Dry blood on 140°C hot plate.

4: Insert cup into loop and move cup to within 2-3 mm of flame. Blood will ignite. Wait until flames die in cup and then insert cup directly into flame.

5: Wait until cup turns cherry red and then withdraw it.

Standards:

Allow instrument to warm up for about 5 minutes.

Prepare a .4 μg/ml and .8 μg/ml Pb standard by diluting 1 and 2 mls of 10 μg/ml stock solution to 25 mls with H₂O. Prepare fresh standard daily.

Using blood with a low known concentration of Pb, mix and pipet into cup as you would unknown. Then add 10 μl of appropriate standard to cups.
With the 503 Peak Reader, standards are read directly in concentration by the following method.

The upper curve is set by taking the 8 μg standard plus value of blood and dialing this concentration in a calibration. For example, if blood concentration is known to be 14, then 8 μg is set into the calibration (80 + 14). After upper curve is established, the linearity of curve is checked with 4 μg standard and blood alone. In this case, for example, the blood should give a reading of 14 and the 4 μg standard should read 54 (40 + 14).

A quality control blood sample of known value is also run with each batch of samples. Our quality control was set by requesting a pint of blood from a man with known exposure to Pb. Upon arrival in our lab, the blood was drawn into 7 ml collection tubes identical to those used to draw our regular samples. Approximately 80 tubes of blood were obtained in this way. 20 of these tubes were run by the long path method to determine Pb value. The remaining tubes were frozen and are used as needed.

Three standards consisting of a blank (no standard added), 4 μg and 8 μg are prepared as above and run with each set of 20. One quality control is run with each set. The samples are repeated if:

1: Quality control is more than + 2 S.D.
2: Duplicates are not within 3 μg of each other.
3: Sample is above 80 μg/100 grams.

Instrumentation:

Instrument is set up following instructions in Perkin-Elmer manual with instrument in peak mode and concentration measurement button depressed.

Peak read button is depressed and then released. Auto zero is then depressed. This clears any stored readings from peak reader.

The calibration button is depressed and desired calibration is dialed in. (This is displayed on digital display.)

Peak read button is depressed and sample is inserted into flame as described above. After sample is withdrawn from flame the peak read button is released. The Auto Conc. is depressed (this releases calibration button), and held down until number dialed in as calibration appears and read sign appears. Calibration is now complete.

The next sample is placed in loop. Peak read button is
depressed. Sample is inserted and removed as described above. The peak read button is released and reading directly in concentration (μg/100 ml or μg/100 grams if you make conversion before calibrating) appears directly on digital readout.

Cups are now clean and ready for next use.

If duplicate results are within limits, the answers are averaged and reported.
REFERENCES CITED


8. Medical Files of Dr. Robert Painter, D.V.M.


10. Private Communications with James P. Sieverson including Internal ASARCO Laboratory Procedure
