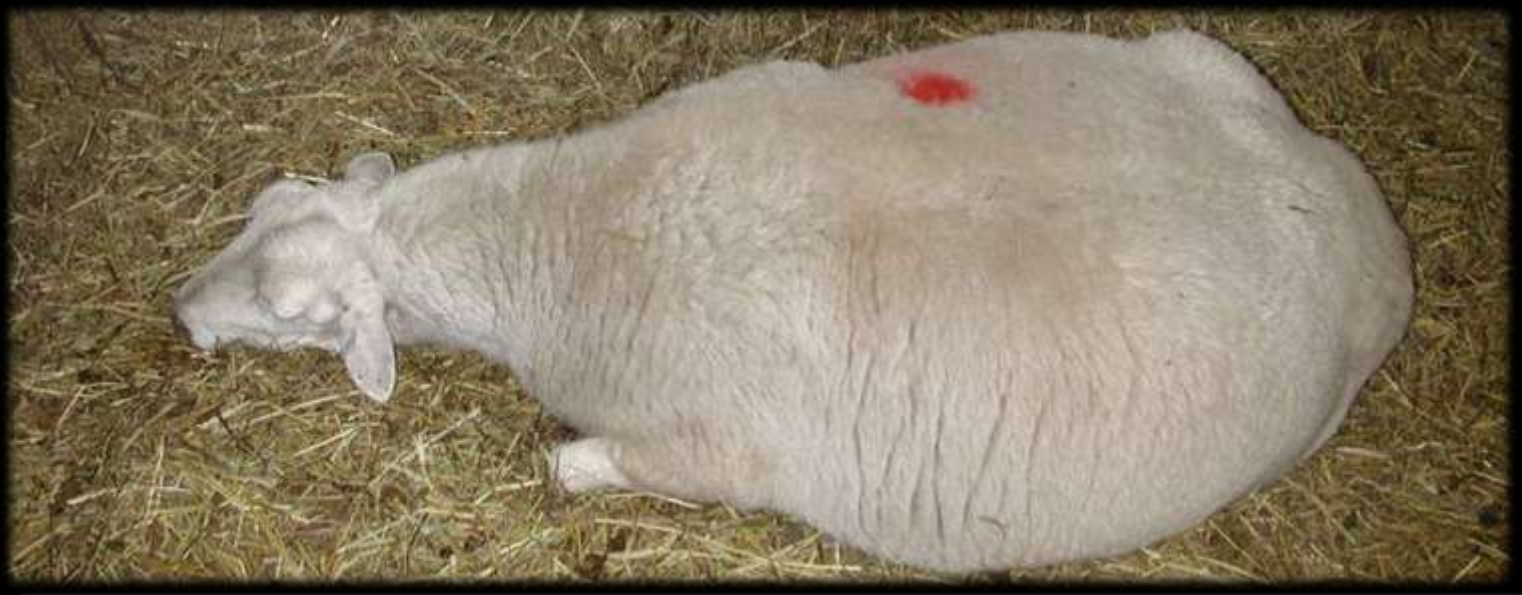
Nutritional disorders



# of sheep and goats

SUSAN SCHOENIAN (Shāy-nē-ŭn)
Sheep & Goat Specialist
Western Maryland Research & Education Center
sschoen@umd.edu - www.sheepandgoat.com

UNIVERSITY OF MARYLAND EXTENSION
SMALL RUMINANT PROGRAM

Nutritional and metabolic diseases

The result of nutrition and feed management problems

DISEASES A-Z

1) Acidosis
2) Bloat
3) Copper toxicity
4) Enterotoxemia
5) Milk fever
6) Poisonings
7) Polioencephalomalacia
8) Pregnancy toxemia
9) Scours (diarrhea)
10) Urinary calculi
11) White muscle disease

- Other names
- Cause(s)
- Risk factors
- Clinical signs
- Treatment(s)
- Prevention

Some abreviations

- Tx - treatment
- Vx – requires veterinarian
- Rx – requires prescription
- OTC - over the counter
- IM – intramuscular injection
- SQ – subcutaneous injection
- IV – intravenously injection

Acidosis

Lactic acidosis, rumen acidosis, grain poisoning, engorgement, grain founder, corn toxicity

CAUSE

- Large quantities of gas are produced in the rumen resulting in:
  - Δ Volatile fatty acids (VFAs)
  - ↑ Lactic acid
  - ↓ Rumen pH

➲ Pressure and inability to expel gas can lead to death.

RISK FACTORS

- Sudden intake of readily digestible carbohydrates: grain, pellets, or by-products, due to:
  1. Inadequate adjustment period
  2. Accidental access
  3. Variation in intake
- Lack of roughage in diet

Acidosis

Livestock can also experience acidosis on a pasture diet.

CLINICAL SIGNS

- Reduced appetite
- Depression
- Abdominal pain
- Bloat
- Rumen contractions
  Slow down → cease
- Diarrhea
  Mild → profuse
- Recumbency
- Death

➔ Death can be rapid!

Acidosis may also cause laminitis, a painful inflammation of the hoof.

