

# Diagnosis and Clinical Analysis of Mineral Issues in Livestock

Spring Forage Field Day, June 2019

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## Overview

- **Copper deficiency**
- **Vitamin A deficiency**
- **Selenium/Vitamin E deficiency**
- **Calcium deficiency**
- **Secondary hyperparathyroidism**
- **Magnesium deficiency**
- **Testing strategies (pros and cons)**



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Today I'll briefly go through some of the more common nutritional diseases seen in livestock here in Hawaii and I'll finish by discussing the pros and cons of the different testing methods.

## Copper deficiency

- Can be caused by increased levels of iron, molybdenum, zinc, & sulfur (copper antagonists)
- Molybdenum and sulfur combine with copper to form copper sulfide (insoluble)



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Copper deficiency can be caused by deficient copper intake, but it's more commonly caused by increased levels of other minerals that antagonize copper, such as iron, molybdenum, zinc, and sulfur.

## Effects of decreased copper

- ↓ copper → defective tyrosinase (important in melanin synthesis)
  - Poor or faded hair coat
- Fatty degeneration of cardiac muscle fibers
- Impaired production of ceruloplasmin (copper-containing enzyme) involved in GI iron absorption; see decreased iron levels
- Demyelination (in lambs/kids)
- Abnormal bone development, foot rot
- Decreased immune response
- Retained placenta, decreased fertility & semen quality
- Sudden death



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Decreased copper levels will cause a number of different changes in the body. For example, copper is necessary to make the enzyme tyrosinase, which is important in melanin synthesis. This is why you see the coat color change in the animals. You can also see fatty degeneration of the muscle fibers in the heart, which can result in heart failure or sudden death. Copper deficiency can in turn cause iron deficiency by decreasing the levels of ceruloplasmin, which is important for iron absorption by the gut. In lambs and kids, you can see demyelination or degeneration of the nerves. Copper deficiency can also cause abnormal bone development, foot problems, decreased immune response, and various reproductive problems.

## Copper deficiency



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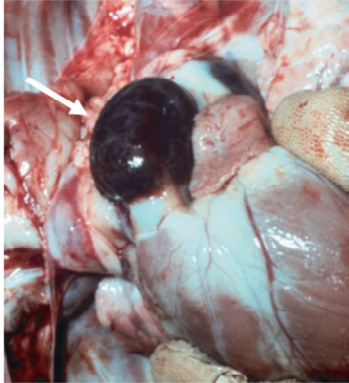
Color Atlas of Diseases and Disorders  
of Cattle, 3<sup>rd</sup> Edition, 2011.



In this radiograph, the copper deficient animal is on the left, and the normal animal is on the right. You can see that the bone near the joints are flared and the growth plates are widened. There's also less bone density in the bone itself. The photo on the right shows an animal with similar lesions.

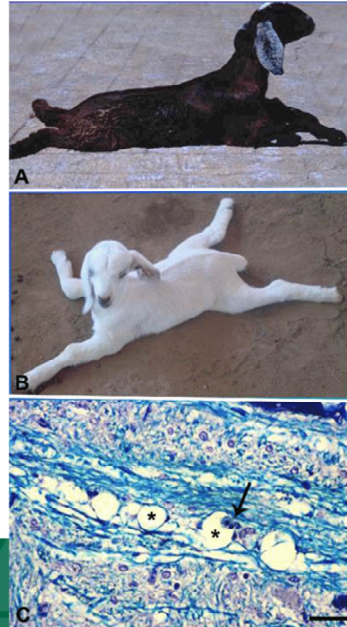
## Copper deficiency:

### Pig: dissecting aneurysm



Zachary, Pathologic Basis of Veterinary Disease, 6<sup>th</sup> Edition

## Enzootic ataxia, (swayback)



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Copper is also important for the strength and integrity of the vessels in the body. In pigs and turkeys, copper deficiency can cause ruptures of the aorta and sudden death. In sheep and goats, there is degeneration of the myelin resulting in the disease known as swayback.

