

# Nutritional Disease and Problems in Sheep

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# Metabolic disorders and nutritive deficiencies of sheep

## 1. Rumen flatulence

### Causes:

- most often by eating legumes rich of protein (green alfalfa);
- animals are not accustomed to them.

## **2. Deficiency of vitamin E** **and selenium**

**Syndrome:** pregnant ewes fed with a diet, which is poor of vitamin E and/or selenium often have lambs suffering of **„white muscle disease“**;

lambs: thin, pulse and EKG are irregular, cause of death is waxy muscle dystrophy in skeletal and heart muscles.

**Treatment:** supplement of selenium (Na-selenite per os and subcutan) and vitamin E for ewes and one-day old lambs

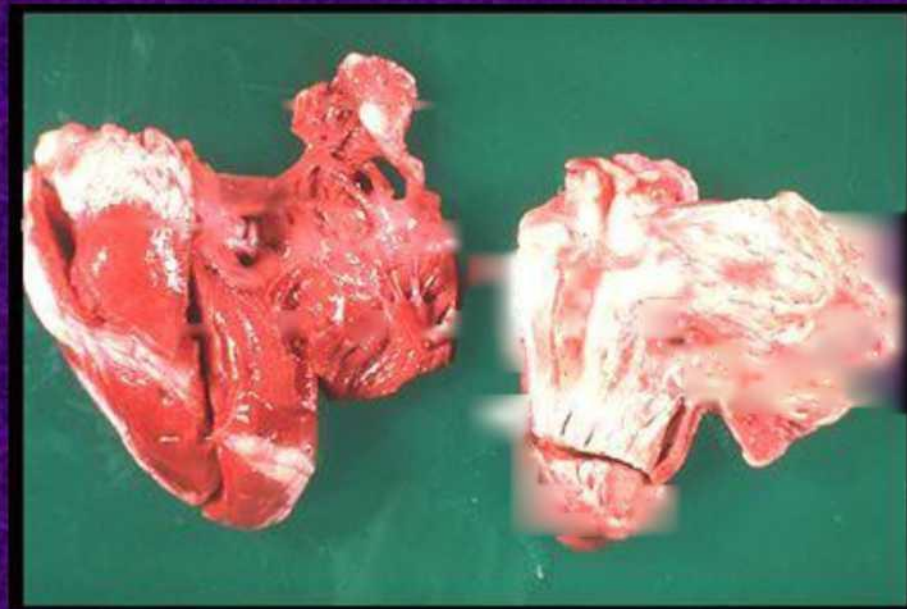
# Symptoms

## Skeletal

- Mild stiffness, obvious pain from walking, stiff gait, hunched appearance and inability to stand
- Lambs/kids tremble in pain when held in a standing position
- Normal appetites and bright personalities—until too weak to nurse
- Newborns born weak and unable to stand
- Sudden exercise may trigger WMD in older lambs or kids

## Cardiac

- Shows signs similar to pneumonia
  - Difficult breathing
  - Frothy nasal discharge (may be blood stained)
  - Fever
- Heart and respiratory rates elevated and often irregular
- Both types may occur at the same time!



# Prevention

- Deficiency occurs when animals are fed poor quality hay or straw or lack access to a pasture
  - Supplement the diet of the risk animals with Se or vitamin E
  - Especially pregnant animals since newborns are also at risk
- Sheep can get supplements either orally in their feed ration or through injections
- Goats can only get injections

### **3. White liver disease of lambs**

- nutritional origin, mainly arises in spring;
- morbidity of 40%, mortality of 15%;
- acute form: 7-10 days; chronic form: 4 to 6 weeks;

**Causes:** deficiency of cobalt, that induces the deficiency of vitamin B<sub>12</sub>;

In chronic case: propionate metabolism in liver is disturbed resulting accumulation of propionate;

- it might be accompanied by mycotoxin production.

### 3. White liver disease of lambs

**Acute form:** inflammation of eyelids and lips

originated in liver caused by secondary photosensibility;

**Cronic form:** aneemia,

high weight loss, *laesios* on the ears, decrease of albumin, cobalt and vitamin B<sub>12</sub> of plasma.

- activity of AST, GGT and SDH significantly increase;

- degenerative fatty liver, necrosis of hepatocytes;

- haemosiderosis of spleen.



## 4. Thiamin deficiency of growing sheep

**Syndrome:** opisthotonus;

in the background: polyencephalomalacia (PEM) and cerebrocortical necrosis (CCN).

**Occurance:** generally in the age of 3 to 4 months.

**Causes:** high level of concentrate, no transition time (importance of preliminary feeding!);

In winter: hay containing antimetabolite of thiamine may induce it, number of thiamine-synthesizing bacteria is reduced in rumen.

## **4. Thiamin deficiency of growing sheep**

**Symptoms:** in the first 2 to 5 days, excited behaviour, animals lay down and hold their head straight upwards („stargaizing”);

With no treatment: death in comatose status.

Similar symptoms: in listeriosis, ataxia caused by copper deficiency, scrapie.