Acidosis

TREATMENT

- Neutralize the acid
  - Diet adjustment: remove grain and feed good quality hay.
  - Oral drenches
    - Sodium bicarbonate
    - Vegetable oil
    - Mineral oil
    - Antacids
  - Other Tx's
    - Anti-inflammatory drugs [Rx]
    - Antibiotics
    - Fluid therapy

PREVENTION

- Gradual introduction of grain, pellets, or by-products to diet.
- Do not crack or grind feeds.
- Adequate roughage intake.
- Feed additives (rumen modifiers)
  - Ionophores (Bovatec®, Rumensin®)
  - Buffers (e.g. baking soda)
  - Yeast

Bloat

CAUSE

- Excess gas in the rumen.
- Failure to expel gas (belch)
  CO_2 and CH_4
- Two kinds of bloat
  1. Frothy or foamy (pasture)
  2. Free gas (feedlot)

RISK FACTORS

- Pasture bloat
  - Legume pastures, > 50% alfalfa, red/white clover, ~~lespedeza, birdsfoot trefoil~~
  - Small grain pastures.
  - Lush, wet pastures
  - Succulent pasture
- Feedlot bloat
  - Excessive consumption of grain

Bloat

CLINICAL SIGNS

- Distended abdomen, mostly on left side.

- Pain
- Depression
- Restlessness
- Diarrhea
- Difficulty breathing
- Respiratory failure
- Staggering
- Recumbency
- Death

Bloat

TREATMENT

- Mild cases
  1. Encourage belching
     Massage stomach, walk
  2. Drench with vegetable oil, baking soda, corn oil, antacid, or commercial anti-bloat medicine [OTC].
  3. Pass stomach tube to relieve pressure of gas.
- Life or death
  4. Rumenotomy - puncture a hole in the rumen with a 16 g needle [Vx]

PREVENTION

- Restrict pasture intake
- Fill animals with dry hay before turning onto lush or legume pastures.
- Gradual changes to diet
- Feed additives
  - Anti-bloat preparations
  - Ionophores (Bovatec®, Rumensin®)

Copper (Cu) toxicity

Copper nutrition is complicated, involving the interaction of other minerals, especially Molybdenum (Mo).

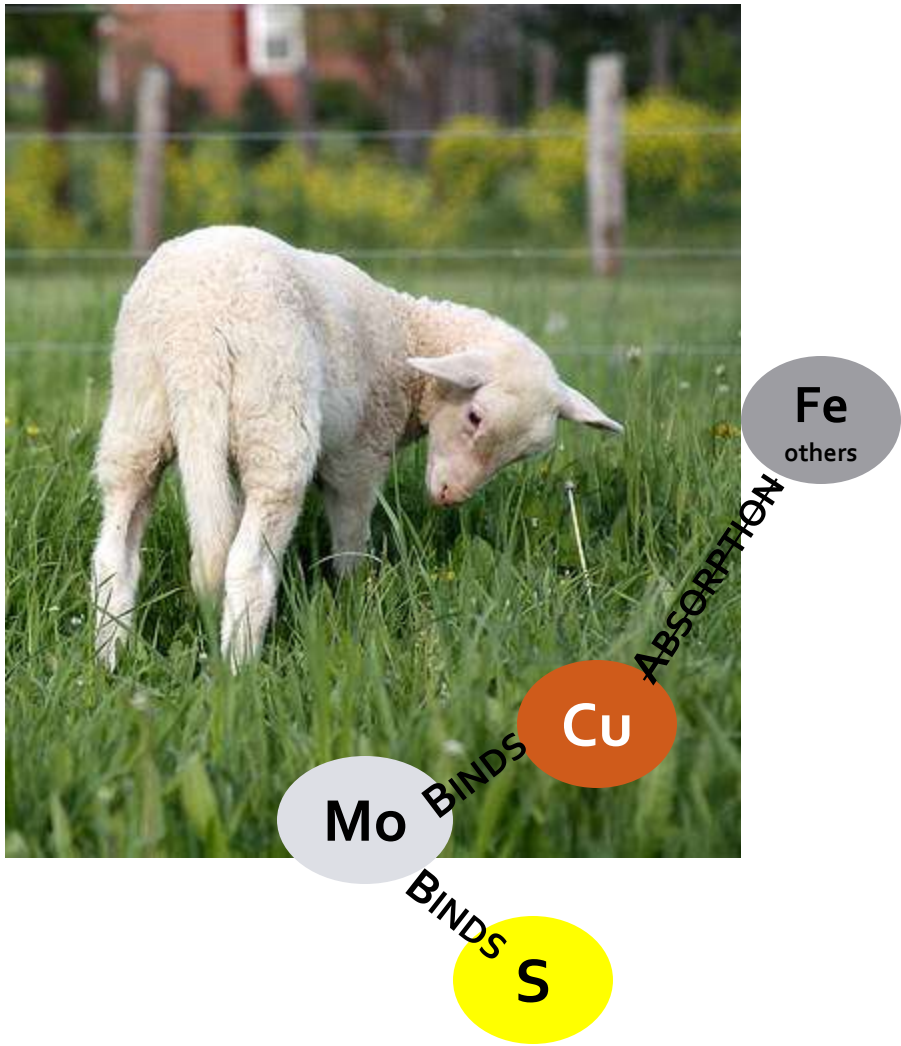
CAUSE

- Chronic vs. acute
- Liver capacity for copper has been exceeded .