## Copper deficiency

- Serum copper not a reliable indicator of copper status
- Liver copper is best indicator (site of copper storage)
- Measure copper-containing enzymes
  - Ceruloplasmin, superoxide dismutase



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Couple of quick notes on animal testing for copper deficiency. Serum levels of copper is not as good as measuring liver levels to detect true copper deficiency in the animal. You can also measure the activity of certain copper-containing enzymes in the serum to get a pretty good idea of true copper levels in the body.

## Copper toxicity

- Ruminants, but especially SHEEP
- Copper accumulates within the liver over a period of time
- Disease can be triggered by ingestion of liver toxins (PAs, mycotoxins)
- Hemolytic anemia, liver necrosis, icterus, hemoglobinuria

Color Atlas of Diseases and Disorders of Cattle (Third Edition)



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I do have to mention that if copper is oversupplemented, you can see the opposite syndrome, especially in sheep. Copper will accumulate in the liver, while the level in the serum remains at or near the normal levels. If copper levels increase above the toxic level, or if the animal ingests liver toxins such as pyrrolizidine alkaloids or mycotoxins, the liver is damaged and releases copper into the serum. The now elevated serum levels of copper will cause the red blood cells to rupture leading to hemolytic anemia. The hemoglobin that is released is filtered by the kidney, which also damages those. In these photos, the kidneys are black because of the hemoglobin that is trapped there. The liver and the kidney should be about the same color, so here you can see that this liver is pale indicating the presence of liver necrosis.



## Calcium deficiency

- Ideal Ca:P ratio is between 1.5-2 parts Ca to 1 part P
- Magnesium deficiency reduces calcium mobilization into blood
- Excess magnesium, phosphorus, and sulfur reduce calcium absorption
- Low blood calcium leads to increased parathyroid hormone secretion (PTH)
  - Leads to increased bone resorption (to maintain blood calcium levels) and osteoporosis



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The next nutritional disease I'm going to talk about is calcium deficiency which actually involves phosphorus and vitamin D levels as well. Ideally, the calcium to phosphorus ratio should be between 1.5-2 parts calcium to each part of phosphorus. Excessive magnesium, phosphorus and sulfur levels can decrease the amount of calcium that is absorbed. When calcium is low, the body increases the amount of parathyroid hormone to try to absorb more calcium from the gut and from the bones, which over time, can lead to osteoporosis.

## Calcium deficiency

- **Can cause rickets-like disease (like phosphorus/Vit D deficiency)**
  - **Milk fever (transient parturient disease)**
  - **Chronic true deficiency:**
    - **Dullness, lethargy, trembling of hindlimbs, weakness of legs, broken or weak bones**
    - **Stillborn calves and retained placenta**
- **Serum, vitreous humor, bone analysis**



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Calcium deficiency presents as two very different clinical syndromes. There is the transient rapid form seen in animals in the period around calving. This is most commonly seen in dairy cattle. The true chronic form of calcium deficiency results in a rickets-like disease where the bones do not develop normally and are weak. You can also see reproductive problems. Serum calcium can be used to diagnose the disease in the living animal. In the recently dead animal, you can use fluids from the eye for testing. You can also measure calcium levels in the bones.

## S-curve of neck; hypocalcemia



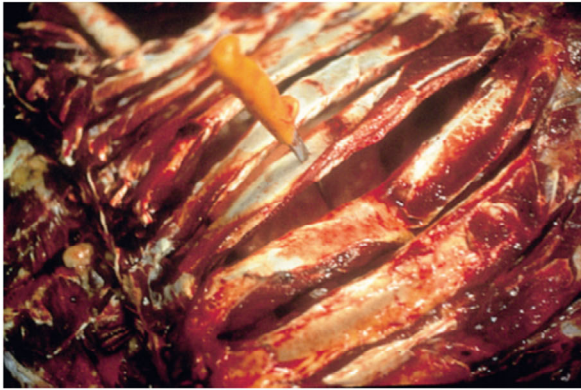
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This is a typical stage II milk fever presentation. The animal is down, but can keep herself upright. The neck is curved in this S-shaped manner as she tries to keep her head upright.