### **Treatment:**

- first dose of thiamin: 0.5-2.0 g/animal /day (50% intravenous, 50% subcutan);
- recovery dose: 0.1-0.5 g thiamin/animal

## **5. Enterotoxemia of fattening lambs**

Other names: „overeating disease”, „pulpy kidney disease”

### **Occurance:**

- suckling lambs;
- lambs fed with milk replacers;
- fattening lambs on high-concentrate diet;
- high-pregnant and lactating ewes.

### **Symptoms:**

- pulpy kidneys (after necroctomy);
- toxins in rumen fluid.

## **5. Enterotoxemia of fattening lambs**

### **Results:**

- sudden death caused by toxin of type D (or sometimes C) produced by *Clostridium perfringens* (it occurs also in the intestinal tract of healthy sheep);
- in the case of feeding a diet rich in starch and sugar (concentrate, milk, fresh grass) *Clostridium perfringens* proliferates.

**Prevention:** vaccine containing toxoids.

Pregnant ewes: passive immunity for suckling lambs at age of 4-6 weeks; later: antiserum for lambs.

## **6. Acute lactic acid toxicity**

**Causes:** high-concentrate diet with no preliminary feeding.

### **Results:**

- concentration of lactic acid increases up to 2% in rumen fluid (normal: 50 mg%);
- pH in rumen: 4-4.2 (normal: pH 6-7);
- protozoa die in acid conditions in the rumen;
- high osmotic pressure develops (saliva, blood plasma);
- *collapsus* (preacute form), death.

## **6. Acute lactic acid toxicity**

**In acute and semi-acute forms:** metabolic acidosis; increase of numbers of *Lactobacilli*, *Coli* and *Proteus* bacteria.

**Symptoms:** in the nervous system, small quantity of yellowish-green faeces, anuria, flatulence, breath smells very acidic.

**Treatment:** 20-50 ml of 5% NaOH solution intraruminally, reduction of inflammation, supply of thiamin.

**Prevention:** preliminary feeding of high-concentrate diet, feeding hay or straw before grazing.

## **7. Urolithiasis**

**Development** of disease: split of *vesica urinaria*; urea flows into the abdominal cavity; „water belly”; death.

**Occurance**: castrated rams, fattening lambs on high-concentrate diet; increase of intake of Ca, P, Mg, K;

Grazing sheep: plants of high SiO<sub>2</sub>-content.

**Symptoms**: sedimentation of minerals in urinary tract, painful and slow urinating; phosphate content of urine increases.

**Prevention**: decrease of P-intake (Ca:P = 2:1);

Acidification of urine (0.5% NH<sub>4</sub>Cl in concentrate),

Drench of 7-14 g NH<sub>4</sub>Cl for 3-5 days; in grazing sheep: 3-4% NaCl in diet, water: *ad libitum*.

## **8. Urea toxicosis**

**Background:** feeding of NPN-compounds to fattening lambs with no preliminary feeding;

Toxic level of urea: 0.4 g per kg body weight.

**Treatment:** drinking of 500 ml household vinegar (20%) diluted 10-20 times;

Intravenous injection of maleic acid containing glucose (0.5ml per kg body weight).



## **9. Pregnancy toxicosis in ewes (sheep ketosis)**

**Occurance:** high-pregnant ewes.

**Background:** insufficient quantity of concentrate in diet; transitional glucose deficiency, energy eventually supplied from fat depots; pregnancy toxicosis (ketosis); long period of *hypoglucaemia* may cause damages in brain tissues.

**Symptoms:** unstable movement of high-pregnant ewes (faltering steps); comatose condition before death.

**Necroctomy shows:** healthy foetus(es) died before the ewe's death; yellow liver with rounded edges.

## **10. Milk fever in ewes**

- a. Classical (Ca ↑)
- b. Not typical (P ↑)

Syndrome: see in dairy cow

# Milk Fever

- Ewes vs. Dairy Cows
- Cause
  - Calcium deficiency ??
  - Stress induced

# Milk Fever

- Late gestation ewes carrying triplets
- Symptoms
  - Depressed, lethargic, recumbent
- Treatment
  - Calcium gluconate
  - Rapid response

# Copper Toxicosis

- Breed Susceptibility
- Mineral interactions-Mo & Su,
  - along with high Zn & Ca
- Normal copper, low molybdenum

# Copper Toxicosis

- Prevention
  - Sheep specific feeds
  - No additional copper
  - Feed some Mo
- Copper & Molybdenum
- are both toxic

# Pregnancy Toxemia

- Cause - Excess fat catabolism and ketone accumulation
- Prevention
  - Over conditioned ewes
  - Increased conc. feeding LG
  - Fetal scans
  - Pre-lambing shearing

# Pregnancy Toxemia

- Treatment
  - Propylene glycol
  - Induced parturition
- Severe cases usually do not recover



# Grass Tetany

- Hypomagnesemia
- Cause: excess potassium
- Lactating ewes on lush, spring pasture
- N fertilization increases risks

# Grass Tetany

- Magnesium oxide in mineral
- poor palatability.
- Sudden death-confirmation by necropsy
- Stress induced