  ↓

  Hemolytic crisis (RBC destruction) -triggered by stress

- ➲ The level of Cu that is toxic varies with the levels of Mo and S that are in the diet. Other minerals also affect copper absorption (e.g. Fe).



Copper (Cu) toxicity

RISK FACTORS

- Animal differences
  - Goats more tolerant than sheep.
  - Medium wool, Down/British breeds, and dairy sheep most susceptible .
  - Young animals absorb Cu more efficiently than older animals.
- Excess copper in diet
  - Feeding minerals or feeds that are formulated for other livestock.
  - Errors in feed formulation.
  - Adding copper to feed or mineral.
- Copper antagonists (Mo, S, Fe)
  Low levels of molybdenum (Mo)
  Cu:Mo should be $\leq$ 10:
- Other
  - Soil chemistry
  - Copper sulfate foot baths
  - Anthelmintics with copper
  - Copper plumbing

The Texel is the most susceptible to Cu toxicity, while sheep with Finn breeding are the least.

Copper toxicity

CLINICAL SIGNS

- Sudden onset
- Weakness
- Teeth grinding
- Thirst
- Dark brown or red-colored urine
- Jaundice
  Yellowing of membranes
- Anemia
- Shallow breathing
- Recumbency
- Death

Source of images: NADIS UK

Copper toxicity

TREATMENT

- Inactivate copper.
  - Ammonium molybdate
  - Ammonium sulfate
  - Curprimine [Rx]

PREVENTION (mostly sheep)

- Don't' add copper to ration or mineral.
- Don't feed minerals or feeds that have been formulated for other species.
- Don't deworm with copper sulfate or copper oxide wire.
- Don't fertilize pastures or hayfields with swine or poultry manure.
- Don't use copper sulfate footbaths.
- If copper toxicity is suspected, test feeds, forages, and soils for levels of Cu, Mo, and S.

Copper deficiency

Copper concentration in legumes

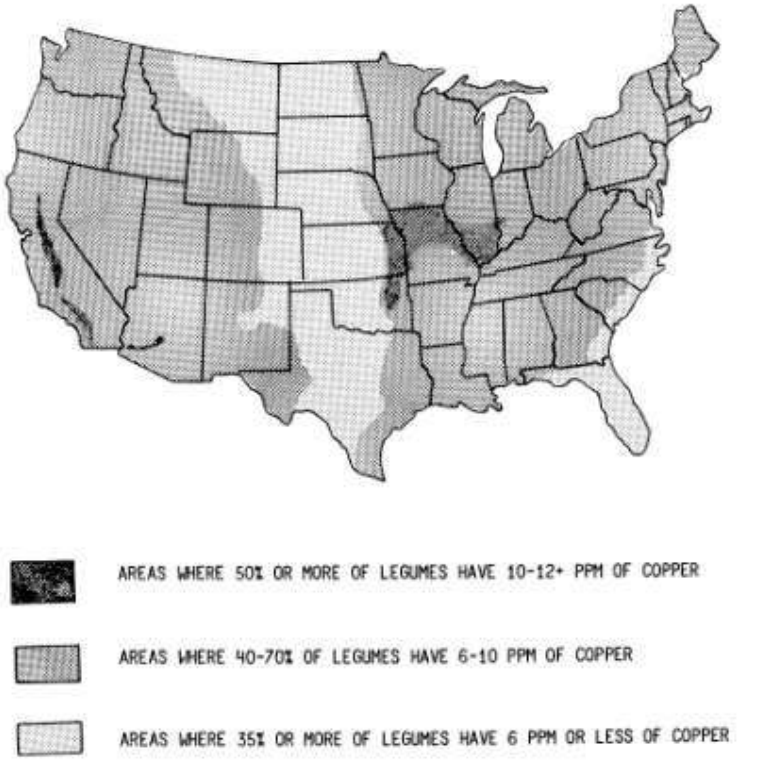


Figure 7. Generalized distribution of copper concentration in legumes of the United States (ppm = 1 $\mu g/g^{-1}$). From Kubota, 1983a, by permission Amer. Society of Agronomy.

RISK FACTORS

- Copper-deficient soils
- Low copper levels in plants
- Excessive consumption of Mo or S in pasture or feed.