## Rickets (calcium, phosphorus, or vitamin D deficiency)



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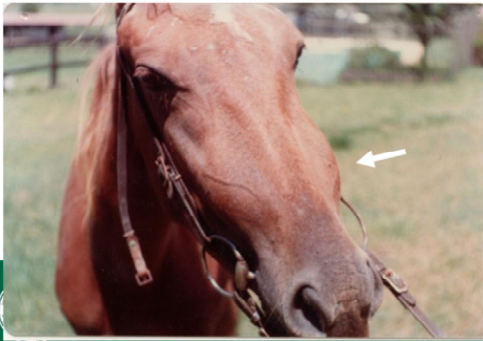
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With rickets, you will see an increase in rib fractures and enlarged fetlocks.

## Secondary hyperparathyroidism “Big Head”, fibrous osteodystrophy, in horses

- Low calcium intake relative to phosphorus intake; also high oxalate intake
- can cause lameness, abnormal cartilage & bone development, fractures, and bony swellings of the head



in Resources

<https://www.eag.org.au/big-head-paula-williams-bsc-hons-by-sc-ans>



In horses here in Hawaii, when the calcium to phosphorus ratio is too low, either with low calcium, or more likely, high phosphorus intake, again the body increases the secretion of parathyroid hormone, which mobilizes calcium from the bone. This results in soft bones that develop swellings, especially of the facial bones. That's why this disease is known as “big head.”

## Phosphorus deficiency

- **Decreased feed intake, unthriftiness, reduced growth rate, impaired reproductive performance, and bone abnormalities**
- **Can cause osteomalacia/rickets**
- **May increase metritis & decrease immune response**
- **Severe deficiency causes pica**
- **Measure bone/bone ash levels**
- **Serum, plasma, or urine, but may take weeks to show low levels**



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Phosphorus deficiency can cause poor growth, unthriftiness, impaired reproductive performance, and bone abnormalities, like rickets as previously mentioned. Phosphorus deficiency may cause a decrease in the animal's immune response. Animals with severe phosphorus deficiency may exhibit "pica" which is the ingestion of items that are not typically consumed, such as bones, sticks, or rocks. This deficiency is best diagnosed by measuring bone or bone ash levels of phosphorus. Serum, plasma, or urine may also be used to detect low phosphorus levels, but it may take weeks for a deficient animal to show low levels in these sample.

## Magnesium deficiency

- Grass tetany
- Usually seen in older cows
- Irritability, trembling, frothing at mouth, hyperesthesia, tetany, incoordination, convulsions & death
- Can cause myocardial degeneration
- Sudden death
- Can cause decreased feed intake and milk yield



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Magnesium deficiency causes a clinical syndrome known as grass tetany. It can look a lot like milk fever clinically. The animals start off looking irritable and then they'll develop fine muscle trembling. They'll froth at the mouth, then go down, convulse, and die. They can also present with just sudden death and have involvement of the heart. Milder forms of the disease will show decreased feed intake and milk yield.



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## Magnesium deficiency

- Test urine or vitreous humor in dead animals
- Monitor urine or serum



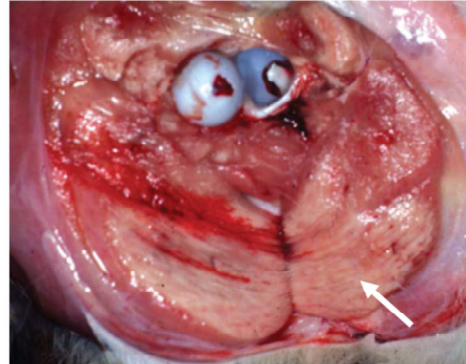
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The best samples to detect magnesium deficiency in an animal that is found dead are urine or vitreous humor from the eye. Urine or serum may be used to monitor live animals.