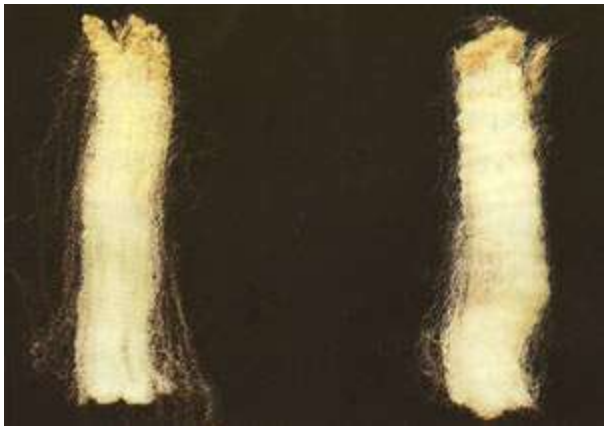
DIAGNOSIS

- Laboratory tests
  - Liver
  - Blood
  - Pasture

Copper deficiency

CLINICAL SIGNS

- Swayback
- Ataxia
  (lack of muscle coordination)
- Rough hair coat
- Dull coat
- Hair loss in goats
- Steely or stringy wool
  (lack of crimp)
- Loss of pigmentation in black-wooled sheep
- Poor performance
- Reproductive problems
- Anemia
- Death



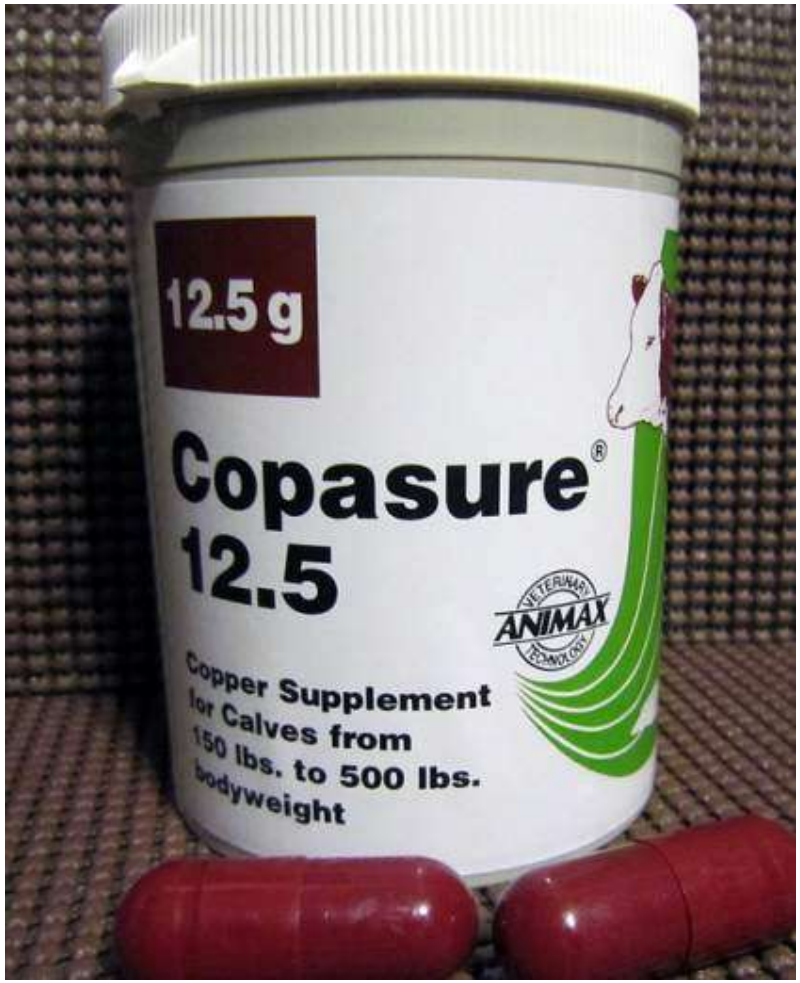
Copper deficiency

TREATMENT

- Supplemental copper
  1. Injections
  2. Oral drenching
     - Copper sulfate
  3) Boluses
     - Copper oxide wire particles

PREVENTION

- Feed properly-balanced rations: Cu : Mo: S
- Do not feed minerals formulated for sheep to goats.
- Fertilize with copper
- Supplemental copper: same as treatment above



Enterotoxemia

Clostridium perfringins type C & D, overeating disease, pulpy kidney disease

CAUSE

- An increase in the bacteria produces an endotoxin that is released into the blood stream and causes an inflammation of the intestine and swelling of the lungs and kidneys.
- Affects mostly lambs and kids shortly after birth, through their feeding period.
- Adults are mostly immune

RISK FACTORS

- Vigorous, healthy, rapidly growing lambs and kids (e.g. singles)
- Sudden intake of large quantities of grain, pellets, or by-product feeds.
  - Accidental access
  - Inadequate adjustment period
  - Variation in intake
- Lush pastures
- Loss of litter mate
- Inadequate roughage intake

Enterotoxemia

Clostridium perfringins bacteria are normally present in low numbers in the gut.

CLINICAL SIGNS

- Sudden death
  [usually best, fastest growing lambs and kids]

- Off feed
- Acute indigestion
- Lethargic
- Colic
- Nervous system signs
- Abdominal discomfort
- Profuse diarrhea

Enterotoxemia

TREATMENT

- Individual
  [usually not successful in severe cases]
  - C & D anti-toxin
  - Penicillin [OTC]
  - Additional Tx's
    - Oral electrolytes
    - Anti-inflammatory drugs [Rx]
    - Thiamine [Rx]
    - Probiotics
    - IV fluids
- Outbreak - whole herd
  - Increase forage in diet
  - Add chlorotetracycline to feed
  - Administer anti-toxin

Enterotoxemia

PREVENTION

- Management
  - Gradual feed changes
  - Steady intake of feed or milk
  - Feed additives (Aureomycin®)
  - Limit access to grain and lush pasture
  - ~~Let creep feed run out~~

- Vaccination
  - Ewes and does: annual booster during late pregnancy
  - Lambs and kids: vaccinate at approximately 6-8 and 10-12 weeks of age.
  - Annual vaccination of all adult animals.
  - Some farms may need to vaccinate more frequently to provide adequate protection.