## Vitamin E/Selenium deficiency

- Vitamin E/Selenium-containing enzymes are antagonists of free radical formation that causes oxidative damage
- Selenium is antagonized by high levels of sulfur
- Disease is aka “White Muscle Disease”
  - Cardiac form
    - Rapid onset/sudden death
  - Skeletal muscle form
    - Slower onset, caused by impairment of glutathione peroxidase
    - See white/yellow, pale to chalky streaks in the muscle
      - Postural and locomotor muscles



Zachary, Pathologic Basis of Veterinary Disease, 6<sup>th</sup> Edition



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Vitamin E and selenium are both involved in the body's mechanism to protect against free radicals that oxidative damage to tissues. Selenium is another mineral that is antagonized by high levels of sulfur. The disease caused by Vitamin E or Selenium deficiency is known as white muscle disease. There are two forms of white muscle disease: the cardiac form primarily affects the heart and can cause rapid or sudden death. The skeletal muscle form, shown here in this image, has a slower onset, and causes white to yellow pale, sometimes chalky streaks in the muscle. The muscles that are most used are the ones that are most affected.

## White muscle disease

“Flying scapula”



Color Atlas of Diseases and Disorders of Cattle

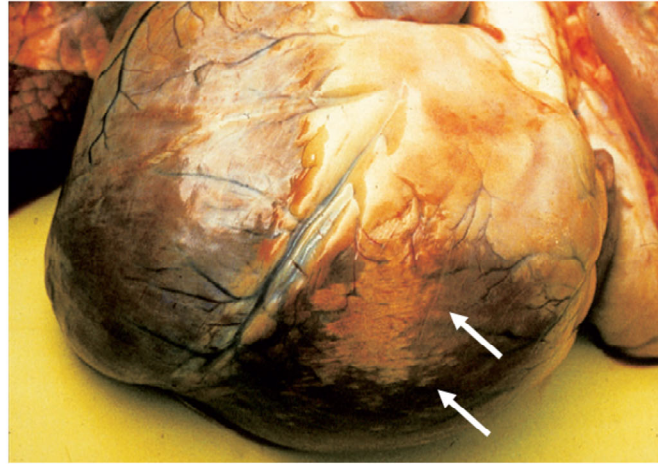


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This is a severe form of the disease caused by rupture of the muscles that support the trunk.

## White muscle disease

Globoid heart with pale patches



Color atlas of Diseases and Disorders of Cattle



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This photo shows one way that heart lesions may appear. Note the white, chalky patches marked by the arrows.

## Selenium deficiency

- Retained placentas, abortions, weak/stillborn calves, decreased fertility, cystic ovaries, metritis, decreased growth rate, reduced immune response
- Liver is the best sample
- Can also measure selenium-containing enzymes
  - Glutathione peroxidase
- Serum levels not easy to interpret



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Signs associated with selenium deficiency include a number of reproductive problems such as a higher proportion of retained placentas, abortions, weak calves, and decreased fertility, as well as decreased growth rate and immune response. The liver is the best sample for diagnosing selenium deficiency.

## Vitamin A deficiency

- Seen more after drought when forage quality is low
- Necessary for vision, maintenance of epithelial tissue, development, and immune function
- Fat-soluble vitamin, not stable in storage, loses ~50% activity over 6-12 months in storage
- Plants make carotene which ruminants convert into vitamin A
  - Generally an indicator of carotene content is the amount of greenness of the plant.
- Can store in the liver if over-supplemented (up to 4 months)



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The next disease I'll go over is vitamin A deficiency. This disease is seen more often after drought when forage quality is low. Vitamin A is necessary for vision, maintenance of the many epithelial tissues in the body, development, and immune function. Vitamin A is not stable in storage and can lose up to 50% of its activity with long term storage. Ruminants are able to convert carotene from plants into vitamin A.