Milk fever

Periparturient hypocalcemia, parturient paresis, sleeping sickness

CAUSE

- Low level of blood calcium (Ca)
- Insufficient intake or absorption of calcium to meet fetal or lactation demands.
- Occurs anywhere from six weeks prior to parturition to 10 weeks after parturition.
  - Non-dairy (before parturition)
  - Dairy (after parturition)

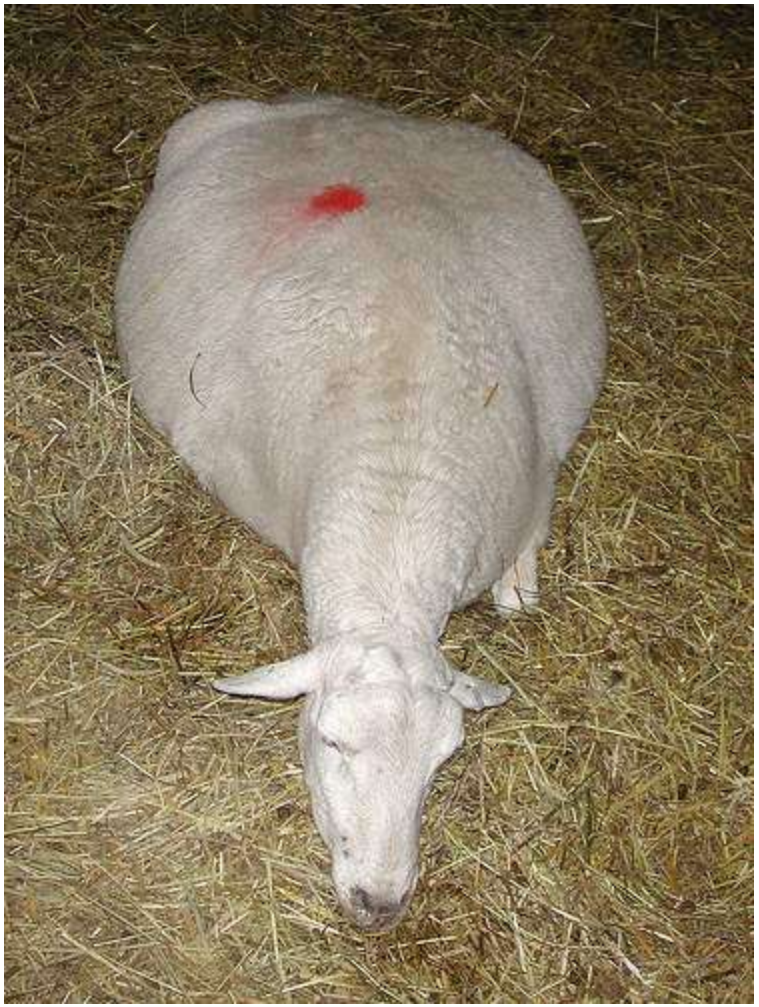
RISK FACTORS

- Calcium-poor diets or diets too high in calcium in late gestation.
- High producing females.
- Stress and handling.

Milk fever

CLINICAL SIGNS

- ~~Fever~~
- Sudden onset of symptoms
  - Uncoordinated
  - Nervous
  - Hyperactivity
  - Sluggish
  - Cold ears
  - Rear legs splayed out
  - Recumbency
  - Comatose
  - Death
- Clinical signs are similar to pregnancy toxemia; diagnosis is based on the response to treatment (calcium).



Milk fever

TREATMENT

- Calcium
  - Oral
    Calcium gluconate
  - Subcutaneous
    Calcium gluconate
  - Intravenous
    Calcium borogluconate
- Other Tx's
  - B-complex vitamins
  - Glucose
  - Dextrose
  - Magnesium

Milk fever

PREVENTION

- Proper levels of calcium in late gestation diet and over the long run.
  - Addition of limestone to the grain diet.
  - Feed better quality hay - part legume
  - ~~Overfeed calcium in grain or forage ration.~~
  - Avoid stressing females.

Poisonings

CAUSE

- Plant poisonings
- Nitrate poisoning
- Cyanide poisoning
- Urea poisoning
- Molds and mycotoxins

RISK FACTORS

- Drought
- Frost
- Poorly-managed pastures
- Stressed plants
- Access to poisonous plants or those that accumulate toxic substances
- Accidental exposure
- Improper mixing of feed
- Contaminated feed

Poisonings

CLINICAL SIGNS

- Vary with toxin and can be non-specific.
  - Acute vs. chronic
  - Sudden death
  - Excessive salivation
  - Labored breathing
  - Gastric distress
  - Neural symptoms
  - Photosensitivity
  - Reproductive problems

Poisonings

TREATMENT

- Varies with toxin and ability to diagnose cause of symptoms.
- Some poisonings have no effective treatment.
- Activated charcoal binds to toxins.
- Removal of the source of the toxin.

PREVENTION

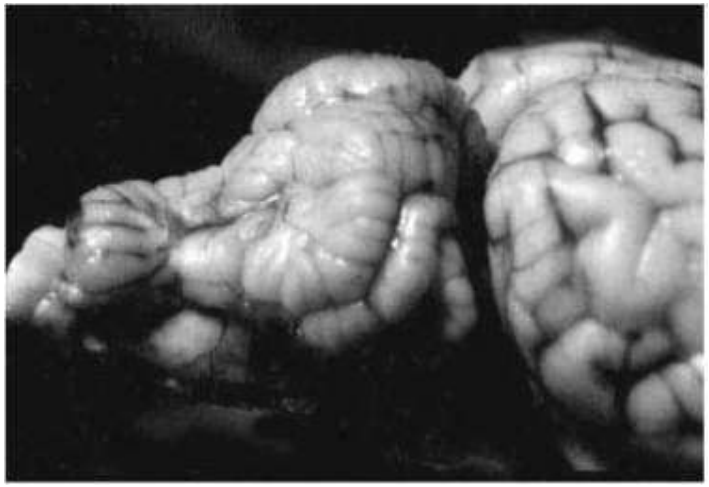
- Good pasture and grazing management.
- Removal of toxic plants.
- Test feeds for mycotoxins.

Polioencephalomalacia

Polio, PEM, cerebrocortical necrosis, cortical necrosis

CAUSE

- Acute or sub-acute
- Metabolic disease with neurological symptoms that are caused by a deficiency of thiamine (vitamin B1).



RISK FACTORS

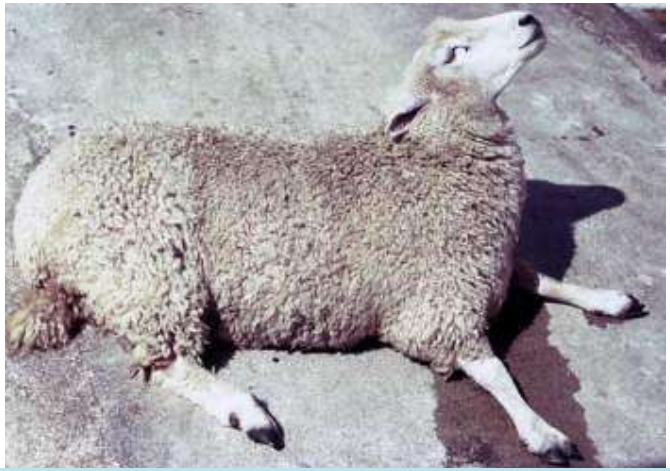
- Disturbance of thiamine metabolism
  - Sudden changes in diet
  - High grain diets
  - High sulfur intake
  - Prolonged treatment with Corid (amprolium).
  - Ingestion of plant thiaminases or thiamine analogs

THIAMINE
VITAMIN B1

Polioencephalomalacia

CLINICAL SIGNS

- Isolation
- Depression
- Lack of appetite
- Diarrhea
- ~~Fever~~
- Lack of muscle coordination
- Staggering
  - Blindness
- Star gazing
- Recumbency
- Death



Differential diagnosis: listeriosis, tetanus

Polioencephalomalacia

TREATMENT

- Thiamine - 10 mg/kg BW
  - Thiamine HCL 200 mg/mL [Rx] IM or SQ
    - B-complex vitamins [OTC] (contains less B1 per ml)
- Severe cases
  - IV injection of thiamine [Rx]
  - Repeated injections of thiamine, IM or SQ [Rx]
  - Anti-inflammatory drugs [Rx]
  - Fluid therapy

PREVENTION

- Good management
- Adequate roughage in diet
- Monitor sulfur intake
- Supplemental thiamine in diet

Pregnancy toxemia

ketosis, twin lamb disease, lambing paralysis, gestational toxemia, fatty liver disease

CAUSE

- Low blood sugar
- Energy imbalance
- Breakdown of energy into toxic ketone bodies which overwhelm liver capacity.

RISK FACTORS

- Inadequate intake of energy in late gestation
  - Poor quality forage
  - Lack of energy in diet
  - Variable feed intake
  - Reduced rumen capacity
  - Lack of feeder space
- Most common in females carrying multiple births.
- Fat or very thin females
- Lack of exercise [?]

Pregnancy toxemia

CLINICAL SIGNS

~3-10 day course

Lagging behind

Anorexia

Depression

Salivation

Nervousness

Wobbly

Recumbency

Death

- Some people can detect a sweet, acetone smell on the animal's breath
- There will be elevated ketones in urine or blood.
- Clinical signs are similar to milk fever.
- Diagnosis is based on response to treatment (glucose).