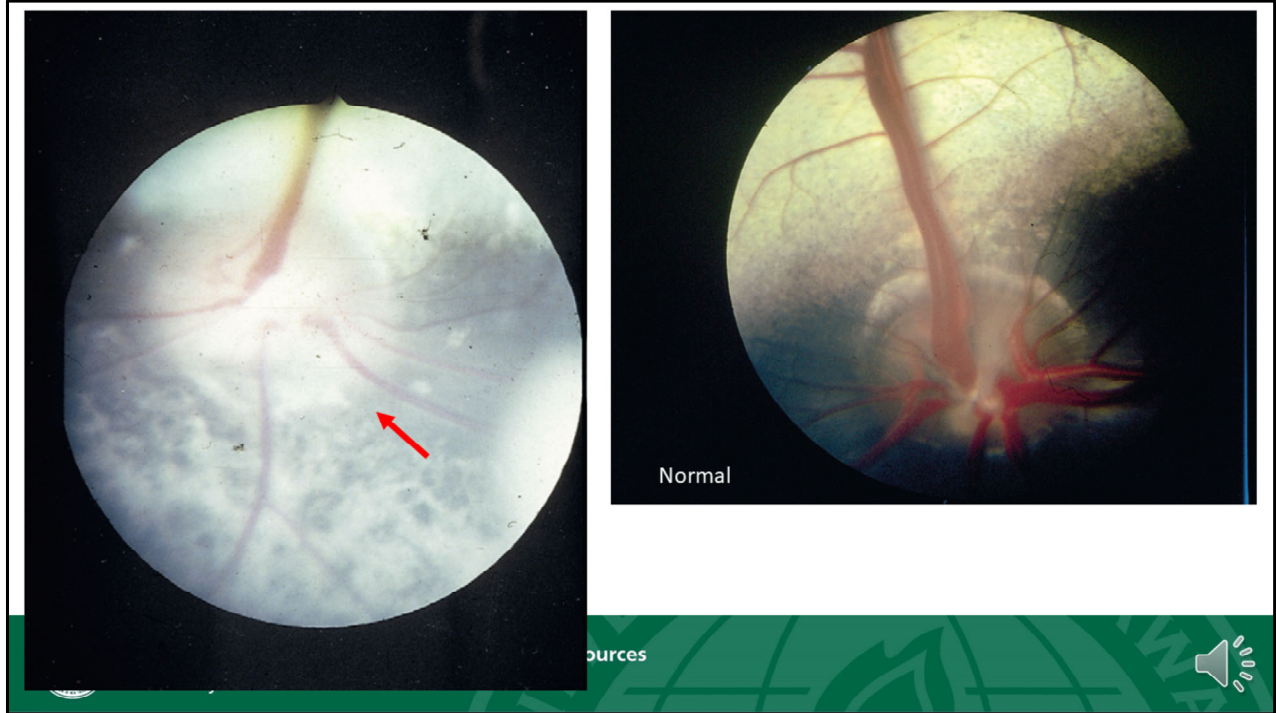
## Hypovitaminosis A

- **Reduced feed intake/growth, rough hair coat, night blindness, diarrhea, respiratory disease, seizures, increased susceptibility to infection, abnormal sperm, abnormal bone growth, low conception rates, abortion, stillbirths, and weak calves.**
- **Calves produced from cows with low vitamin A levels may have microphthalmia or constriction of the optic nerve**
  - See dilated pupil, retinal degeneration, and edema of the optic disc



Animals with Vitamin A deficiency may have decreased growth, rough hair coat, night blindness, diarrhea, respiratory disease, seizures, increased susceptibility to infection, abnormal sperm, abnormal bone growth, low conception rates, and increased rates of abortion, stillbirths and weak calves.

Calves produced from cows with low Vitamin A levels may have microphthalmia or small almost undetectable eyes. The optic nerve which is responsible for vision may be constricted and you may see changes in the such as dilated pupils, retinal degeneration, and edema of the optic disc.



The image on the right shows the back of the eye of a normal animal. The whitish round object towards 6 o'clock is the optic disk.  
The image on the left shows the eye of an animal with Vitamin A deficiency. The arrow is pointing toward the optic disk which is swollen and has blurred edges.



## Vitamin A deficiency

- Liver biopsy or post mortem liver analysis
- Calves born with clinical signs will probably not benefit from supplemental vitamin A



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The best way to detect vitamin A deficiency is by measuring it in the liver, either in the live animal via a liver biopsy or on a sample obtained after the animal has died. One thing to note is that calves that are born with clinical signs will probably not benefit from supplementing with Vitamin A, since the damaging effects are usually not reversible.

## Fluorosis

- Associated with volcanic ash in Hawaii
- Excess fluoride accumulates in the bone & kidney
  - Cumulative effect; long exposure times lead to highest risk
- Increased intake during tooth development (6-36 mo.) can cause soft, chalky, discolored enamel
  - Abnormal teeth & abnormal wear
  - Delayed eruption of permanent incisors
- Lameness, stiffness, abnormal hooves
- Delayed estrus and decreased reproductive performance



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Fluorosis is a disease caused by increased intake of fluoride. We see it here in Hawaii associated with volcanic ash. The excessive amounts of fluoride will accumulate in the bone and kidney. The effect is cumulative, so animals that are exposed for long periods of time are at highest risk of developing disease.

If young animals are exposed to increased levels of fluoride during the period of tooth development, the enamel doesn't form properly and becomes soft, chalky and discolored. You might observe abnormal appearing teeth or abnormal wearing of teeth. You may also notice delayed eruption of permanent incisors.

Other symptoms that you may see with increased levels of fluoride are lameness, stiffness of gait, abnormal hooves, delayed estrus and decreased reproductive performance.

## Fluorosis

Bony enlargements on medial aspect of metatarsal bones.

Color Atlas of Diseases and Disorders of Cattle (Third Edition)



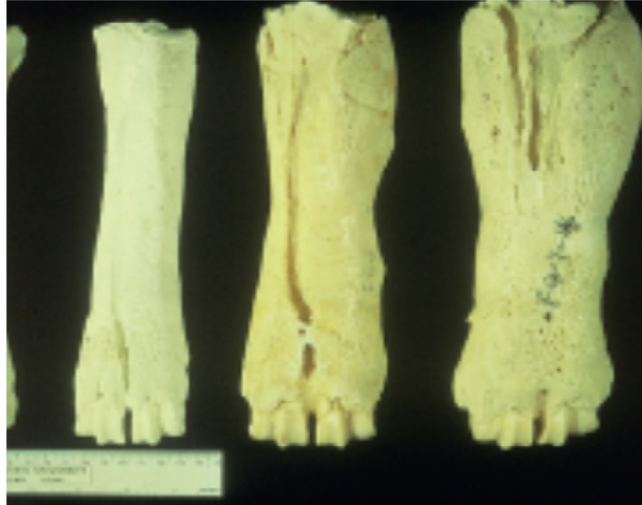
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This photo shows bony swellings in the fetlock area of this affected animal.

## Fluorosis

Extensive periosteal  
plaques on metatarsal  
bones



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In this photo, we see examples of varying levels of bony proliferation and swellings of the bones.

## Fluoride toxicosis

- Testing of forage is recommended for airborne fluoride pollution
- Testing urine fluorine levels
- Radiographic changes in teeth and skeleton
- Ribs or mandibles are best for testing fluoride levels



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Forage testing is recommended if you are concerned about airborne fluoride exposure. The animal's urine can be tested for fluorine levels, but the bones themselves, especially the ribs or lower jaw bones are the best samples for testing.