Pregnancy toxemia

TREATMENT

Get rid of the nutritional drain

1. Induce labor with steroids [Rx]
2. Caesarian section [Vx]

Glucose replacement

1. Oral propylene glycol
   Alternatives: Karo™ syrup, molasses
2. SQ glucose
3. IV glucose

Other Tx's

1. Calcium
2. Lactated ringers
3. Sodium bicarbonate

Pregnancy toxemia

PREVENTION

- Sufficient energy in diet of females during late pregnancy
  - Concentrates
  - Better quality forage
- Identify females carrying twins and triplets and feed them accordingly.
- Moderate body condition.
- Avoid stress
- Encourage exercise
- Adequate feeder space

Scours (diarrhea)

CAUSE

- Scours are not a disease.
- Scours are a symptom.
- There are many causes:
  1. Infectious
  2. Non-infectious

Scours (diarrhea)

NON-INFECTIOUS

- Parasitic
  - Worms ~~barber pole worm~~
- Nutritional
  - Dietary changes
  - Simple indigestion or allergy
  - Poor quality feed
  - High moisture content of feed
  - Toxins in feed
- Management
  - Poor sanitation
  - Overcrowding
- Stress
  - Weaning
  - Weather
  - Shipping/transportation

INFECTIOUS

- Bacterial
  - *E. coli*
  - *Salmonella*
  - Clostridial diseases
  - Johne's disease
- Viral
  - Rotavirus
  - Coronavirus
- Protozoan
  - *Eimeria* (coccidia)
  - *Cryptosporidia*
  - *Giardia*

Scours (diarrhea)

CLINICAL SIGNS

- Increased frequency, fluidity, or volume of feces.
  May have mucous or blood

- Dehydration
- Dirty legs and hocks
- Soiled wool
- Rough hair coat
- Ill thrift
- Poor performance

Scours

Most cases of diarrhea are self-limiting and will go away on their own

TREATMENT

- Depends upon underlying cause (and age of animal)
  - Non-infectious
    - Bismuth Subsalicylate [OTC]
    - Kaolin-Pectin [OTC]
    - ~~Immodium AD [OTC]~~
    - Probiotics [OTC]
    - Fluid therapy
  - Infectious
    - Antibiotics
      - Penicillin [OTC]
      - Spectinomycin® [Rx]
      - Corid, sulfa drugs [Rx]

PREVENTION

- Gradual changes in diet
- Roughage (dry) in diet
- Good sanitation
- Coccidiostats

Urinary calculi

water belly, kidney stones, urolithiasis, calculosis

CAUSE

- Calculi (stones) lodge in the urinary tract of mostly male animals and prevent urination.
- Stones are usually composed of phosphate salts, but may also be composed of calcium salts.

RISK FACTORS

- Primarily wethers (castrates)
  - Early castration
- Sometimes intact males
- Imbalance of Ca and P in diet.
- Concentrate diets excessive in P
- High grain: low roughage diets
- Forage diets excessive in Ca
- Lack of good quality water

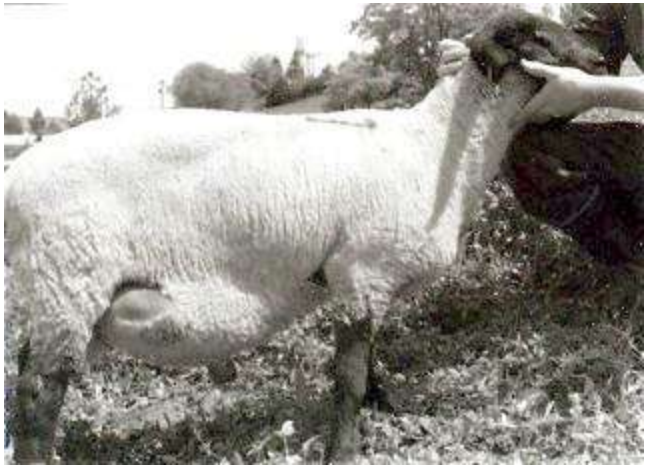
Ca: P

CALCIUM: PHOSPHORUS

Urinary calculi

CLINICAL SIGNS

- Isolation
- Discomfort
- Restlessness
- Anxiety
- Abdominal pain
- Urine dribbling
- Humped up appearance
- Distention of abdomen (edema)
- Rupture of bladder
- Death



Urinary calculi

TREATMENT

- Very early Ammonium chloride drench
- Early amputation of urethral process
- Later Urethrostomy Euthanasia

PREVENTION

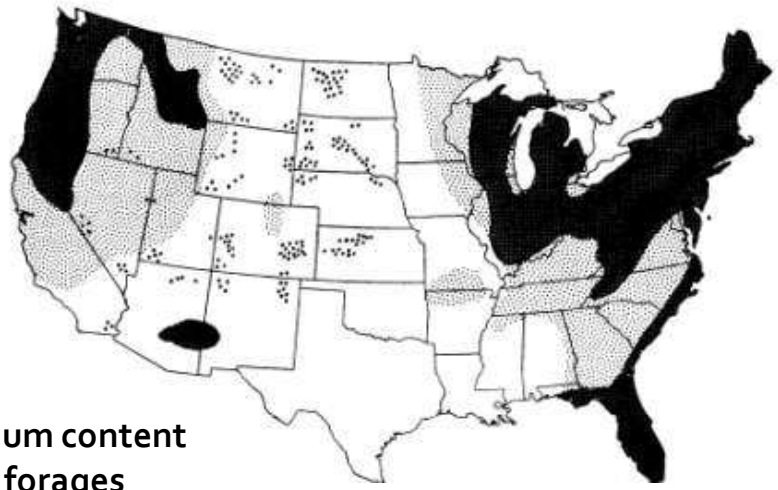
- 2:1 ratio of Ca to P in the diet
- ~~Feed alfalfa-only diet to males~~
- Addition of ammonium chloride to the diet
- Salt to stimulate water intake
- Adequate forage in the diet
- Ample supply of fresh water at all times
- Adequate vitamin A
- ~~Delay castration [?]~~

White muscle disease

Nutritional muscular dystrophy or myopathy, stiff lamb disease

CAUSE

- Degeneration of skeletal and cardiac muscles caused by a deficiency of vitamin E and/or selenium.
  - Congenital vs. acquired
  - Cardiac vs. skeletal



Selenium content of forages

RISK FACTORS

- Dietary deficiency of selenium and/or vitamin E.
  - Se: selenium-deficient soils and plants
  - Vitamin E: poor quality hay or lack of access to pasture

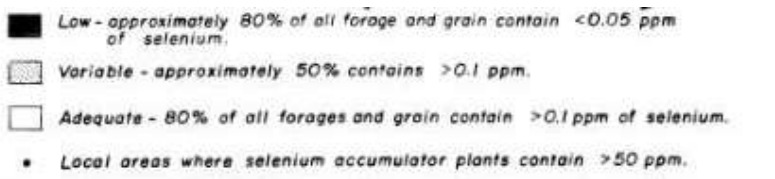
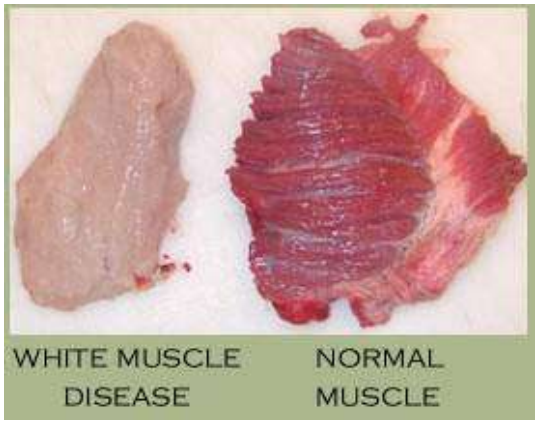


Figure 8. Geographical distribution of low-, variable-, and adequate-Se areas in the United States (ppm = 1 μg/g). From Kubota and Allaway, 1972, by permission Soil Science Society of America.

White muscle disease

CLINICAL SIGNS

- Cardiac
  - Similar to pneumonia
  - Irregular and elevated heart and respiratory rates
  - Death
- Skeletal
  - Stiffness
  - Weakness
  - Pain
  - Muscle trembles
  - Arched back
  - Hunched appearance
  - Stilted gait
  - Inability to stand



White muscle disease

TREATMENT

- Cardiac
  - Ineffective to treat
- Skeletal
  - Supplemental selenium and vitamin E (Bo-Se®)

White muscle disease

PREVENTION

- Good nutrition
  - Balanced rations
  - Good quality forages
  - Access to pasture
  - Free choice minerals
- Supplemental selenium

  0.10 to 0.30 ppm Se in total diet
  Daily intake not to exceed 0 0.7 mg/head/day
  - Feed and mineral supplementation
  - Oral gel
  - Injections

Other problems associated with a deficiency of selenium

- Increased mortality
- Ill-thrift
- Scouring
- Poor growth rates
- Infertility
  - Failure of embryo to implant
- Retained placentas
- Diminished fiber growth
- Periodontal disease
- Impaired immunity
- Chronic health problems

Unfortunately, none of these symptoms are specific to a Se deficiency. Only white muscle disease offers a definitive diagnosis.

Bo-Se ® injections [Rx only]

Selenium + vitamin E

- Labeled dosage (SQ or IM)
  - 1 ml/40 lbs for lambs 2 weeks of age and older (1 ml min.)
  - 2.5 ml/100 lbs. for ewes
  - ~~Pregnant ewes~~
    Has caused abortions
  - Not approved for goats or lambs under 2 weeks of age.
  - Seek advice of a small ruminant veterinarian before giving selenium injections to your animals.
- Feed supplementation is preferred to giving injections for providing adequate Se to sheep and goats.
- ➔ Should confirm selenium deficiency by post-mortem, blood test, or measured response to Se supplementation.

An overdose can be toxic.

Preventing nutritional problems

- Feed balanced rations
- Life cycle feeding
- Simple rations
- Gradual feed changes
- Adequate roughage in diet
- Regular body condition scoring
- Maintain animals in moderate body condition (2-4).
- Exercise and sunlight.

This is the final webinar in the 2012 six-part webinar series on sheep and goat feeding and nutrition.

Thank you for your attention.

Any questions?

Susan Schoenian
sschoen@umd.edu
www.sheepandgoat.com