## Forage analysis

- Provides a real time indication of nutrition in forage
- Does not necessarily represent animal tissue levels
  - Acute vs. chronic changes
- Nutrient Requirements of Beef Cattle, 7<sup>th</sup> Revised Edition, 2000.
  - <https://www.nap.edu/catalog/9791/nutrient-requirements-of-beef-cattle-seventh-revised-edition-update-2000>



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Forage analysis is useful because it provides a real time snapshot of the nutrition available in the forage. However, it does not necessarily represent what is going on in the animal, especially with respect to chronic changes. I've attached a link here for the nutrient requirements of cattle that you can access on the internet, which can assist you in evaluating whether your forage is meeting your animals' nutritional needs.

## Live animal testing

- **Serum**
  - Separate from clot within 1-2 hours after collection (potassium, zinc)
  - Avoid hemolyzed samples (will falsely elevate iron, manganese, potassium, selenium, and zinc)
  - Royal blue-top tube
  - OK to freeze
- **Whole blood**
  - Royal blue-top tube
  - Do NOT freeze; keep refrigerated



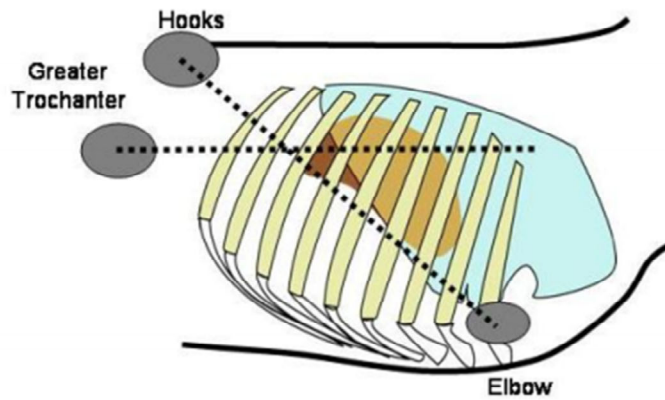
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In the live animal, serum can be a useful sample, but it must be separated from the clot very soon after collection. The serum should be collected in a royal blue top tube for best results. Once it is separated, it's okay to freeze the sample if it won't be shipped or tested right away. Whole blood can also be used, but it's important to remember NOT to freeze it.

## Live animal testing

- Milk
- Urine
  - OK to freeze
- Liver biopsy
  - Liver Biopsy Procedure in Cattle (DCPAH extension publication), Herdt, 2013



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Other samples that can be used for testing in a live animal are milk, urine and liver tissue collected by biopsy. Urine is OK to freeze and store if it can't be tested right away.



## Post-mortem Tissue Testing

- Liver tissue at necropsy or slaughter
  - gives you a better indication of total body nutritional status
- Kidney
- Bone
- Ocular fluid (aqueous and vitreous humor)
- Urine
- Fat
- Brain
- Rumen content



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This is a complete list of tissues that I recommend are saved at the time of necropsy or slaughter.

The best tissue for most types of testing is the liver. Kidney can also be a useful organ, especially for elements like lead. Bone is a good tissue to test for calcium, phosphorus, fluoride. Ocular fluid, urine, fat, for the fat-soluble compounds, brain, and rumen content are also good samples to save.

## Animal selection

- Age of animal being tested (fetuses v. adult)
- Groups of animals vs. individual (especially if ill)
- Ideally, test between 5-10 animals at the same stage of production



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Some other criteria to consider when deciding which animals to sample to get the best information are:

The age of the animal being tested. For some mineral issues, an aborted fetus can be used to detect some deficiencies. But for others, adult animal levels provide you the most accurate numbers.

In general you will want to sample groups of age matched animals at the same stage of production vs. testing just one ill individual. An animal that is ill with a non-nutritional disease may have abnormal values.

## References

- Mineral levels in animal health diagnostic data, Puls, 1988.
- Pathologic basis of disease in animals, McGavin.

• **THANK YOU!!!**



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